2-Pentanol, 4-methyl-: Human health tier II assessment

28 June 2013

CAS Number: 108-11-2

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.

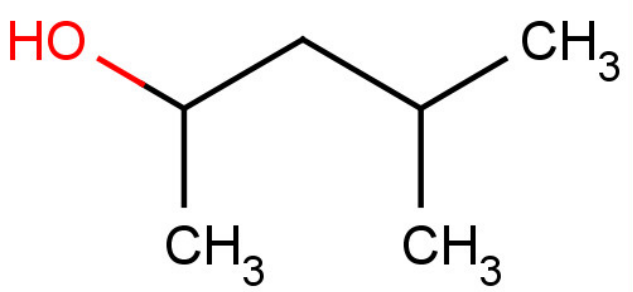
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	Methyl isobutyl carbinol (MIBC) 4-Methyl-2-pentanol 1,3-Dimethylbutanol 4-methylpentan-2-ol Methyl amyl alcohol
Structural Formula	
Molecular Formula	C6H14O
Molecular Weight (g/mol)	102.18
Appearance and Odour (where available)	Colourless liquid with a mild odour.
SMILES	C(C)(O)CC(C)C

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:

- manufacturing of other chemicals;
- mining and metal extraction (as a flotation agent); and
- as a solvent (in surface coating).

The chemical is also listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume of 1000–10000 tonnes.

International

The following international uses have been identified through the 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), Substances and Preparations in the Nordic countries (SPIN) database, the European Commission Cosmetic Substances and Ingredients (CosIng) database, United States (US) Personal Care Products Council International Nomenclature of Cosmetic Ingredients (INCI) directory and other data sources via eChemPortal including the US Environmental Protection Agency's (EPA) Aggregated Computer Toxicology Resource (ACToR), and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB):

The chemical has reported cosmetic use in:

- perfumes.

The chemical has reported domestic use including:

- surface active agents; and
- paints, lacquers and varnishes.

The chemical has reported commercial use including:

- as an additive to surface coatings as a solvent to maintain binder softness until the binder fuses; and
- as a solvent for dyestuff, oils, gums, resins, waxes, nitrocellulose, and ethylcellulose.

The chemical has reported site-limited use including:

- in the production of lube oil additives for anti wear and corrosion inhibitors (primary use); and
- as a flotation frother for treating copper ores, and coal and tar sand mining.

Restrictions

Australian

No known restrictions have been identified.

International

No known restrictions have been identified.

Existing Work Health and Safety Controls

Hazard Classification

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

Xi; R37 (irritation)

Exposure Standards

Australian

The chemical has an exposure standard of 104 mg/m^3 (25 ppm) time weighted average (TWA) and a short term exposure limit (STEL) of 167 mg/m^3 (40 ppm) in HSIS (Safe Work Australia).

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit of 80–110 mg/m^3 (TWA) in countries such as Canada, Denmark, France, Germany, Norway, Sweden, Switzerland, United Kingdom, and the USA.

An exposure limit (STEL) of 40–170 mg/m^3 in countries such as Canada, Greece, Poland, Spain, Sweden, United Kingdom, and the USA.

Health Hazard Information

Toxicokinetics

Following oral exposure in rats, the chemical was rapidly absorbed (Cmax- maximum concentration observed after administration, at approximately 30 minutes post-dosing) and the half-life was approximately 2 hours and 15 minutes in blood. The chemical was not detected in blood by nine hours and was rapidly metabolised to methyl isobutyl ketone (MIBK: CAS No. 108-10-1) and subsequently to the primary metabolite of MIBK, 4-hydroxy-4-methyl-2-pentanone (HMP; CAS No. 123-42-2). A similar absorption pattern was also observed following oral dosing of MIBK with Cmax occurring at approximately 15 minutes post-dosing. As dosing with the chemical or MIBK would result in similar internal exposure to MIBK and HMP with minimal exposure to the chemical due to its rapid metabolism, the toxicity database on the metabolites (MIBK and HMP) can be used appropriately to support the evaluation of the chemical, particularly for long term toxicity (OECD, 2005; REACH).

Acute Toxicity

Oral

The chemical was of low acute toxicity in animal tests following oral (gavage) exposure. The median lethal dose (LD50) in rats is 2260 mg/kg bw (OECD, 2005; REACH).

Dermal

The chemical was of low acute toxicity in animal tests following dermal exposure. The median lethal dose (LD50) in rabbits is 2870 mg/kg bw (OECD, 2005; REACH).

Inhalation

The available data indicate that the chemical should be classified with the risk phrase ‘Harmful by inhalation’ (Xn; R20). The chemical exhibited typical organic solvent effects in rats following acute inhalation exposure with anaesthetic effects occurring at high vapour concentrations (OECD, 2005; REACH).

Rats were exposed to 10 or 16 mg/L of the chemical (10000 or 16000 mg/m^3) for four hours (OECD TG 403). All exposed animals were anaesthetised within the first hour of exposure and regained consciousness within 30 minutes at 10 mg/L or two hours after cessation of the exposure at 16 mg/L. One female died at 16 mg/L. The four hour LC50 for the chemical was determined to be >16 mg/L. In another study, rats were exposed to saturated vapours (approximately 19 mg/L) of the chemical for up to two hours or to 8.4 mg/L for eight hours. No mortality was observed after the two hour exposure to 19 mg/L and five out of six rats died following the eight hour exposure to 8.4 mg/L within the 14 day observation period.

Corrosion / Irritation

Respiratory Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to respiratory system' (Xi; R37) in HSIS (Safe Work Australia). Although no animal data are available, observations in humans support this classification (see 'Observation in humans' section below) (OECD, 2005).

Skin Irritation

The available data indicate that the chemical should be classified with the risk phrase ‘Irritating to skin’ (Xi; R38).

When the chemical (undiluted) was applied to the skin of rabbits (n = 3) for four hours under semi-occlusive conditions (OECD TG 404), well-defined erythema with slight oedema was observed in all three animals. Desquamation of the stratum corneum, characterised by dryness and sloughing of the skin, also developed in all animals. Dermal responses were fully resolved by either day 10 or 12 in two animals. However, very slight erythema was still observed in one animal at day 14. No signs of toxicity or ill health were observed in any rabbit during the observational period. The individual mean scores over 24, 48 and 72 hours were 2.0, 2.0 and 2.0 for erythema and 2.0, 1.0 and 1.7 for oedema (REACH).

In another study, the chemical was applied to the skin of three rabbits for a single exposure period of 15 mins (10 mL) or for five repeated exposure periods of 5–12 hours over a 15 to 21 day period. Repeated application of the chemical resulted in severe drying of the skin with some sloughing and cracking (OECD, 2005).

Eye Irritation

The available data indicate that the chemical should be classified with the risk phrase 'Irritating to eyes' (Xi; R36).

The chemical caused severe irritation (irritation grade 5) following instillation of the undiluted chemical (0.02 mL) into the eyes of rabbits. The irritation grade 5 was defined as severe injury with necrosis, visible on fluorescein staining, covering approximately 75 % of the cornea, or more severe necrosis over a smaller area (OECD, 2005).

In another study, instillation of the undiluted chemical (0.1 mL) into eyes of rabbits (n = 3) resulted in moderate irritation with signs of conjunctivitis, oedema and corneal injury. The effects were reversible by day seven. Draize scores of 11, 25, and 17 were observed at 1, 24 and 72 hours (out of maximum score of 110) (OECD, 2005).

In another study (OECD TG 405), 0.1 mL of undiluted chemical was instilled into the conjunctival sac of three New Zealand White rabbits. All animals developed corneal opacification, iridial inflammation, and conjunctival irritation. Individual mean scores over 24, 48 and 72 hours were 1.0, 0.7 and 2.0 for corneal opacity, 0.7, 0.7 and 0.3 for iritis, 2.3, 1.3 and 1.3 for conjunctival redness and 1.0, 0.3 and 0.3 for chemosis. All reactions had resolved by either day seven or 14 after instillation (REACH).

Observation in humans

Exposure of human volunteers (12/sex) to vapours of the chemical at 50 ppm for 15 minutes resulted in eye irritation in most subjects with nose and throat irritation experienced at higher concentrations. The maximum tolerable concentration was reported to be 25 ppm (OECD, 2005; REACH).

Sensitisation

Skin Sensitisation

The chemical was not found to induce dermal sensitisation when tested according to OECD TG 406 using the guinea pig maximisation test (GPMT).

In this study, induction was carried out with an intradermal injection of 1 % solution of the chemical in Freund’s adjuvant followed by epicutaneous induction with 0.5 mL of the undiluted chemical. The challenge exposure also was conducted with 0.5 mL of the undiluted chemical (OECD, 2005; REACH).

Repeated Dose Toxicity

Oral

The chemical is not considered to cause serious damage to health by repeated oral exposure (OECD, 2005; REACH).

In a repeated dose oral toxicity study (OECD TG 422), Sprague-Dawley rats were dosed (gavage) with 0, 30, 100, 300, or 1000 mg/kg bw/d of HMP (the ultimate metabolite of the chemical) in distilled water for approximately 45 days. Males at 100 mg/kg bw/d and greater had an increased incidence and/or severity of hyaline droplets in the tubular epithelium in the kidneys. Males at 1000 mg/kg bw/d also had altered blood parameters, dilatation of the distal tubules of the kidneys, hepatocellular hypertrophy, and vacuolization of the zona fasciculata of the adrenals. Decreased locomotor activity and stimulation responses were observed in both sexes at 300 and 1000 mg/kg/d. In females, dilatation of the distal tubules and fatty degeneration of the proximal tubular epithelium in the kidneys were observed at 300 mg/kg bw/d. At 1000 mg/kg bw/d, females showed reduced body weight gain, increased liver weight along with hepatocellular hypertrophy and kidney lesions similar to those noted at 300 mg/kg bw/d. The NOAEL was 30 mg/kg bw/d for males, based on a male-rat specific lesion not relevant to human hazard assessment (hyaline droplet nephropathy). The NOAEL for females was 100 mg/kg bw/d, and the only effect seen in the males at this dose was the rat-specific kidney lesion assessment. The LOAEL was 100 mg/kg bw/d for males and 300 mg/kg bw/d for females (OECD, 2005; REACH).

Dermal

Although a reliable repeat dose dermal toxicity study was not available, severe drying of the skin with some sloughing and cracking was noted in rabbits following repeated application of the chemical for either a single exposure period of 15 min or for five repeated exposure periods of 5–12 hours over a 15 to 21 day period (OECD, 2005).

Inhalation

As no adverse systemic effects were reported in 6-week and 14-week inhalation toxicity studies in rats, the chemical is likely to be of minimal toxicity by the inhalation route.

In a 6-week inhalation toxicity study (OECD TG 407/412), Wistar rats were exposed (whole body) to vapours of the chemical at concentrations of 0, 0.211, 0.825, or 3.70 mg/mL (0, 50.5, 198, or 886 ppm) six hours per day, five days per week. There were no exposure related deaths, clinical signs of toxicity, effects on body weights, haematological changes, and histopathological effects during the course of the study. The NOAEC was the highest concentration tested, 3.70 mg/L (886 ppm or 3698 mg/m^3) (OECD, 2005; REACH).

In a repeated dose inhalation toxicity study (OECD TG 413), B6C3F1 mice and Fischer 344 rats were exposed to atmospheres containing the metabolite (MIBK) vapours at 0, 50, 250 or 1000 ppm (0, 0.20, 1.02 or 4.09 mg/mL), 6 hours/day for five days per week for 14-weeks. Male rats in the 250 and 1000 ppm groups showed an increase in hyaline droplets within the proximal tubule cells of the kidney, which are lesions specific to male rats. The NOAEC was 50 ppm (0.20 mg/L) in male rats, based on a male-rat specific lesion not relevant to humans (hyaline droplet nephropathy). The NOAEC was 1000 ppm (4.09 mg/L) for female rats, and for male/female mice, based on lack of adverse effects on clinical health or growth of the rats or mice at the highest dose (OECD, 2005; REACH).

Genotoxicity

The chemical and its ultimate metabolite (HMP) are not considered to be genotoxic based on several in vitro studies.

The chemical tested negative (with and without metabolic activation) in several in vitro assays including Ames reverse mutation assays, and in a gene mutation assay with *Saccharomyces cerevisiae*. The chemical also tested negative in a cytogenetic assay using rat liver cells (RL4) without metabolic activation (OECD, 2005; REACH). No mutagenic activity was observed in a mouse lymphoma assay (OECD TG 476) with mouse lymphoma L5178Y cells (REACH).

The ultimate metabolite of the chemical (HMP) also tested negative in bacterial reverse mutation assay and in chromosomal aberration assay using Chinese hamster lung cell line (CHL/IU) (OECD, 2005; REACH).

Carcinogenicity

The chemical and its metabolite (MIBK) are not considered to be carcinogenic based on available data.

In a chronic inhalation toxicity study (OECD TG 451), Fischer 344 rats were exposed (whole body) to the metabolite (MIBK) at concentrations of 0, 450, 900 or 1800 ppm for six hours per day, five days per week for two years. Although mortality was observed in all groups, the survival was significantly decreased in males at 1800 ppm compared to the controls (32/50 vs. 19/50). The treatment has no effect on the survival of females across all groups. Chronic progressive nephropathy (CPN) was observed in all rats (including controls). Although CPN observed was similar to that which occurs in aged rats, there were treatment related significant increases in both the incidence (1800 ppm) and severity in all exposed groups. Males exposed to 900 ppm and 1800 ppm of the metabolite (MIBK) also demonstrated other kidney lesions that typically accompany CPN. The kidney lesions observed were suggestive of a2µ-globulin nephropathy, a mechanism leading to xenobiotic-induced renal carcinogenesis which is specific to the male rat and not relevant to humans. There were exposure concentration-related increases in minimal to mild transitional epithelial hyperplasia in the renal pelvis of male rats, which were significant at 900 and 1800 ppm. There were also significant positive trends for adenomas, and adenomas or carcinomas (combined) in the 900 and 1800 ppm groups. A NOAEC of 450 ppm (1840 mg/m^3) was derived for neoplastic and non-neoplastic lesions, based on the non-neoplastic lesions observed in the kidneys at higher dose levels and the irrelevance to humans of the tumour types observed in the kidneys of male rats (REACH).

Reproductive and Developmental Toxicity

Results of reproductive and developmental toxicity studies conducted in animals indicate that the chemical or its metabolites are not expected to be specific reproductive or developmental toxins (OECD, 2005; REACH).

In a two-generation inhalation reproduction toxicity study (US EPA OPPTS Guideline 870-3800; OECD TG 416), rats were exposed (whole body) to the metabolite (MIBK) at 0, 500, 1000 and 2000 ppm (0, 2.04, 4.09 and 8.18 mg/L or 0, 2012, 4093

and 8178 mg/m^3) for six hours/day, seven days per week. There were no effects on reproductive parameters, offspring growth, and developmental landmarks at any exposure level. The NOAEC for parental systemic toxicity (apart from male nephropathy) was considered to be 1000 ppm (4.09 mg/L), based on transient reduced body weight gain and food consumption. The NOAEC for reproductive toxicity was considered to be 2000 ppm (8.18 mg/L), the highest concentration tested (OECD, 2005; REACH).

In another reproductive toxicity study (OECD TG 422), rats were treated with the metabolite (HMP) by oral route (gavage) for 45 days, with doses of 30, 100, 300, and 1000 mg/kg bw/d in distilled water, beginning 14 days prior to mating. Some effects in reproductive parameters (decreased fertility and implantations) and pup viability at the highest dose (1000 mg/kg bw/d) were seen in the presence of maternal toxicity (reduced weight gain, statistically significant changes in haematology, clinical biochemistry and relative organ weights; renal and hepatic histopathological lesions). A developmental and reproductive NOAEL of 300 mg/kg bw/d, based on reduced live pup births and other pup viability parameters and lower reproductive indices (fertility and implants) at 1000 mg/kg bw/d, were determined (OECD, 2005; REACH).

In a developmental toxicity study (OECD TG 414), female rats and mice were exposed (whole body) to vapours of the metabolite (MIBK) at 0, 300, 1000, or 3000 ppm (0, 1.23, 4.09, or 12.3 mg/L) (1229, 4106, 12292 mg/m^3) on gestational days six through 15, for six hours/day. Foetal toxicity was observed only in the presence of maternal toxicity. The NOAEC for maternal toxicity was 1000 ppm (4.09 mg/L) in both species, based on clinical signs of toxicity, increased kidney weights and decreased food consumption (rat only) and increased liver weights (mice only) at 3000 ppm. The NOAEC for foetotoxicity was 1000 ppm (4.09 mg/L) in both species, based on reduced foetal body weights, increased occurrence of retarded ossification, and an increased incidence of dead foetuses (mice only) at 3000 ppm. There were no developmental effects associated with the exposure to the metabolite at any concentration in both species and the NOAEL for developmental toxicity was 3000 ppm (12.3 mg/L) (OECD, 2005; REACH).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic acute effects (acute toxicity by the inhalation route of exposure) and local effects (skin, eye and respiratory irritation). Following repeated application of the chemical, severe drying of the skin with some sloughing and cracking could also occur.

Public Risk Characterisation

Although use in cosmetic products in Australia is not known, the chemical is reported to be used overseas in cosmetics (perfumes), where the general public may be exposed to the chemical through dermal and/or inhalation routes. Use concentrations in these products are not known; however the concentration of the chemical is not expected to be high when used as a perfume. Therefore, skin, eye and respiratory irritations are not expected from exposure to low concentrations of the chemical in cosmetic products.

Occupational Risk Characterisation

Given the critical health effects (systemic acute/local), the risk to workers from this chemical is considered low if adequate control measures to minimise occupational exposure (dermal, ocular and inhalation) to the chemical are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business at a workplace (such as an employer), has adequate information to determine appropriate controls.

NICNAS Recommendation

Assessment of the chemical is considered to be sufficient provided that the recommendation is adopted for the amendment of the classification and labelling of the chemical and all requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Regulatory Control

Public Health

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

Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted Globally Harmonized System of Classification and Labelling of Chemicals (GHS) as below. This does not consider classification of physical hazards and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful by inhalation (Xn; R20)	Harmful if inhaled - Cat. 4 (H332)
Irritation / Corrosivity	Irritating to eyes (Xi; R36) Irritating to skin (Xi; 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)

[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 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/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;
- 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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