# 2-Furancarboxaldehyde: Human health tier II assessment

**22 March 2013**

## CAS Number: 98-01-1



## 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.

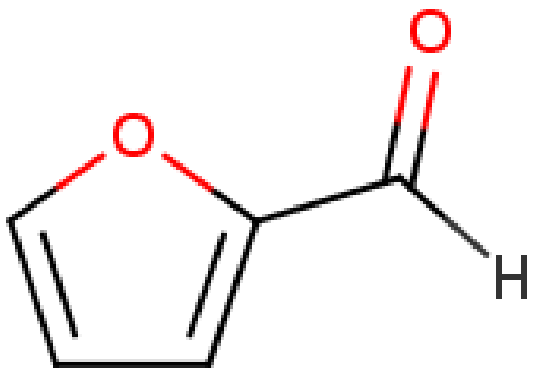
For more detail on this program please visit:www.nicnas.gov.au

**Disclaimer**

NICNAS has made every effort to assure the quality of information available in this report. However, before relying on it for a specific purpose, users should obtain advice relevant to their particular circumstances. This report has been prepared by NICNAS using a range of sources, including information from databases maintained by third parties, which include data supplied by industry. NICNAS has not verified and cannot guarantee the correctness of all information obtained from those databases. Reproduction or further distribution of this information may be subject to copyright protection. Use of this information without obtaining the permission from the owner(s) of the respective information might violate the rights of the owner. NICNAS does not take any responsibility whatsoever for any copyright or other infringements that may be caused by using this information.

Acronyms & Abbreviations

## Chemical Identity

| | |
|---|---|
| Synonyms | 2-Furaldehyde<br>Furfural<br>Furfuraldehyde<br>2-Furancarboxaldehyde<br>Furaldehyde |
| Structural Formula |  |
| Molecular Formula | C5H4O2 |
| Molecular Weight (g/mol) | 96.09 |
| Appearance and Odour (where available) | Colourless to yellowish liquid |
| SMILES | C1(C=O)=CC=CO1 |

# Import, Manufacture and Use

## Australian

No specific Australian use has been identified. However, the chemical is listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported introduction volume of less than 1000 tonnes.

## International

The following international uses have been identified via European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) Dossiers, the Organisation for Economic Cooperation and Development Screening information data set International Assessment Report (OECD SIAR), Galleria Chemica, Substances and Preparations In the Nordic countries (SPIN) database, the European Commission Cosmetic Ingredients and Substances (CosIng) database, United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary & eChemPortal (OECD High Production Volume chemical program (OECD HPV), the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR), and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB)):

The chemical has reported cosmetic use including:

- as an ingredient in fragrance compounds up to 0.1% (OECD, 2008); and
- as a solvent.

The chemical has reported domestic use including:

- in adhesive and binding materials; and
- in cleaners and detergents.

The chemical has reported commercial use including:

- as a solvent to refine lubricating oils;
- as an ion exchange agent;
- in shoe dyes; and
- as an analytical reagent.

The chemical has reported site-limited use including:

- manufacturing chemical products, refined petroleum products and nuclear fuel;
- production of lysine;
- refining rare earths and metals;
- as a wetting agent in manufacturing abrasive wheels and brake linings;
- as an intermediate for tetrahydrofuran and furfuryl alcohol, phenolic and furan polymers; and
- as an additive or polymer production aid.

The chemical has reported non industrial uses such as in food (at concentrations between 4.2 to 63 mg/kg) and in fungicides, insecticides and herbicides (SCCS, 2012).

# 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):

Xn; R40 (Carc. Cat. 3)

T; R23/25 (Acute toxicity)

Xn; R21 (Acute toxicity)

Xi; R36/37/38 (Irritation)

## Exposure Standards

### Australian

The chemical has an exposure standard of 7.9 mg/m³ (2 ppm) Time Weighted Average (TWA).

### International

The following are identified (Galleria Chemica):

An exposure limit (OEL, TWA, STEL, PEL or STV) of 5 - 20 mg/m³ (1.25 - 5 ppm) in Austria, Bulgaria, Canada, China, Colombia, Czech Republic, Denmark, Finland, Germany, Greece, Hungary, Iceland, Indonesia, Ireland, Italy, Japan, Korea (South), Latvia, Malaysia, Mexico, New Zealand, Nicaragua, Norway, Peru, Philippines, Poland, Portugal, Russia, Singapore, South Africa, Spain, Sweden, Switzerland, Taiwan and USA.

US DOE Temporary Emergency Exposure Limits (TEELs): 2 ppm (TEEL-0 and TEEL-1), 10 ppm (TEEL-2) and 100 ppm (TEEL-3).

# Health Hazard Information

## Toxicokinetics

The chemical is extensively absorbed and rapidly eliminated in humans after inhalation, and in rats after oral administration (SCCNFP, 2004).

Animal studies demonstrate that up to 85% of the orally administered dose in rats was detected in the urine within 24 h and 7% eliminated via exhalation as carbon dioxide. Metabolism was through oxidation or acetylation of the aldehyde group, followed by glycine conjugation. 2-Furoylglycine is the major urinary metabolite (~80% of the administered dose). Furoic acid, furanacrylic acid and furanacryluric acid were reported as minor metabolites (CICAD, 2000).

In humans, absorption of the chemical vapour via both the lungs and skin has been demonstrated. Metabolism appears similar to that in rats, with the majority of the retained dose being excreted as urinary 2-furoylglycine. Furoic acid and furanacryluric acid are also reported as minor metabolites. Dermal absorption from liquid furfural has also been observed (CICAD, 2000).

## Acute Toxicity

### Oral

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

Oral LD50 (mg/kg bw) = 65 (rat), 400 (mouse), 541 (guinea pig), 800 (rabbit) and 950 (dog) (ChemIDplus).

A number of studies involving repeated dosing of rats by the oral route used doses above the reported rat LD50 of 65 mg/kg bw. In these studies, mortalities were seen although not necessarily to the extent expected based on the reported LD50 value.

### Dermal

The chemical is classified as hazardous with the risk phrase ‘Harmful in contact with skin’ (Xn; R21) in HSIS (Safe Work Australia). Based on the data available (LD50 = 192 mg/kg bw in rat), the existing hazard classification should be amended to 'Toxic in contact with skin' (T; R24).

Dermal LD50 in rats = 192 mg/kg bw (US EPA, 2010). Clinical observations reported include abdominal breathing and nasal discharge in few animals. At gross necropsy, froth in the trachea, congestion of lungs and haemorrhage/oedema, enlarged spleen, petechiae (bleeding into the skin) in the thymus, distended urinary bladder and the hydrometra (uterus) were observed (US EPA, 2010).

When the undiluted chemical (at 45-1000 mg/kg bw) was applied to the shaved non-abraded skin of rabbits (occlusive) for a 48 h period, all rabbits died within 12 h in the 1000 mg/kg bw group (OECD, 2008).

Lowest lethal dermal dose (LDLo) in rabbits = 620 mg/kg bw (ChemIDplus).

### Inhalation

The chemical is classified as hazardous with the risk phrase ‘Toxic by inhalation’ (T; R23) in HSIS (Safe Work Australia). The data available support this classification.

Inhalation LC50 (6 h) = 175 ppm in rat, 350 ppm in mouse and 370 ppm in dog.

Human lowest toxic concentration (TCLo) = 0.31 mg/m³ (ChemIDplus).

## 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). The data available support this classification.

In a study where mice were exposed to the chemical, a rapid decrease in the respiratory rate was observed (RD50 (exposure concentration producing a 50% respiratory rate decrease) = 920 mg/m³ and 1128 mg/m³) (OECD, 2008).

In several repeated exposure studies respiratory tract irritation has been reported. Dose-related histopathological changes (such as focal atrophy of the olfactory epithelium) were reported in the nose of hamsters exposed to the chemical vapours at concentrations up to 2165 mg/m³ for 6 hours/day, 5 days/week for a period of 13 weeks. The NOAEL and LOAEL for local effects were 77 and 448 mg/m³ respectively (OECD, 2008).

### Skin Irritation

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

Reversible skin irritations were reported in guinea pigs after three daily 4-h dermal applications of the neat chemical. Only a very mild reaction was reported with 5% concentration (OECD, 2008).

The undiluted chemical (45-1000 mg/kg bw) applied to the shaved non-abraded skin of rabbits (occlusive) for 48 h caused mild local irritation at 45-500 mg/kg bw. All rabbits died within 12 h in the 1000 mg/kg bw group, but no evidence of irritation was observed at the site of application (OECD, 2008).

### Eye Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in HSIS (Safe Work Australia). The data available support this classification.

Slight oedema of the conjunctivae was reported after instillation of 0.001- 0.002 mL of undiluted chemical to the eyes of 15 male rabbits. Marked irritation with eyelid spasm was observed for about five days after exposure to 0.04 mL. The eyes appeared normal on day seven. Application of 0.09-1 mL of the chemical resulted in eyelid spasm for seven days with gross corneal opacity. The eyes appeared normal by day nine. The vapour of the chemical is reported to be irritating to the eyes of rabbits, but irritation scores are not available (OECD, 2008).

### Observation in humans

The main effect of the chemical in humans was reported to be skin and mucous membrane irritation (SCCNFP, 2004).

In humans, the chemical vapour (20-63 mg/m³) is reported to cause eye irritation (itching, burning, tearing and/or redness) and respiratory tract irritation (frequent nasal irritation such as stuffiness, dryness or soreness and sometimes dryness of the mouth or throat) (OECD, 2008).

## Sensitisation

### Skin Sensitisation

The chemical was reported to cause some slight positive skin reactions in the available guinea pig studies. The data available are not conclusive to warrant a hazard classification.

Three male Hartley guinea pigs received seven daily intradermal injections of 0.1 mL suspension containing 1% chemical (in saline with 1% Tween 80). Three weeks after the final induction, an intradermal challenge injection was administered (0.1 mL suspension of 0.25, 0.5 or

1.0% chemical in saline with 1% Tween 80). A positive skin reaction was reported after 24 h of challenge in one of the three guinea pigs challenged with the chemical at all concentrations (REACH, 2012).

In a Buehler test (with 10 male and 10 female Hartley Albino guinea pigs), very slight skin reactions were observed after challenge. As the reactions were also found in the negative controls (i.e. treated with the chemical in the induction phase, but not treated with the chemical in the challenge phase), it was stated that the chemical was not considered to be a skin sensitiser (REACH, 2012).

In a Maximisation test (with 10 male and 10 female Hartley guinea pigs), 0.2 mL of the undiluted chemical was used in the dermal induction. Intradermal induction was also conducted using 5% of the chemical in propylene glycol. Topical induction under occlusion for 48 h elicited very slight erythema in 13/20 animals and very slight oedema in 8/20 animals. Only slight skin reactions were observed in 2/20 animals after challenge and therefore, the chemical was not considered a skin sensitiser (REACH, 2012).

### Observation in humans

Chronic skin exposure in humans may produce eczema, allergic skin sensitisation and photosensitisation (OSHA).

There were cases of irritant dermatitis and eczema in humans. There were also reports of allergic skin sensitisation and photosensitisation (SCCNFP, 2004). The SCCS later reviewed the SCCNFP (2004) opinion on the chemical and reported that the chemical is not considered a human sensitiser (SCCS, 2012).

A maximisation test was conducted in 25 healthy male and female volunteers with the chemical at 2% concentration (five alternate days under occlusion): no sensitisation reactions were reported (SCCS, 2012; SCCNFP, 2004).

## Repeated Dose Toxicity

### Oral

Based on the data available, the chemical is not considered to cause serious damage to health by repeated oral exposure.

In an oral gavage study, groups of 20 male and female rats (F-344) were dosed 0, 11, 22, 45, 90 or 180 mg/kg bw/d, 5 d/week for 13 weeks. Mortalities were seen in groups dosed above the rat acute oral LD50 value of 65 mg/kg bw/day. Increased incidence of minimal to mild cytoplastic vacuolisation of hepatocytes was reported in all treated groups. There was no clear relationship between dose and incidence or severity of the observed effect. The reported no-observed-adverse effect level (NOAEL) for systemic toxicity is 45 mg/kg bw/d. This was based on significantly increased liver and kidney weights and cytoplastic vacuolisation of hepatocytes in exposed male rats at 90 mg/kg bw/d (US EPA, 2010).

In an oral gavage study, groups of 20 male and female (B6C3F1) mice were dosed 0, 75, 150, 300, 600 or 1200 mg/kg bw/d, 5 d/week for 13 weeks. Mortalities were seen in groups dosed above the mouse acute oral LD50 value of 400 mg/kg bw/d. Increased relative liver weights were observed in males at 300 mg/kg bw/d and in females at 75, 150 and 300 mg/kg bw/d. Centrilobular coagulative necrosis of hepatocytes was also observed at 300 mg/kg bw/d (1/10) and 150 mg/kg bw/d (1/10). Mild mononuclear inflammatory cell infiltration was seen in all treated groups. No NOAEL for systemic toxicity was established (US EPA, 2010).

### Dermal

Based on the limited data available, the chemical is not considered to cause serious damage to health by repeated dermal exposure.

In a 28 day study, the chemical was applied to the shaved skin of Wistar rats (10/sex/dose) at 0, 25, 50 and 100 mg/kg bw/d for 6 h/d, 5 d/week. No mortalities in any of the treated groups were reported. Female rats dosed at 100 mg/kg showed drowsiness, dyspnoea, clonic convulsion, hyperactivity and tremor after 3-4 h of dosing. No other treatment-related effects were reported (US EPA, 2010). Based on the information available, a NOAEL of 50 mg/kg bw/d can be established.

### Inhalation

From the repeat dose inhalation studies (28 days to 60 days or 13 weeks) available in rats, rabbits and hamsters, irritation effects to the respiratory tract were observed in all species. The rat seems to be the most sensitive species for respiratory tract effects with LOAELs established at 20 mg/m³. Based on the information available, the chemical is not considered to have high repeat dose inhalation toxicity, apart from severe respiratory tract irritation. Irritation effects were considered for hazard classification under respiratory irritation.

In a 28 day inhalation study, groups of rats were exposed to 0 or 160 mg/m³ (0 or 40 ppm), 1 h/d for 5, 15, or 30 days. Respiratory irritation, hyperplasia and degeneration of the olfactory epithelium, and lung congestion, oedema, and inflammation were observed at 160 mg/m³ (40 ppm) (CICAD, 2000).

In an inhalation study, a group of 10 Sprague Dawley rats was exposed to the chemical vapour in air (by nose-only exposure) at 0 (air), 2, 4, 8, or 20 mg/m³ (0, 0.002, 0.004, 0.007 or 0.017 mg/L respectively) for 6 h/d, 5 d/week for four weeks. There were no reported mortalities or clinical signs of toxicity. The NOAEL was 8 mg/m³ and LOAEL was 20 mg/m³ based on the incidence of microscopic lesions, i.e. transitional respiratory epithelial hyperplasia, and mixed inflammatory cell infiltration of the nasal cavity in males only (US EPA, 2010).

In another inhalation study, groups of five rats (F344) were exposed to chemical vapour (nose only exposure) at 0, 40, 80, 160 mg/m³ for 6 h/d, 5 d/week for 28 weeks and to 320, 640 and 1280 mg/m³ for 3 or 6 h/d, 5 d/week for 28 weeks. Due to excessive mortalities in the higher dose groups, additional groups at 20 mg/m³ were added to the study. Treatment related pathological effects were limited to the olfactory and respiratory epithelium of the nasal cavity. There was respiratory epithelial atypical hyperplasia and respiratory epithelial squamous metaplasia in all treated animals exposed for six hours. These effects were observed in some animals of the 3-h exposure groups. The severity of the damage was reported to be less intense in the 3-h exposure groups compared with the 6-h exposure groups. The LOAEL was 20 mg/m³ and no NOAEL was established (US EPA, 2010).

In a repeat dose inhalation study, groups of rabbits were exposed to 208, 520, or 1040 mg/m³ (52, 130, or 260 ppm) 4 h/d, 5 d/week, for at least 60 exposures. Respiratory irritation, hyperplasia and degeneration of the olfactory epithelium, and lung congestion, oedema, and inflammation were observed in the groups of rabbits exposed to 1040 mg/m³. There was also histopathological evidence of kidney damage and anaemia at 520 mg/m³. A NOAEL of 208 mg/m³ (52 ppm) was identified in rabbits (CICAD, 2000).

Groups of hamsters were exposed to the chemical at 0, 80, 460, or 2208 mg/m³ (0, 20, 115, or 552 ppm) 6 h/d, 5 d/week, for 13 weeks. The reported effects were respiratory irritation, hyperplasia and degeneration of the olfactory epithelium, lung congestion, oedema, and inflammation at 2208 mg/m³, and slight atrophy and hyperplasia of the olfactory epithelium at 460 mg/m³. A NOAEL of 80 mg/m³ (20 ppm) was identified (CICAD, 2000).

## Genotoxicity

Most of the in vitro assays gave positive results for genotoxicity. However, all in vivo genotoxicity studies (except for the studies in *Drosophila*, which were mostly at very high doses) showed negative results. Therefore, the chemical is not considered genotoxic.

Negative or weakly positive results have been reported for most in vitro genotoxicity bacterial tests. Positive results were obtained for reverse mutation in *Salmonella typhimurium* in a few studies at relatively high concentrations in the absence of metabolic activation (SCCS, 2012).

The chemical was genotoxic in cultured mammalian cells at the gene and chromosome level in the absence of metabolic activation. It induced sister chromatid exchange (SCE) in cultured Chinese hamster ovary (CHO) cells (11.7-3890 μg/mL) and human lymphocytes (3.5-14x10?² mM) (SCCS, 2012).

The chemical was not genotoxic in any in vivo assays in rats or mice. Negative results were reported for chromosome aberrations in mouse bone marrow cells (administered up to 200 mg/kg bw by a single intraperitoneal injection or in the diet at 4000 ppm for five days), SCE in mice (up to 200 mg/kg bw) or unscheduled DNA synthesis (UDS) in rats and mice (administered orally at 5, 16.7 or 50 mg/kg bw and 50, 175 or 320 mg/kg bw, respectively) (SCCS, 2012).

The chemical was genotoxic in *Drosophila* in somatic cells (wing spot test by inhalation at 3750-7500 ppm) and germ cells (sex-chromosome loss by injection at 100 ppm). The chemical did not induce reciprocal translocations and sex linked recessive lethal mutations (at 1000 ppm), with only a doubtful increase in one study in *Drosophila* (SCCS, 2012).

Six workers exposed to furfural in a furoic resin plant (exposure period not reported) showed no significant difference in sister chromatid exchange frequency in peripheral blood lymphocytes in comparison with six control individuals. The furfural concentrations in the atmosphere of the plant were not reported (SCCS, 2012).

### Carcinogenicity

The chemical is currently classified as a Category 3 carcinogen with the risk phrase 'Limited evidence of carcinogenic effect' (R40) in Australia (Safe Work Australia). The IARC monograph (1995) on the chemical states that 'There is inadequate evidence in humans for the carcinogenicity of furfural. There is limited evidence in experimental animals for the carcinogenicity of furfural. It is not classifiable as to its carcinogenicity to humans (Group 3)'. The data available support the existing classification.

Studies were conducted by administering the chemical (99% pure) in corn oil by gavage to groups of F344/N rats (0, 30, and 60 mg/kg bw/day) and B6C3F1 mice (0, 50, 100 and 175 mg/kg bw/day) for two years. Some evidence of carcinogenic activity of the chemical in male rats was reported based on the occurrence of uncommon cholangiocarcinomas in two animals, and bile duct dysplasia with fibrosis in two other animals. There was no evidence of carcinogenic activity in female rats that received doses up to 60 mg/kg bw/day. There was clear evidence of carcinogenic activity in male mice, based on increased incidences of hepatocellular adenomas and hepatocellular carcinomas. There was some evidence of carcinogenic activity in female mice, based on increased incidences of hepatocellular adenomas. Renal cortical adenomas or carcinomas in male mice and squamous cell papillomas of the forestomach in female mice could have been related to exposure to the chemical (IARC, 1995; OECD, 2008 and HSDB).

Induction of tumours in long term rodent studies is likely due to non-genotoxic mechanisms. New studies have indicated a threshold mechanism for carcinogenicity of the chemical (SCCS, 2012).

### Reproductive and Developmental Toxicity

Reproductive and developmental toxicity studies available in rats used doses above the acute oral LD50 value reported for rats. Based on the NOAEL for maternal toxicity (<50 mg/kg bw/d) and developmental toxicity (>150 mg/kg bw/d), the chemical is not considered a reproductive or developmental toxicant.

In 2-year gavage studies (5 d/week), no effects were found on the reproductive organs (epididymis, penis, preputial gland, prostate, seminal vesicles, testes, coagulating gland, clitoral gland, ovaries, uterus, vagina, and tissues from all endocrine glands) of both male and female F344/N rats up to 60 mg/kg bw/d and B6C3F1 mice up to 175 mg/kg bw/d (OECD, 2008).

In a developmental toxicity study (OECD TG 414), groups of 25 Sprague Dawley female rats were exposed to the chemical at 50, 100, and 150 mg/kg bw/d once daily by gavage from gestation day six through 15. High mortality was reported (>10%) in groups dosed above the rat acute oral LD50 value of 65 mg/kg bw. Clinical signs were observed in all treated dams beginning at one hour post dosing, and included tremors, bilateral exophthalmia (protrusion of the eyeball), head held low at 50 mg/kg bw/d, and hypoactivity, vocalisation, laboured respiration, rales, gasping and rapid respiration at 100 mg/kg bw/d. The NOAEL for maternal toxicity was considered to be less than 50 mg/kg bw/d based on clinical observations such as exophthalmia during gestation day 6-18 at all dose levels. No developmental effects were seen at any dose (US EPA, 2010 and SCCS, 2012).

In another developmental toxicity study, rabbits were orally dosed up to 225 mg/kg bw/d from gestation day 0 through 28. The only apparent effect of treatment was the unkempt appearance in 1/24 rabbits at 75 mg/kg bw/day for 8 days and in 6/25 rabbits at 225 mg/kg bw/d. There were no dead foetuses or premature deliveries. There were no treatment related external, visceral or skeletal malformations or variations. The rabbit maternal and developmental NOAELs were > 225 mg/kg bw/d (US EPA, 2010).

## Risk Characterisation

### Critical Health Effects

The main critical effects to human health are irritation to the respiratory system, eyes and skin, and acute toxicity from once only or short term exposure, and the potential for carcinogenicity from long term exposure.

### Public Risk Characterisation

Although use in cosmetic/domestic products in Australia is not known, the chemical is reported to be used in cosmetic/domestic products overseas. Given the potential use of this chemical in cosmetics, adhesive and binding materials, cleaning products and detergents, the public risk from this chemical is considered to require further management.

The chemical is reported to be used in cosmetic products overseas at concentrations up to 0.1%. In the European Union, the average maximum use level of furfural in food categories ranges from 4.2 to 63 mg/kg of food (OECD, 2008).

The Scientific Committee on Cosmetic Products and Non-food products (SCCNFP) is of the opinion that furfural can be safely used as a fragrance/flavour ingredient at a maximum concentration of 0.036% in the fragrance compound. The maximum concentration of furfural that can be safely used as a fragrance/flavour ingredient in toothpaste is 0.002% in the fragrance compound (SCCNFP, 2004).

A pragmatic concentration limit of 10 ppm in finished cosmetic products, including in oral products, is considered safe (SCCP, 2012).

### Occupational Risk Characterisation

Given the critical health effects, the risk to workers from this chemical is considered high if adequate control measures to minimise occupational exposure to the chemical are not implemented. 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

Further risk management is required. Sufficient information is available to recommend the chemical to be risk managed for public safety from its potential use in cosmetics and/or domestic products through scheduling, and occupational health and safety through classification and labelling.

### Regulatory Control

#### Public Health

The chemical is recommended for scheduling to mitigate risk from its use in cosmetic and domestic products.

Matters to be taken into considerations include the potential for carcinogenicity, irritation to skin, eye and to the respiratory system and acute toxicity of the chemical.

Maximum concentration limits recommended for safety are available from international documents (SCCNFP, 2004; OECD, 2008; SCCS, 2012).

#### Work 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 environmental hazards.

| Hazard | Approved Criteria (HSIS)[a] | GHS Classification (HCIS)[b] |
|---|---|---|

| Hazard | Approved Criteria (HSIS)[a] | GHS Classification (HCIS)[b] |
|---|---|---|
| Acute Toxicity | Toxic if swallowed (T; R25)* Toxic in contact with skin (T; R24) Toxic by inhalation (T; R23)* | Toxic if swallowed - Cat. 3 (H301) Toxic in contact with skin - Cat. 3 (H311) Toxic if inhaled - Cat. 3 (H331) |
| Irritation / Corrosivity | Irritating to 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) |
| Carcinogenicity | Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40)* | Suspected of causing cancer - Cat. 2 (H351) |

[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 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

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