Sorbic aldehyde: Human health tier II assessment

02 March 2018

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
2,4-Hexadienal, (E,E)-	142-83-6
2,4-Hexadienal	80466-34-8

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

Grouping Rationale

The chemical 2,4-hexadienal has isomers based on the configuration through the carbon-carbon double bond. The two main isomers are 2,4-hexadienal E,E (CAS No.142-83-6) and 2,4-hexadienal E,Z (CAS No. 53398-76-8, not listed on AICS). The commercially available 2,4-hexadienal (CAS No. 80466-34-8) is a mixture of the E,E (80 %) and E,Z (10–16 %) isomers. The E,E isomer (CAS No 142-83-6) and mixed isomers (CAS No. 80466-34-8) are assessed together in this report. Where data are not available for the mixed isomers, information on E,E isomer is used as a read across data for several toxicity endpoints.

Import, Manufacture and Use

Australian

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

International

The following international uses have been identified through: Galleria Chemica; the European Commission Cosmetic Ingredients and Substances (CosIng) database; the United States (US) National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments including from the International Agency for Research on Cancer (IARC) and the National Toxicology Program (NTP).

The commercially available chemical is synthetic, manufactured from acetaldehyde, while the E,E isomer is a naturally occurring volatile substance in plants, fruits and seafood.

The E,E isomer has reported cosmetic use as a fragrance ingredient. Under the International Nomenclature of Cosmetic Ingredients (INCI) the isomer is listed as '2,4-hexadienal', for perfuming functions.

The chemicals have reported site-limited uses, including as:

- intermediates in various organic synthetic reactions;
- corrosion inhibitors for steel used in oil fields; and
- monomers in reactions with silane comonomers for manufacturing polyalkenyloxysilane polymer.

The chemicals also have reported non-industrial uses, including as:

- raw materials in manufacturing food additives (e.g. sorbic acid, a widely used food preservative);
- components of insect repellents; and
- pharmaceutical intermediates for manufacturing mitomycins and antihypercholesterolaemics.

Restrictions

Australian

No known restrictions have been identified.

International

The International Fragrance Association (IFRA) has recommended that the chemicals be prohibited from being used in perfumes (IFRA, 2013).

Existing Worker Health and Safety Controls

Hazard Classification

None of the chemicals are listed on the Hazardous Chemical Information System (HCIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

The chemicals belong to the family of alpha,beta-unsatured aldehydes, which are direct-acting alkylating agents. As strong electrophilic reagents, the chemicals are expected to interact with DNA. E,E-sorbic aldehyde was shown to form adducts with deoxyguanosine (a pre-mutagenic DNA lesion) in vitro. The chemicals are also cytotoxic, possibly from decreasing membrane

lipid fluidity (NTP, 2003). The reactions of the aldehyde are expected to be mostly at the point of contact, forming products such as sorbic acid.

Toxicokinetics

No toxicokinetic data are available on the chemicals. However, similar aldehydes have been reported to be readily absorbed from the gastrointestinal tract (GIT), metabolised and excreted in the urine. In mammals, aldehydes are preferentially oxidised into corresponding acids before being excreted as such or as conjugates. The chemicals are expected to react quickly with blood components (NTP, 2003).

Acute Toxicity

Oral

The chemicals have moderate acute oral toxicity, based on a median lethal dose (LD50) value of 300 mg/kg bw in rats (NTP, 2003; HSDB; RTECS), warranting hazard classification.

Dermal

The chemicals have high acute dermal toxicity, warranting hazard classification (see **Recommendation** section).

An LD50 of 270 µL/kg bw was reported for rabbits (NTP, 2003; HSDB; RTECS), equivalent to about 240 mg/kg bw for the E,E isomer (pure chemical based on a density of 0.898 as reported in the HSDB). No details of the study were available.

Inhalation

No data are available.

Corrosion / Irritation

Corrosivity

No eye or skin irritation studies are available. The E,E isomer caused ulcers and inflammatory effects in the forestomach of rats and mice following repeated oral gavage administration (see **Repeated dose toxicity: Oral** section). The rodent forestomach has a similar lining to the human oesophagus. The oral exposure leads to increased contact time and potentially higher concentrations compared with inhalation. The available data are not sufficient to derive a conclusion on the irritation potential of the chemicals.

Sensitisation

Skin Sensitisation

The chemicals are considered to be skin sensitisers, warranting hazard classification (see **Recommendation** section).

A local lymph node assay (LLNA) following OECD Test Guideline (TG) 429 was conducted in female CBA mice by topically exposing them to 25 µL of the E,E isomer (in acetone and olive oil (AOO)) on both ears, for three consecutive days. The results indicated stimulation index (SI) values of 0.9, 1.5, 2.2, 4.2 and 14.8 at 0.5, 1, 2.5, 5 and 10 % of the chemical, respectively. The

effective concentration needed to produce a three-fold increase in lymphocyte proliferation (EC3) was calculated to be 3.5 %, indicating a moderate potential for skin sensitisation (Kern et al., 2010).

Repeated Dose Toxicity

Oral

Based on the available information, the chemicals are not considered to cause severe systemic effects following repeated oral exposure. However, the E,E isomer caused ulcers and inflammatory effects in the forestomach of rats and mice following gavage administration.

In a 16-day study, groups of Fischer 344/N (F344/N) rats and B6C3F1 mice (n=5/sex/dose) were administered the E,E isomer in corn oil by gavage doses of 0, 3, 9, 27, 80 or 240 mg/kg bw/day, for five days a week.

In rats given the highest dose, three males and three females died before the end of the study. Mean body weight gain was significantly lower at the highest dose, compared with the control group. Liver weight was significantly increased in females at the 240 mg/kg bw/day dose. Clinical signs at the highest dose included diarrhea, ataxia, lethargy and nasal/eye discharge in males, and lethargy, paleness and abnormal breathing in females. In the forestomach, ulceration was reported in rats at the highest dose, with a lesser incidence at the 80 mg/kg bw/day dose, but combined with mild to moderate epithelial hyperplasia.

In mice, one male and one female died at the highest dose before the end of the study, and clinical signs included lethargy and ruffled fur. Marked ulceration and/or necrosis of the forestomach were reported in all mice at the 240 mg/kg bw/day dose. At the 80 mg/kg bw/day dose there was minimal to mild epithelial hyperplasia and hyperkeratosis, but no forestomach effects were reported at lower doses (NTP, 2003).

In a 14-week study, groups of F344/N rats (n=10/sex/dose) were administered the E,E isomer in corn oil at doses of 0, 7.5, 15, 30, 60 or 120 mg/kg bw/day, five days per week. All animals survived until the end of treatment. Haematological effects, including minimal to mild decreases in leukocyte and lymphocyte counts and increases in neutrophil counts in animals dosed at 120 mg/kg bw/day, were considered as not toxicologically relevant, but rather as secondary effects to stress. Mean body weight and body weight gain were significantly lower at ≥30 mg/kg bw/day, compared with controls. At the highest dose, the incidences of epithelial hyperplasia, degeneration and chronic active inflammation of the forestomach in males and females and the incidences of nasal atrophy, osteofibrosis and exudate in males were significantly greater, compared with the control group. There were no significant effects on the weight or histology of reproductive organs at any dose. A no observed adverse effect level (NOAEL) of 60 mg/kg bw/day was established (NTP, 2003).

A 14-week study was conducted in B6C3F1 mice (n=10/sex/dose), orally dosed with the E,E isomer in corn oil at 0, 7.5, 15, 30, 60 or 120 mg/kg bw/day, for five days per week. Based on the increased incidences of epithelial hyperplasia of the forestomach in females and olfactory epithelial atrophy in males dosed at 120 mg/kg bw/day, a NOAEL of 60 mg/kg bw/day was established (NTP, 2003).

Although the forestomach is a common target tissue in rodent bioassays (NTP, 2003), it was reported that the E,E isomer could have had 'direct interaction with target-tissue macromolecules causing gene mutations or promotion of injury by reactive oxidative species (ROS) following glutathione depletion and/or stimulation of inflammation or inflammatory processes' (Chan et al., 2003).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

The available data are insufficient to conclude on the genotoxic potential of the chemical. Some DNA adduct formation (not statistically significant compared with controls) in the forestomach of rats was observed when the E,E isomer was repeatedly administered orally at 90 mg/kg bw/day. However, this was restricted to the site of contact. The IARC (2012) stated that a genotoxic mechanism could contribute to tumour induction.

The following in vitro genotoxicity data were available for the E,E isomer without further details (NTP, 2003; IARC, 2012):

- negative results in bacterial gene mutation assays in *Samonella typhimurium* strains TA98, TA100, TA1535 and TA1537, with or without metabolic activation;
- clear dose-responsive positive results over 0.1–1 µmol/plate in a preincubation test in *S. typhimurium* strain TA104 without metabolic activation;
- positive results in *S. typhimurium* strain TA100 at 0.010.75 µL/plate in a preincubation assay with or without metabolic activation;
- positive results in a mouse lymphoma cell mutation assay in L5178Y cells without metabolic activation;
- positive results for DNA strand breaks in L1210 mouse leukaemia cells;
- positive results for DNA oxidative damage in Chinese hamster lung fibroblasts V79 at 300 µM; and
- positive results for DNA damage in human epithelial colorectal adenocarcinoma cells.

The following in vivo genotoxicity data were available for the E,E isomer (NTP, 2003; IARC, 2012) :

- inconclusive results for the induction of micronucleated erythrocytes in rats and mice intraperitoneally (i.p.) injected with the chemical up to 200 and 160 mg/kg bw respectively;
- negative results for the induction of micronucleated normochromatic (mature) erythrocytes in peripheral blood samples obtained from male or female mice after 14 weeks' exposure to the chemical at 7.5–120 mg/kg bw/day by oral gavage;
- positive results for DNA adduct formation in the forestomach of rats orally exposed for 90 days to 90 mg/kg bw/day of the chemical, although the effect was not significant and no DNA adducts were detected in the rat liver; and
- no DNA adducts in the forestomach of mice orally exposed to the isomer.

Carcinogenicity

Based on the available data, the chemicals are considered to have carcinogenic potential, warranting hazard classification (see **Recommendation** section).

The IARC has classified the E,E isomer as ‘Possibly carcinogenic to humans’ (Group 2B), based on sufficient evidence for carcinogenicity in experimental animals. The IARC (2012) stated that 'there is moderate evidence that tumour induction occurs via a genotoxic mechanism'.

Studies in rats and mice showed that the E,E isomer induced forestomach squamous-cell neoplasms and squamous epithelial hyperplasia after oral exposure. These are usually rare spontaneous neoplasms in laboratory animals (IARC, 2012). However, the relevance of those results to humans was reported as questionable by the Joint FAO/WHO Expert Committee on Food Additives (JECFA), given the conditions of exposure used in these studies. They concluded that 'the results are due to the irritating effect of high bolus doses of trans,trans-2,4-hexadienal delivered to the contact site (the forestomach) by gavage and not the effects of systemic concentrations in the whole animal' (JECFA, 2004).

In a carcinogenicity study, groups of F344/N rats (n=50/sex/dose) received the E,E isomer in corn oil at gavage doses of 0, 22.5, 45 or 90 mg/kg bw/day, five days per week for two years. Survival was not affected by the treatment. The chemical induced significant increases in the incidence of squamous cell papilloma of the forestomach in the 45 and 90 mg/kg bw/day groups. The incidence of epithelial hyperplasia (focally extensive to diffuse thickenings of all layers of the squamous epithelium) in the

forestomach was significantly increased in all dose groups, with mild to moderate severity when dosed at 45 and 90 mg/kg bw/day, respectively. At the highest dose, males showed inflammation and cysts in the forestomach with a significantly greater incidence (NTP, 2003). Although the incidence of testes interstitial cell adenoma was significantly increased in males dosed at 90 mg/kg bw/day (41/50, 45/50, 45/50, 46/50), this effect was not considered biologically significant because of its occurrence at a high and variable rate in male F344/N rats. There was a dose-related increase in the incidence of splenic pigmentation in males (7/50, 9/50, 18/50, 20/50). The occurrence and incidence of squamous cell papilloma or adenoma of the tongue in most treated groups, the increased incidence of malignant phaeochromocytoma in the kidneys at 90 mg/kg bw/day and decreased incidences of pancreatic acinar cell adenoma (10/50, 5/50, 9/50, 6/50) and mononuclear cell leukaemia observed (11/50, 14/50, 9/50, 17/50) were not considered related to the chemical (NTP, 2003).

Groups of B6C3F1 mice (n=50/sex/dose) were administered the E,E isomer in corn oil at doses of 0, 30, 60 or 120 mg/kg bw/day, five days/week for two years. Survival was not affected by the treatment. The incidences of squamous cell papilloma and squamous cell papilloma or carcinoma (combined) in the forestomach were significantly greater in males dosed at 120 mg/kg bw/day and in females at 60 and 120 mg/kg bw/day. Non-neoplastic lesions of the forestomach consisted of squamous epithelial hyperplasia (significantly increased in females dosed at 60 mg/kg bw/day and in both sexes at 120 mg/kg bw/day) and ulcers in males (significantly increased at 120 mg/kg bw/day). Two male mice that were dosed at 120 mg/kg bw/day exhibited squamous cell carcinoma of the tongue, exceeding historical control incidence, and reported as possibly related to the treatment (NTP, 2003).

The NTP report (2003) concluded that rats and mice that received the E,E isomer 'had significantly greater occurrences of neoplasms of the forestomach. The forestomach in rodents is similar in tissue type to the oesophagus in humans. These tumors included papillomas and malignant carcinomas. Normally such tumors of the forestomach are rare in rodents'.

Reproductive and Developmental Toxicity

Only limited data are available on reproductive toxicity and no data are available on developmental toxicity of the chemicals. On the basis of the reactivity of the chemicals, only metabolites of lower toxicity are expected to be in systemic circulation.

In a 14-week repeat dose toxicity studies described earlier in rats and mice (see **Repeated dose toxicity: Oral** section), no significant effect in sperm parameters or oestrous cycles were observed (NTP, 2003).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- local effects (skin sensitisation);
- systemic long-term effects (carcinogenicity); and
- systemic acute effects from oral and dermal exposure.

Public Risk Characterisation

Given the IFRA restrictions on 2,4-dienal compounds in fragrance ingredients (prohibited use), it is unlikely that the public will be exposed to the chemicals from use as fragrance ingredients. The current controls are considered adequate to mitigate the risk to public health posed by fragranced products containing the chemicals.

The NTP report (2003) stated that 'Based on the large number of foods and food products that contain 2,4-hexadienal either naturally or as an additive, low level human exposure to this compound is widespread'. It is not expected that exposure to actual amounts of the chemical is a significant risk (JECFA, 2004).

Occupational Risk Characterisation

During product formulation, dermal and inhalation exposure may occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemicals at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical systemic long-term, systemic acute and local health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise dermal and inhalation exposure are implemented. The chemicals 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.

The data available support an amendment to the hazard classification in the HCIS (Safe Work Australia) (see **Recommendation** section).

NICNAS Recommendation

Assessment of these chemicals is considered to be sufficient, provided that the recommended amendment to the 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

Public Health

The chemicals are prohibited for use as fragrance ingredients according to IFRA standards.

Work Health and Safety

The chemicals are 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	Not Applicable	Harmful if swallowed - Cat. 4 (H302) Toxic in contact with skin - Cat. 3 (H311)
Sensitisation	Not Applicable	May cause an allergic skin reaction - Cat. 1 (H317)
Carcinogenicity	Not Applicable	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 industry

Control measures

Control measures to minimise the risk from oral and dermal exposure to the chemicals 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 that 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 chemicals, 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 chemicals.

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 chemicals has not been undertaken as part of this assessment.

References

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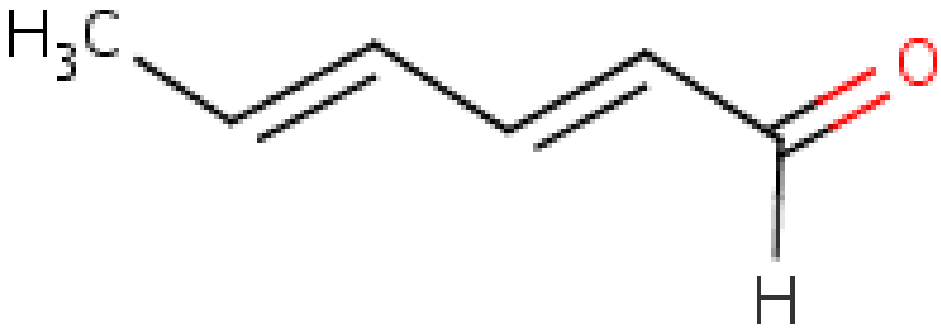
Last Update 02 March 2018

Chemical Identities

Chemical Name in the Inventory and Synonyms	**2,4-Hexadienal, (E,E)-** 2,4-hexadienal, (trans,trans)- 1,3-pentadiene-1-carboxaldehyde 2-propyleneacrolein sorbic aldehyde (E,E) sorbaldehyde
CAS Number	142-83-6
Structural Formula	

	
Molecular Formula	C6H8O
Molecular Weight	96.12

Chemical Name in the Inventory and Synonyms	**2,4-Hexadienal** sorbic aldehyde
CAS Number	80466-34-8
Structural Formula	

	
Molecular Formula	C6H8O
Molecular Weight	96.12

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