FOREWORD INTRODUCTION

2-PROPEN-1-OL

CAS N°: 107-18-6

SIDS Initial Assessment Report

For

SIAM 21

Washington DC, United States, 18-21October 2005

1. Chemical Name:	2-Propen-1-ol
2. CAS Number:	107-18-6
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5. Roles/Responsibilities of the Partners:	
• Name of industry sponsor /consortium	Allyl Alcohol Consortium
• Process used	Industry collected data, prepared the updated IUCLID dossier, and drafted versions of the SIAR and SIAP.
6. Sponsorship History	
• How was the chemical or category brought into the OECD HPV Chemicals Programme	2-Propen-1-ol is sponsored by Japan under the ICCA Initiative and is submitted for first discussion at SIAM 21.
7. Review Process Prior to the SIAM:	Japanese government peer-reviewed the documents and audited selected studies.

8. Quality check process: Japanese government peer-review committee performed spot checks on randomly selected endpoints and compared original studies with data in the SIDS Dossier.

9. Date of Submission: July 22, 2005

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11. Comments: None

SIDS INITIAL ASSESSMENT PROFILE

CAS No.	107-18-6
Chemical Name	2-Propen-1-ol
Structural Formula	**CH_2=CH-CH_2-OH**

SUMMARY CONCLUSIONS OF THE SIAR

Human Health

Animal studies demonstrate that 2-propen-1-ol appears to be oxidised readily in the liver, giving a variety of metabolic products, such as acrolein, acrylic acid, glycidaldehyde, and glyceraldehyde. Among these metabolites, the most reactive metabolite, acrolein may cause hepatotoxicity in the liver.

The inhalation LC_{50} is 140-150 mg/m^3 for 8 hours exposure in rats. The dermal LD_{50} (rabbit) is 89 mg/kg bw. The oral LD_{50} values are 70 and 99-105 in rats, 96 in mice and 71 mg/kg bw in rabbits. The intraperitoneal LD_{50} values are 37 and 42 in rats, and 60 mg/kg bw in mice. A 55-year old man died within 100 minutes of oral ingestion of 2-propen-1-ol. The amount ingested was assumed to be 212 g of 2-propen-1-ol at the maximum. Death was attributed to acrolein-induced cardiotoxicity.

2-Propen-1-ol is considered to be slightly irritating to the skin and irritating to eyes in animals. Moreover, 2-propen-1-ol may cause irritation of the eye and nasal mucosa in humans. 2-Propen-1-ol is considered not to be a skin sensitizer in guinea pigs [OECD TG 406].

In a repeat dose inhalation toxicity study, male rats were exposed to 2-propen-1-ol at nominal concentrations of 0, 2.4, 4.7, 12, 47, 95, 142, 237 or 355 mg/m^3 for 7 hours/day, 5 days/week for 12 weeks. Histopathology showed that there was slight congestion of the lungs and liver at the dose of 355 mg/m^3 (150 ppm). The NOAEL for inhalation toxicity in male rats is 12 mg/m^3 (5 ppm) based on a significant decrease in body weight gain in groups exposed to 47 mg/m^3 (20 ppm) and higher.
In a repeated dose oral toxicity study, 2-propen-1-ol had adverse effects on kidney tissues in rats, administered in the drinking water continuously for 15 weeks at or above a level of 100 ppm (8.3 mg/kg bw/day in males and 6.9 mg/kg bw/day in females). The NOAEL was 50 ppm of 2-propen-1-ol in drinking water (equivalent to 4.8 mg/kg bw/day in male rats and 6.2 mg/kg bw/day in female rats) based on adverse effects on kidney tissues (increases in absolute kidney weight and relative kidney weight) for females and on an increase in relative stomach weight for male and females at 100 ppm.

The *in vitro* studies, including reverse mutation assays in bacteria (*S. Typhimurium*: positive in T1535 with S9, TA100 without S9; negative in TA97, TA98, TA100 and TA1535 without S9), microbial forward mutation and fungal point mutation assays (*Streptomyces coelicolor* and *Aspergillus nidulans*, respectively: negative) and gene mutation in mammalian cells (V79 cells: positive) gave conflicting results, , while the *in vivo* studies concerning micronucleus and the dominant lethal assay in rodents gave negative results. Based on these data *in vitro* and *in vivo*, there is equivocal evidence that 2-propen-1-ol may be genotoxic.

A carcinogenicity study was conducted with male and female Fischer 344 rats via drinking water (300 mg/L, total dose of 3.2 g) for 106 weeks, followed by observation until natural death (123-132 weeks). The study gave no clear evidence of carcinogenicity in male rats, but there was equivocal evidence of carcinogenicity in the liver of female rats.

Reproductive/developmental toxicity was studied in SD rats by gavage at doses of 0, 2, 8 or 40 mg/kg bw/day [OECD TG 421]. Males were dosed from 14 days before mating for total of 42 days, and females were dosed from 14 days before mating throughout the mating and pregnancy period to day 3 of lactation. The autopsy was conducted on the day after the final administration. No deaths were found in any group. Clinical findings in parental animals at 40 mg/kg bw/day were salivation, decrease in locomotor activity, irregular respiration (male and female), lacrimation and loose stool (male). Histopathological examinations at 40 mg/kg bw/day revealed

atrophy of the thymus and hyperplasia of luteal cells in the ovary in females, necrosis, fibrosis, proliferation of bile duct, hypertrophy, and brown pigment deposition in perilobular hepatocytes, and diffuse clear cell changes in males and females, and hyperplasia of squamous epithelium in the forestomach in males. In male rats, no changes in histopathological findings or weight of the testes and epididymis were found. In females, extension of mean oestrous cycle length and increase in females with irregular oestrous cycle were observed at 40 mg/kg/day group. There were no adverse effects on the other reproductive performance parameters (such as the mating index, fertility index, numbers of corpora lutea or implantations, implantation index, delivery index, gestation index, gestation length, parturition or maternal behaviour). In examination of offspring, decrease in viability index on day 4 and total litter loss (from one dam) were observed at 40 mg/kg bw/day group. There were no-treatment-related findings in the external appearance, general conditions and necropsy findings in the offspring. The NOAEL is considered to be 8 mg/kg bw/day for general toxicity and reproductive/developmental toxicity.
In a prenatal developmental study conducted in SD rats, 2-propen-1-ol was administered by gavage at doses of 0, 10, 35, or 50 mg/kg bw/day to pregnant rats on gestation days 9 to19 [OECD TG 414]. At doses of 10 mg/kg bw/day and higher significant toxicity in dams was observed. Maternal toxicities at 35 and 50 mg/kg bw/day were mortalities, clinical findings, reductions in body weight gain and feed consumption, macroscopic liver findings and increased liver weights. One female at 10 mg/kg bw/day also had macroscopic liver findings. An increased frequency of total litter loss was observed at 35 and 50 mg/kg bw/day dose levels. In case of total litter loss, severe toxicities were observed in the dam (loss of body weight, severe decreases in feed consumption, and evidence of significant liver toxicity). Despite the severe maternal toxicity observed, there were no 2-propen-1-ol related increases in malformation rates or incidence of variations. 2-Propen-1-ol had no effects on intrauterine growth or survival in the fetuses from dams that survived to necropsy. Therefore, 10 mg/kg bw/day was considered to be the LOAEL for maternal toxicity, based on liver findings, and 10 mg/kg bw/day was considered to be the NOAEL for developmental toxicity, based on an increased frequency of total litter loss at 35 and 50 mg/kg bw/day, when 2-propen-1-ol was administered orally by gavage to pregnant rats.

Environment

2-Propen-1-ol is a colourless liquid and is miscible with water. Melting point, boiling point, vapour pressure and partition coefficient are -129 °C, 96.9 °C, 25 hPa (20 °C) and log Kow = 0.17, respectively. 2-Propen-1-ol is not expected to be hydrolyzed under normal environmental conditions. Indirect photo-oxidation by hydroxy radicals in the atmosphere is predicted to occur with a half-life of 4.32 hours. 2-Propen-1-ol is readily biodegradable under aerobic conditions within 14 days (BOD = 86 %). The estimated BCF is 3.2 and there is low potential for bioaccumulation. Fugacity Model Mackay level III calculations indicate that 2-propen-1-ol will be distributed mainly to air (67.6 %) water (25.1 %) and soil (7.3 %) compartment if released to air, while 2-propen-1-ol will stay exclusively in the water compartment (99.7 %) if released to water. If released to soil, 2-propen-1-ol will be distributed mainly to the water (19.4 %) and soil (80.4 %) compartment. If released simultaneously to air, soil and water, 2-propen-1-ol will be distributed mainly to water (62.1 %) and soil (36.7 %) compartment. Henry's Law constant is 4.99×10^{-6} atm.m^3/mole.

Acute toxicities to fish (96-h LC_{50}) are 0.59 mg/L (Medaka) [OECD TG 203] and 0.32 mg/L (Fathead minnow). Acute toxicity to *Daphnia magna* (48-h EC_{50}) is 2.1 mg/ L [OECD TG 202]. The 48-h LC_{50} in Polychaete (*Ophryotrocha diadema*) is 0.33-1.0 mg/ L. Acute toxicities to green algae (*Pseudokirchneriella subcapitata*) are 5.4 mg/L (72-h E_rC_{50}) and 2.3 mg/L (72-h E_bC_{50}) [OECD TG 201]. The NOEC of 21-d chronic toxicity in *Daphnia magna* is 0.92 mg/L [OECD TG 211]. The NOEC value in green algae (*Pseudokirchneriella subcapitata*) is 0.93 mg/L (72-h for growth rate and biomass) [OECD TG 201].

Exposure

The production volume of 2-propen-1-ol was estimated at 136,100 t/year worldwide in 2003 and 45,000 t/year in Japan in 2001. Two producers in Japan account for approx 30-40 % of global production. 2-Propen-1-ol is an important starting material, and is used in the manufacture of 1,4-butandeiol, 2-methyl-1,3-propandiol, allyl diglycol carbonate, diallyl phthalate, diallyl isophthalate, allyl glycidyl ether, epichlorohydrin, allyl methacrylate, styrene 2-propen-1-ol and resins for coating applications, flavorings such as allyl hexanoate, contact herbicide, as an intermediate for manufacturing pharmaceuticals, fire retardants and herbicides.

2-Propen-1-ol is exclusively used as an intermediate in chemical synthesis. Occupational exposure is possible by the inhalation and dermal routes at the manufacturing and user sites. No consumer use is known for 2-propen-1-ol. However, monitoring data provided by the sponsor country indicate that potential indirect exposure via the environment is anticipated.

Consumers may be potentially exposed to 2-propen-1-ol from ingestion of foods. 2-Propen-1-ol has been detected in crab meat, mussels and garlic. 2-Propen-1-ol is rapidly formed in the body from the hydrolysis of allyl esters used as flavour agents in food. The estimated intake of 2-propen-1-ol from this route is 18µg/kg bw/day in Europe and 5.8 µg/kg bw/day in the USA.

MOE, Japan monitored 2-propen-1-ol concentrations in the environment such as air, well water, sea water and river water throughout Japan. Based on these studies the estimated human exposure (EHE) is calculated to be 0.027 ug/kg bw/day under the standardised Japanese condition. A second Japanese monitoring study performed in the Kitakyushu-city area reported that no 2-propen-1-ol was detected in sea water, river water, reservoir water and effluent of sewage treatment plant in addition to well water, tap water and rain water at the limit of detection of 0.008ug/L.

RECOMMENDATION AND RATIONALE FOR THE RECOMMENDATION AND NATURE OF FURTHER WORK RECOMMENDED

Human Health: The chemical is a candidate for further work. The chemical possesses properties indicating a hazard for human health (acute toxicity, repeated dose toxicity, irritation, genotoxicity, carcinogenicity, reproductive/developmental toxicity). This chemical is manufactured in a closed system in Japan, but used to produce various products and occupational exposure through inhalation and dermal routes is possible in both production and user sites. Monitoring data provided by the sponsor country indicate that potential indirect exposure to consumers via the environment is anticipated. Therefore, an exposure assessment and, if necessary, risk assessment for workers and consumers should be performed.

Environment: The chemical is a candidate for further work. The chemical possesses properties indicating a hazard to the environment (acute toxicity in algae, fish and daphnia and chronic toxicity in daphnia). Based on data presented by the sponsor country (relating to production by two producers which account for approx 30-40 % of global production and relating to the use, the total reported releases and the transfers in the sponsor country), potential environmental exposure is anticipated. Therefore, member countries are invited to perform an exposure assessment, and if necessary, a risk assessment for the environment.

SIDS Initial Assessment Report

1 IDENTITY

1.1 Identification of the Substance

CAS Number: 107-18-6

IUPAC Name: 2-Propenol

Molecular Formula: C_3H_6O

Structural Formula: CH_2=CH-CH_2-OH

Molecular Weight: 58.08

Synonyms: Allyl alcohol
2-Propen-1-ol
2-Propenol
2-Propenyl alcohol
Propene-1-ol-3
Vinyl carbinol
Orvinylcarbinol
Propenyl alcohol
3-Hydroxypropene
Propenol
1-Propene-3-ol

1.2 Purity/Impurities/Additives

Purity: >99.0 % (w/w) in commercial product

Impurities: Acetic acid: ≤0.01 % (w/w).

Water: ≤0.3% (w/w).

1.3 Physico-Chemical properties

Physical-chemical properties are shown in Table 1.

Table 1 Summary of physico-chemical properties

Property	Value	Reference
Physical state	Liquid	Merck Index, 2001
Melting point	-129 °C	ICSC, 2000
Boiling point	96.9 °C	Kirk-Othmer Encyclopedia, 1983
Flash point	21.1 °C (open cup) 23.9 °C (closed cup)	Merck Index, 2001
Density	0.854 g/cm^3 (20 °C)	Merck Index, 2001
Vapour pressure	25 hPa (20 °C)	Kirk-Othmer Encyclopedia, 1983
Water solubility	Miscible	Merck Index, 2001
Partition coefficient n-octanol/water (log Kow)	0.17	ICSC, 2000
Flammability	Auto flammability (378 °C) Flammable	Merck Index, 2001 ICSC, 2000
Henry's Law constant	4.99 x 10^{-6} atm.m^3/mole	Hine and Mookerjee, 1975
Appearance	Color: colorless Odor: pungent, mustard-like odor	Merck Index, 2001

1.4 Category Justification

Not applicable

2 GENERAL INFORMATION ON EXPOSURE

2.1 Production Volumes and Use Pattern

The production volume of 2-propen-1-ol was estimated at 136,100 t/year worldwide [Lyondell Chemical Company, 2003] and 45,000 t/year in Japan in 2001 [MOE, Japan, 2004]. Two producers in Japan account for approx 30-40 % of global production. 2-Propen-1-ol is an important starting material, and is used in the manufacture of a wide range of chemicals, as shown in Table 2 as well as an intermediate for manufacturing pharmaceuticals, fire retardants and herbicides [Lyondell Chemical Company, 2003; Showa Denko K.K., 2004].

Table 2 2-Propen-1-ol products and their uses

Product Name	CAS No.	Use
1,4-butanediol	110-63-4	optical resins
2-methyl-1,3-propanediol	2163-42-0	optical resins
diallyl diglycol carbonate	142-22-3	optical resins
diallyl phthalate	131-17-9	plasticizer
diallyl isophthalate	1087-21-4	plasticizer
allyl glycidyl ether	106-92-3	silane coupling agents for a multitude of applications (such as water treatment and glass adhesion)
epichlorohydrin	106-89-8	manufacture of epoxy resins and synthetic glycerol and epichlorohydrin elastomer
allyl methacrylate	96-05-9	resins for coating applications
styrene allyl alcohol	25119-62-4	resins for coating applications
allyl hexanoate	123-68-2	flavorings

2.2 Environmental Exposure and Fate

2.2.1 Sources of Environmental Exposure

2-Propen-1-ol is used as a raw material exclusively and converted to other compounds. 2-Propen-1-ol may enter the environment at the production site and at chemical industries manufacturing the downstream products. In addition, 2-propen-1-ol in itself is not expected to be used as a commercial product and will not directly enter into the environment through consumer products.

2-Propen-1-ol is used as a starting material to produce a wide range of chemicals which are then further converted to products which consumers may be exposed to. It is anticipated that unreacted 2-propen-1-ol will be removed from the chemical products during purification. Therefore, the level of residual 2-propen-1-ol in final products would be expected to be negligible.

There are two manufacturing sites in Japan, Oita Plant of Showa Denko K.K. and Ohtake Plant of Daicel Chemical Industries, Ltd. 2-Propen-1-ol is classified as a Class I compound of PRTR law in Japan, and all users and manufacturers have to report the amount of release and transport into the environment.

The reported releases and transfers of 2-propen-1-ol from users and manufacturers throughout Japan in 2003 are summarised by Japanese Ministry of Environment [MOE Japan, 2005] and are shown in Table 3.

Table 3 Total reported releases and transfers of 2-propen-1-ol to PRTR law in Japan (2003)

Total Reported Releases (kg)				
Air	Water	Soil	Landfill	Total
7,280	11,248	0	0	18,528

Total Reported Transfers (kg)			Total Reported Releases and Transfers (kg)
Waste transfers	Transfers into sewage	Total	
157,450	550	157,950	176,478

In addition, MOE, Japan monitored 2-propen-1-ol in air (5 points, 1995), well water (15 points, 2001), seawater (11 points, 2001) and river water (65 points, 2001) throughout Japan. The monitored 2-propen-1-ol concentrations and the resulting Estimated Human Exposure (EHE) are shown in Table 4 [MOE, Japan, 2004]. Thus, EHE as average exposure to 2-propen-1-ol is calculated to be 0.027 ug/kg bw/day under the standardized Japanese conditions.

Another Japanese study reported the monitoring of 2-propen-1-ol in the environment in Kitakyushu-city area [Kadokami and Sato, 1993] and showed that no 2-propen-1-ol was detected in seawater (9 points), river water (10 points), reservoir water (2 points), and effluent of sewage treatment plant (2 points) in addition to well water, tap water and rain water (each one point) with a detection limit of 0.008ug/L.

Table 4 Total daily exposure of 2-propen-1-ol to humans

		Concentrations	Average exposure (ug/kg bw/day)
Air	Environmental air	0.05ug/m^3	0.015
	Room air	-	-
Water	Drinking water	-	-
	Well water	<0.3ug/L	0.012
	Public water	<0.3ug/L	(0.012)
Food		-	-
Soil		-	-
Total oral exposure		-	0.012
Total exposure		-	0.027

Note 1) Underlined values show that exposure amounts were regarded as 'less than minimum detection limit'

2) A value in brackets is not used for calculation of total oral exposure.

3) Calculated by using these parameters, respiratory volume: 15 m^3, daily water intake: 2L, daily food intake: 2000kg, body weight: 50kg.

Chemical profiles by Scorecard ED in USA reported the largest releases by transfers of 2-propen-1-ol to EPA Toxics Release Inventory (TRI) and the data are shown in Table 5.

Table 5 Total reported releases and transfers of 2-propen-1-ol in USA (2002)

Total Reported Releases (kg)				
Air	Water	Land	Environment from facilities	Total
65,621	6,320	86	301,912	373,939

Total Reported Transfers and Wastes (kg)			Total Reported Releases, Transfers and Wastes (kg)
Off-site transfers	Production-related Wastes	Total	
1,527,258	4,812,953	6,340,211	6,714,150

2.2.2 Photodegradation

A half-life of 4.32 hours and rate constant of $2.59x10^{-11}$ cm^3/molecule-sec has been obtained for the indirect photo-oxidation of 2-propen-1-ol with hydroxyl radicals ($1x10^6$ OH/ cm^3) in air, while reaction with ozone yielded a removal half-life of 5.52 hours (100 ppb ozone), with formaldehyde, hydroxyacetaldehyde and a monofunctional carbonyl moiety formed as reaction products [Grosjean et al., 1993]. Alternatively, using AOPWIN v1.91, a calculated half-life of 4.32 hours and rate constant of $2.97x10^{-11}$ cm^3/molecule-sec were obtained for reaction of 2-propen-1-ol with hydroxyl radicals ($1.5x10^6$ OH/cm^3) in air, while reaction with ozone yielded a removal half-life of 22.92 hours [Allyl Alcohol Consortium, 2005c].

2.2.3 Stability in Water

2-Propen-1-ol is expected to be stable in water because it contains no functional groups that are susceptible to hydrolysis. [Lyman et al., 1990].

2.2.4 Transport between Environmental Compartments

Fugacity Model Mackay level III calculations [Allyl Alcohol Consortium, 2005a] indicate that 2-propen-1-ol will be distributed mainly to air (67.6%), water (25.1 %) and soil (7.3 %) compartment if released to air, while 2-propen-1-ol will stay exclusively in the water compartment (99.7 %) if released to water. If released to soil, 2-propen-1-ol will be distributed mainly to the water (19.4 %) and soil (80.4 %) compartment. If released simultaneously to air, soil and water, 2-propen-1-ol will be distributed mainly to water (62.1 %) and soil (36.7 %) compartment. Data are shown in Table 6.

Table 6 Environmental distribution of 2-propen-1-ol using Mackay Level III fugacity model

Compartments	Release 100% to Air (1000kg/h)	Release 100% to Water (1000kg/h)	Release 100% to Soil (1000kg/h)	Equal emission scenario to each compartment (1:1:1)
Air	67.6 %	0.1 %	0.1 %	1.0 %
Water	25.1 %	99.7 %	19.4 %	62.1 %
Soil	7.3 %	0.0 %	80.4 %	36.7 %
Sediment	0.1 %	0.3 %	0.1 %	0.2 %

2.2.5 Biodegradation

2-Propen-1-ol is readily biodegradable under aerobic conditions within 14 days (BOD = 86 %) [OECD TG 301C] [MITI Japan, 1992].

2.2.6 Bioaccumulation

An estimated BCF of 3.2 was calculated by BCFWIN v2.14 using the experimental log Kow value of 0.17 [Allyl Alcohol Consortium, 2005b]. Therefore, there is low potential for bioaccumulation.

2.2.7 Other Information on Environmental Fate

An estimated Koc value of 1.32 by PCKOCWIN v1.66 indicates that 2-propen-1-ol is not expected to adsorb to suspended solids and sediments [Allyl Alcohol Consortium, 2005d]. 2-Propen-1-ol has half-lives of 10.2 and 9.5 days at 20 °C with Texas soil (pH 7.8, 3.25 % organic matter) and Mississippi soil (pH 4.8, <1 % organic matter), respectively [Loehr, 1989]. Leaching of 2-propen-1-ol from soil surface to deeper layers was found to rank in order of sand > sandy loam > humus sand. The percent leached after 2 days and 400 mL of water with sand containing 0.51% and 2.89% organic matter were 100 and 83.3%, respectively [Scheunert, et. al., 1981]. Volatilization from water surface is expected based upon a Henry's Law constant of 4.99 x 10^{-6} atm.m^3/mole.

2.3 Human Exposure

2.3.1 Occupational Exposure

2-Propen-1-ol is produced in a closed system. The atmospheric concentration was measured at one production site in Japan [JISHA, 2004]. The monitored data are shown in Table 7.

Table 7 Work place monitoring data for 2-propen-1-ol

Operation	n	Monitoring data		S.D.	Frequency	Working time hrs/day
		(Mean concentration) (ppm)	(Maximum concentration) (ppm)			
Sampling in operation	7	0.08	0.37	0.13	1 time/day	0.5
Analytical work	2	0.02	0.02	-	1 time/day	0.5
Sampling products	4	0.71	1.94	0.90	1 time/day	0.17
Drum filling	4	0.05	0.12	0.05	10-20 times/month	5

[Monitoring method] Air sample was suctioned at the breathing zone of the worker at the suction rate of 0.2 L/min. and adsorbed through a collection tube and analyzed by GC.

Mean concentrations of 2-propen-1-ol in the production site are less than 1 ppm (2.4 mg/m^3, Japanese OEL).

The official recommendation (for example TLV; Threshold Limit Values) for 2-propen-1-ol is established in OECD countries. For example, the 0.5 ppm (1.2 mg/m^3) to skin as TLV-TWA (Time Weighted Average) value is recommended by ACGIH [ACGIH, 2002]. The 1 ppm (2.4 mg/ m^3) is adopted in the sponsor country. Olfactory recognition level ranges from 0.78 (1.87 mg/m^3) to 25 ppm (60 mg/m^3) and eye irritation occurs at 25 ppm (60 mg/m^3) [Dunlap et al., 1958].

The OEL and other regulations for 2-propen-1-ol in several countries are summarized in Table 8 [RTECS, 2005].

Table 8 Standards and regulations for 2-propen-1-ol

Standards and regulations	Country	Adopted Value
MSHA (STANDARD-air)	USA	TWA 2 ppm (5 mg/m^3), Skin
OSHA PEL (general industry, construction, shipyards and federal contractors)	USA	8H TWA 2 ppm (5 mg/ m^3), Skin
OEL	ARAB Republic of Egypt	TWA 2 ppm (5 mg/ m^3), Skin
OEL	AUSTRALIA	TWA 2 ppm (5 mg/ m^3), STEL 4 ppm, Skin
OEL	AUSTRIA	MAK 2 ppm (5 mg/ m^3), Skin
OEL	BELGIUM	TWA 2 ppm (4.8 mg/ m^3), STEL 4 ppm (9.5 mg/ m^3), Skin
OEL	DENMARK	TWA 2 ppm (5 mg/ m^3), Skin
OEL	FINLAND	TWA 2 ppm (5 mg/ m^3), STEL 4 ppm (10 mg/ m^3), Skin
OEL	FRANCE	VME 2 ppm (5 mg/ m^3), VLE 4 ppm (10 mg/ m^3), Skin
OEL	GERMANY	MAK 2 ppm (5 mg/ m^3), Skin
OEL	THE NETHERLANDS	MAC-TGG 5 mg/ m^3, Skin
OEL	JAPAN	OEL 1 ppm (2.4 mg/ m^3), Skin
OEL	NORWAY	TWA 2 ppm (5 mg/ m^3)
OEL	THE PHILIPPINES	TWA 2 ppm (5 mg/ m^3), Skin
OEL	POLAND	MAC(TWA) 2 mg/ m^3, MAC(STEL) 10 mg/ m^3, Skin
OEL	RUSSIA	TWA 1 ppm
OEL	SWEDEN	NGV 2 ppm (5 mg m^3), KTV 6 ppm (14 mg/ m^3), Skin
OEL	SWITZERLAND	MAK-W 2 ppm (5 mg/ m^3), KZG-W 4 ppm (10 mg/ m^3), Skin
OEL	TURKEY	TWA 2 ppm (5 mg/ m^3), Skin
OEL	UNITED KINGDOM	TWA 2 ppm (4.8 mg/ m^3), STEL 4 ppm (9.7 mg/ m^3), Skin

Note:
MSHA: Mine Safety and Health Administration
OEL: Occupational Exposure Limit
OSHA: Occupational Safety and Health Administration
PEL: Permissible Exposure Limit

Occupational exposures at production sites may occur by the inhalation and dermal routes. Normally, workers wear impervious clothing, gloves, face shields, and other appropriate protective clothing necessary to prevent any possibility of skin contact with 2-propen-1-ol according to the plant regulations. In addition, employees are required to use splash proof goggles and face masks when handling liquid 2-propen-1-ol [Showa Denko MSDS, 2003].

2.3.2 Consumer Exposure

Production and use of 2-propen-1-ol in the manufacture of esters for use in resins and plasticizers, and as an intermediate in the production of pharmaceuticals and other organic chemicals may result in its release to the environment through various waste streams. As consumer products containing 2-propen-1-ol are not known at the present time, consumers are expected not to be exposed to 2-propen-1-ol via this route. However, consumers may be exposed to 2-propen-1-ol through the environment by ambient air.

Consumers may be potentially exposed to 2-propen-1-ol by ingestion of foods. When the volatile components of foods was investigated, 2-propen-1-ol was detected at concentrations of 0.3 ug/kg dry weight in the legs and claws, and 0.1 ug/kg dry weight in the body meat of crabs (Charybdis feriatus) [Chung, 1999], 1.08 ug/g wet weight in rotten mussels [Yasuhara, 1987] and in crushed garlic as a volatile component. Concentrations in garlic ranged from 0.11-121ug/g garlic depending on the method used to remove the garlic oils from the water phase [Yu TH et al., 1989]. 2-Propen-1-ol is rapidly formed in the body from the hydrolysis of allyl esters used as flavour agents in food. The estimated intake of 2-propen-1-ol from this route is 18 ug/kg bw/day in Europe and 5.8 ug/kg bw/day in the USA [WHO, 1997].

3 HUMAN HEALTH HAZARDS

3.1 Effects on Human Health

3.1.1 Toxicokinetics, Metabolism and Distribution

There are many studies concerning the hepatotoxicity and mechanistic action of 2-propen-1-ol. Only a few typical studies are cited in this section.

2-Propen-1-ol is metabolized rapidly after administration *in vivo*. When rats were administered orally with 120 mg/kg bw of 2-propen-1-ol, the concentration in blood was 9-15 ug/mL between 15 and 120 minutes after administration. In the meantime, when rats were administered intravenously with 30 mg/kg bw of 2-propen-1-ol, the concentration in blood was 24 ug/mL at several minutes and decreased to 4 ug/mL at 15 minutes after administration. At one hour after administration, 2-propen-1-ol almost disappeared from the blood. When 2-propen-1-ol was administered intravenously on a continuous basis, it disappeared in blood at a rate of approximately 23 mg per hour [Kodama and Hine, 1958].

Similarly, Penttila et al. (1988) reported that periportal and perivenous cells isolated from rat liver oxidized 2-propen-1-ol at rates of 3.4 and 3.1 μmol/(g.min), respectively. Cellular GSH was rapidly depleted (95%) by oxidation of 700 uM 2-propen-1-ol.

Patel et al. (1980) investigated the biotransformation of 2-propen-1-ol in rat liver and lung preparations. 2-Propen-1-ol was metabolized to acrolein by alcohol dehydrogenase in liver and cytosolic fractions but not in liver microsomes or in lung preparations (where alcohol dehydrogenase is absent). They showed that the reaction is alcohol dehydrogenase-dependent as it was significantly inhibited by pyrazole, a known inhibitor of alcohol dehydrogenase. Acrolein was oxidized to acrylic acid by liver aldehyde dehydrogenase in the presence of NAD+ or NADP+ in liver, cytosolic and microsomal fractions. Incubation of 2-propen-1-ol and acrolein with liver and lung microsomes in the presence of NADPH resulted in the formation of glycidol and glycidaldehyde, respectively. Epoxide production proceeded rapidly in the first few minutes but

levels then fell. This may be explained by epoxide hydrase activity in lung and liver microsomes which converts the epoxides to glycerol and glyceraldehydes, respectively.

Reid (1972) reported that pretreatment of rats with 4-methylpyrazole blocked the hepatic necrosis due to ^{14}C-2-propen-1-ol and reduced the amount of radiolabeled material bound in the liver by 80%. He concluded that the binding of ^{14}C-2-propen-1-ol to macromolecules (via sulfhydryl groups) and the subsequent necrosis were dependent on the oxidation of 2-propen-1-ol to acrolein.

Kaye (1973) reported the isolation of 3-hydroxypropyl mercapturic acid (the end product of conjugation to glutathione) from the urine of rats after subcutaneous injection of either 2-propen-1-ol or acrolein (6.3% and 10.5% conversion, respectively). Sanduja et al. (1989) investigated the excretion in urine of 3-hydroxypropyl mercapturic acid in rats given 2-propen-1-ol (64 mg/kg bw) or acrolein (13 mg/kg bw) by gavage. Recoveries were 28.3% and 78.5%, respectively.

Hormann et al. (1989) investigated the time course of 2-propen-1-ol induced toxicity in isolated rat hepatocytes. They observed an initial rapid depletion of glutathione (GSH), a subsequent increase in malondialdehyde (MDA) and decrease in protein sulfhydryl groups (PSH) and the eventual loss of membrane integrity. Addition of sulfhydryl compounds (N-acetylcysteine and dithiothreitol) markedly delayed the depletion of GSH, prevented significant loss of PSH and protected the cells against viability loss. In contrast, antioxidants (butylated hydroxytoluene and Trolox C) and the iron chelating agent desferoxamine suppressed 2-propen-1-ol induced MDA production without affecting the depletion of cellular thiols or the loss of viability. These results suggest that the inactivation of protein thiol groups is critical for 2-propen-1-ol toxicity, whereas lipid peroxidation is not essential to the toxic process.

3.1.2 Acute Toxicity

There are various studies on the acute toxicity by different administration routes. The available data are shown in Table 9.

Table 9 Acute toxicity of 2-propen-1-ol

Route	Species	Result	Reference
Inhalation	Rat (Long-Evans, male)	LC_{50} = 1900-2130 mg/m^3 (1 hr-exposure) LC_{50} = 300 -330 mg/m^3 (4-hr exposure) LC_{50} = 140-150 mg/m^3 (8-hr exposure)	Dunlap et al., 1958
Dermal	Rabbit (albino, male)	LD_{50} = 89 mg/kg bw	Dunlap et al., 1958
Oral	Rat (Long-Evans, male)	LD_{50} = 99-105 mg/kg bw	Dunlap et al., 1958
	Mouse (Swiss Webster, male)	LD_{50} = 96 mg/kg bw	
	Rabbit (albino, male)	LD_{50} = 71 mg/kg bw	
	Rat (Osborne-Mendel, combined)	LD_{50} = 70 mg/kg bw	Jenner et al., 1964
Intraperitoneal	Rat (Long-Evans, male)	LD_{50} = 42 mg/kg bw	Dunlap et al., 1958
	Mouse (Swiss Webster, male)	LD_{50} = 60 mg/kg bw	
	Rat (Fischer 344, male)	LD_{50} = 37 mg/kg bw	Klinger et al., 1986

Studies in Animals

Inhalation

Rats (Long-Evans; 6 males/group) were exposed for 1, 4 or 8 hours to 2-propen-1-ol at concentrations between 95-5450 mg/m^3 (nominal). Coma and diarrhoea were observed. On histopathological examination, toxicity in the lung (congestion), liver (congestion and necrosis) and kidneys (heme casts and cloudy swelling) was observed. The LC_{50} for 1, 4 and 8 hours were 1900-2130, 300-330 and 140-150 mg/m^3, respectively [Dunlap et al., 1958].

Dermal

Rabbits (3 males/group) were exposed for 24 hours to 25 - 200 mg/kg bw of 2-propen-1-ol (applied by injecting the 2-propen-1-ol though the occlusive dressing onto the surface of the skin and sealing the puncture site with rubber cement). Apathy, along with flushing of the skin, ataxia and diarrhoea were observed. On histopathological examination, toxicities in the lungs (congestion), liver (congestion and necrosis) and kidneys (heme casts and cloudy swelling) were observed [Dunlap et al., 1958]. The LD_{50} was 89 mg/kg bw.

Oral

Dunlap et al. (1958) studied the oral toxicity of 2-propen-1-ol in several species. Rats (Long-Evans; 5 males/group), mice (Swiss Webster; 6 males/group), and rabbits (albino; 3 males/group) were dosed by gavage with graded amounts of a 1% (rats and mice) or a 2% (rabbits) solution of 2-propen-1-ol, respectively. Apathy, ataxia, attitude of bellicosity, flushing of skin, diarrhoea were observed as major clinical signs. The common gross findings in rats and rabbits that died were oedema and congestion of the lungs (confirmed microscopically), visceral congestion, and discoloured livers, some with necrotic areas. The common toxicity in each test was observed in

livers (congestion and necrosis of periportal sinusoids, central pallor and necrosis). The LD_{50} values in rats, mice and rabbits were 99-105, 96 and 71 mg/kg bw, respectively. In addition, Jenner et al. (1964) reported a LD_{50} of 70 mg/kg bw in rats.

Other Routes of Exposure

Regarding the acute toxicity by intraperitoneal administration, three values were reported in rats and mice. The LD_{50} values were 37 and 42 mg/kg bw for rats, and 60 mg/kg bw for mice [Dunlap et al., 1958; Klinger et al., 1986].

Studies in Humans

Oral

Oral ingestion of 2-propen-1-ol (estimated maximum of 212g) by a 55-year-old man resulted in death within 100 minutes. At autopsy, bloody, reddish fluid was found in the mouth, larynx, oesophagus, and trachea. The mucous membranes of the trachea, stomach, and duodenum were congested and inflamed. The stomach contained a pungent green-black fluid, and all internal organs exhibited a strong pungent odour. Total amounts of 2-propen-1-ol in gastric contents, bile, and urine were 3.6 g, 15 mg, and 0.5 mg, respectively. The concentration in blood was 309 mg/L. Acrolein was not detected in gastric contents and only in small amounts in bile and urine. The concentration of acrolein in blood was 7.2 mg/L. Death was attributed to acrolein-induced acute cardiotoxicity [Toennes et al., 2002].

Conclusion

The inhalation LC_{50} is 140-150 mg/m^3 for 8-hr exposure in rats. The dermal LD_{50} (rabbit) is 89 mg/kg bw. The values reported for oral LD_{50} in rats, mice and rabbits are 70 and 99-105, 96 and 71 mg/kg bw, respectively. The values reported for intraperitoneal LD_{50} are 37 and 42 mg/kg bw in rats and 60 mg/kg bw in mice, respectively. A 55-year old man died within 100 minutes of oral ingestion of 2-propen-1-ol. Death was attributed to acrolein-induced cardiotoxicity.

3.1.3 Irritation

The available data are shown in Table 10.

Table 10 Irritation of 2-propen-1-ol

Route	Species	Exposure Time/ Method	Result	Reference
Skin	Rabbit	Occlusive, 24-hr	Slightly irritating	Dunlap et al., 1958
Eye	Rabbit	4-hr / Directive 84/449/EEC, B.5	Irritating	Jacobs and Martens, 1989
	Rabbit	4-96-hr/ OECD 405	Irritating	Jacobs, 1992
	Rabbit	48-hr / Draize test	Irritating	Dunlap et al., 1958

Skin Irritation

Studies in Animals

Undiluted 2-propen-1-ol (0.5 mL) was applied occlusively to the intact and abraded skin of 3 male rabbits for 24 hrs. Slight erythema was observed only at the site of one animal, while no other reactions were noted. Under these test conditions, 2-propen-1-ol was considered to be slightly irritating to the skin [Dunlap et al., 1958].

Eye Irritation

Studies in Animals

Three reports were available. Although test conditions were a little different in each test, the results of each test showed conjunctival and corneal damage [Dunlap et al., 1958; Jacobs and Martens, 1989; Jacobs, 1992]. 2-Propen-1-ol was unanimously irritating to the eyes of rabbits.

Studies in Humans

Groups of 24 volunteers were exposed to 2-propen-1-ol from one to three times a week for exactly five minutes. During the observation time they noted the degree of subjective response at one-minute intervals. There were no cases of pulmonary discomfort or noticeable effect on the central nervous system. Eye irritation, occurring immediately, was not more than slight until the level of 25 ppm (60 mg/ m^3) was reached, although irritation to nasal mucosa was regarded as at least moderate by four of seven subjects at 12.5 ppm (30 mg/ m^3). Olfactory cognition was checked off as more than moderate only at 6.25 ppm (15 mg/ m^3), and by only two of the five subjects [Dunlap et al. (1958)].

Respiratory Tract Irritation

Studies in animals

2-Propen-1-ol induced a decrease in the respiratory rate due to sensory irritation in mice. The effects were very rapid, normally reaching a plateau within the first 10 minutes. After exposure, the sensory irritating responses died away very rapidly. No pulmonary irritation was observed at the concentrations causing a 50% decrease in respiratory rate (RD50) in mice. The RD50 values within the first 10 minutes and for the mean value of the period from 21 to 30 minutes due to sensory irritation were calculated to be 9.24 mg/m^3 (3.9 ppm) and 11.4 mg/ m^3 (4.8 ppm), respectively [Nielsen et al., 1984].

Studies in Humans

See Section 3.1.3 Irritation - Eye Irritation - *Studies in Humans*

Conclusion

2-Propen-1-ol is considered to be slightly irritating to the skin and irritating to eyes in animals. Moreover, 2-propen-1-ol may cause irritation of the eye and nasal mucosa in humans.

3.1.4 Sensitisation

Studies in Animals

Skin

In a guinea pig maximisation test (OECD TG 406, GLP) a group of 20 male guinea pigs were induced intracutaneously using 1% w/v 2-propen-1-ol in water, followed by 7 days later by topical induction using 2.5% w/v 2-propen-1-ol in water which was applied over the sites of the intracutaneous injections and covered occlusively for 48 hours. Challenge was carried out 21 days after the first induction. Undiluted 2-propen-1-ol (0.1 mL) was applied to the shaven backs of the test animals and covered with occlusive tape for 24 hours. Dermal reaction to the challenge was assessed at 24 and 48 hours after challenge. None of the test animals showed any positive response at either 24 or 48 hours after removal of the challenge patches and therefore 2-propen-1-ol is not considered to be a skin sensitiser in guinea pigs [Allyl Alcohol Consortium, 2004c].

Conclusion

2-Propen-1-ol is not considered to be a skin sensitiser in guinea pigs.

3.1.5 Repeated Dose Toxicity

There are five studies available on repeated dose toxicity by inhalation and oral routes. The data are shown in Table 11. Moreover, repeated dose studies in rats and mice have been conducted recently by NTP but no detailed reports are available currently.

Studies in Animals

Table 11 Repeated dose toxicity of 2-propen-1-ol

Route	Species	Dose level	Exposure Time	Effects observed	NOAEL/ LOAEL	Reference
Inhalation	Rat (Long-Evans) male	2.4-355 mg/m^3 (1-150 ppm)	12 weeks, 7 hours/ day, 5 days/ week	Decreases in body weight gain, increases in lung and kidney weights, increases in clinical signs, lesions and microscopic findings	NOAEL (decreases of body weight): 12 mg/m^3 (5 ppm) LOAEL: 47 mg/m^3 (20 ppm)	Dunlap et al., 1958

Oral	Rat (Long-Evans) male/female	1-1,000 ppm in drinking water	13 weeks, continuous	Increases in relative kidney (both sexes) and liver weights (males)	NOAEL: 100ppm (11.6 mg/kg bw/day (male), 13.2 mg/kg bw/day (female)) LOAEL: 250 ppm (25.5 mg/kg bw/day (male), 34 mg/kg bw/day (female))	Dunlap et al., 1958
Oral	Rat (Wistar) male/female	50-800 ppm in drinking water	15 weeks, continuous	Increases in relative kidney weight (200 ppm and above for males, 100 ppm and above for females), decrease in water intake (in all treated groups) and body weight (200 ppm and above for males and 800 ppm for females)	NOAEL: 50ppm (4.8 mg/kg bw/day in male, 6.2 mg/kg bw/day in female)	Carpanini et al., 1978
Oral	Rat (Sprague-Dawley) male/female	0-40 mg/kg bw/day by gavage	Males, 42 days. Females, from 14 days before mating to day 3 of lactation	Bile duct proliferation (males and females), atrophy of thymus and hyperplasia of luteal cells in ovary (females) at 40 mg/kg bw/day	NOAEL: 8 mg/kg bw/day (male and female)	Allyl Alcohol Consortium, 2004b
Oral	Rat (Sprague-Dawley) female	0-50 mg/kg bw/day by gavage	gestation days 9 to19	mortalities, clinical findings, reductions in body weight gain and feed consumption and increased liver weights (35 mg/kg bw/day and above) macroscopic liver findings (10 mg/kg bw/day and above)	LOAEL: 10 mg/kg bw/day	Lyondell Chemical Company, 2005b

Inhalation

Rats (Long-Evans; 10 males/group) were exposed to 2-propen-1-ol at nominal concentrations of 0, 2.4, 4.7, 12, 47, 95, 142, 237 or 355 mg/m^3 (1, 2, 5, 20, 40, 60, 100 and 150 ppm) for 7 hours/day, 5 days/week for 12 weeks [Dunlap et al., 1958]. Four rats from the 355 mg/m^3 (150 ppm) group died during the first exposure period, two rats were dead by the following morning and two rats died during the second exposure period. The remaining two rats from this group died by the 10th exposure period (end of second week). The livers of the rats appeared to be hemorrhagic, and the lungs were pale and spotted. The kidneys were normal. The enteric tract was usually bloated with air and mucous, and the enteric vessels were engorged. There were six deaths in animals exposed to 237 mg/m^3 (100 ppm) (time period inadequately characterized), and one rat death following four exposures to 142 mg/m^3 (60 ppm) 2-propen-1-ol. Clinical signs in the 355 mg/m^3 group included

gasping, severe depression, nasal discharge, eye irritation and corneal opacity. Similar but less intense clinical signs were present in animals exposed to 95-237 mg/m^3 (40-100 ppm). No clinical signs were present in animals exposed to 47 mg/m^3 (20 ppm). At 47 mg/m^3 (20 ppm) and below, the rats behaved normally, and showed no gross or microscopic lesions. Body weight gain was significantly decreased in groups exposed to 47 mg/m^3 (20 ppm) and higher. Relative lung and kidney weights were significantly increased at 95 mg/m^3 (40 ppm) and 142 mg/m^3 (60 ppm), respectively.

From this study in rats exposed to 2-propen-1-ol over 12 weeks, the NOAEL and LOAEL were found to be 12mg/m^3 (5 ppm) and 47mg/m^3 (20 ppm) in terms of decreases of body weight gain, respectively.

Oral

Four studies were available.

Dunlap et al. (1958) reported results for male and female Long-Evans rats exposed to 2-propen-1-ol via drinking water for 13 weeks, at concentrations of 0, 1, 5, 100, 250, 500, and 1,000 ppm. The concentrations were equivalent to 0, 0.13, 0.62, 5.9, 11.6, 25.5, 41.0, and 72.0 mg/kg bw/day for males, and 0, 0.17, 0.94, 7.3, 13.2, 34.0, 43.7, and 67.4 mg/kg bw/day for females. Pale discolouration of the liver, with a soft spongy, yellowish appearance, was noted in two females on 72.0 mg/kg bw/day (1,000 ppm). Microscopically these livers showed well-localized necrosis with regeneration. The appearance and distribution of the lesions suggested infarction. Water intake decreased in all treated groups in a dose-related manner, presumably reflecting unpalatability of the dosing solutions. Body weight gain was significantly decreased in both sexes ingesting doses of 500 ppm and above, with a dose-related increase in relative kidney weight (significant at 250 ppm and above in both sexes) and relative liver weight (significant at 250 and 1,000 ppm only in males). The possible confounding effects due to decreased water intake cannot be excluded. Under the conditions of this study, the NOAEL for oral toxicity of 2-propen-1-ol in drinking water was considered to be 100 ppm (11.6 mg/kg bw/day for males, 13.2 mg/kg bw/day for females), based on the increases in relative kidneys and liver weights.

In a study by Carpanini et al. (1978), male and female Wistar rats were given 2-propen-1-ol via drinking water for 15 weeks, at concentrations of 0, 50, 100, 200, 800 ppm. The concentrations were equivalent to 0, 4.8, 8.3, 14.0, and 48.2 mg/kg bw/day for male, and 0, 6.2, 6.9, 17.1, and 58.4 mg/kg bw/day for females. Water intake and urine concentrating ability decreased in the groups given 2-propen-1-ol in a dose-related manner, with significant reductions in body weight and food intake in males at 200 ppm and above, and females at 800 ppm group. The majority of these findings may appear secondary to a reduction in water intake that was particularly pronounced in high dose animals. Increases in absolute kidney weight (at 100 ppm and above for females), relative kidney weight (at 200 ppm and above for males, and 100 ppm and above for females), and relative stomach weight (at 100 ppm and above for males and 100 and 800 ppm for females) were observed. Local irritation (stomach) or dehydration (kidney) may have contributed in part to these findings. These results revealed that 2-propen-1-ol administration in drinking water at or above a level of 100 ppm (a mean 2-propen-1-ol intake of 8.3 mg/kg bw/day in males and of 6.9 mg/kg bw/day in females) has adverse effects on kidney tissues in the rats. Therefore, the NOAEL established in this study was 50 ppm of 2-propen-1-ol in the drinking water (a level equivalent to a mean intake of 4.8 mg/kg bw/day in male rats and 6.2 mg/kg bw/day in female rats).

In a reproduction/developmental toxicity screening test, conducted according to OECD TG 421 [Allyl Alcohol Consortium, 2004b], SD rats (12 animals/sex/group) were administered 2-propen-1-ol given by gavage at doses of 0 (vehicle; water for injection), 2, 8 or 40 mg/kg bw/day. Males were dosed for a total of 42 days before mating, and females were dosed from 14 days before

mating throughout the mating and pregnancy period to day 3 of lactation. The autopsy was conducted on the day after the last administration. No deaths were found in any group. Salivation, decreased locomotor activity, irregular respiration in males and females, and lacrimation and loose stools in males were observed at 40 mg/kg bw/day. No effects of 2-propen-1-ol on body weight and food consumption were detected. Evidence of general toxicity in parent animals at 40 mg/kg bw/day included rough surface of the liver, thickening of forestomach and limiting edge of the stomach in males, enlargement, yellowish patches and rough surface of the liver in females. Histopathological examinations revealed atrophy of the thymus and hyperplasia of luteal cells in the ovary in females at 40 mg/kg bw/day. In livers, necrosis, fibrosis, proliferation of bile duct, hypertrophy, and brown pigment deposition in perilobular hepatocytes, and diffuse clear cell changes in males and females, and hyperplasia of squamous epithelium in the forestomach in males dosed at 40 mg/kg bw/day.

In a prenatal developmental study conducted in SD rats, 2-propen-1-ol was administered by gavage at doses of 0, 10, 35, or 50 mg/kg bw/day to pregnant rats on gestation days 9 to19 [OECD TG 414]. At doses of 10 mg/kg bw/day and higher significant toxicity in females was observed. Toxicities at 35 and 50 mg/kg bw/day were mortalities, clinical findings, reductions in body weight gain and feed consumption, macroscopic liver findings and increased liver weights. One female at 10 mg/kg bw/day also had macroscopic liver findings. Therefore, 10 mg/kg bw/day was considered to be the LOAEL for female toxicity, based on liver findings [Lyondell Chemical Company (2005b)].

Conclusion

The NOAEL for inhalation toxicity in male rats is considered to be 12 mg/m^3, and the NOAEL for oral toxicity in rats is 4.8 mg/kg bw/day for males and 6.2 mg/kg bw/day for females.

3.1.6 Mutagenicity

Ten studies were available. These are seven *in vitro* test reports and three *in vivo* test reports, and these data are shown in Table 12-1 and 12-2.

Table 12-1 Genotoxicity studies in vitro

Type of test	Test system	Dose	Result	Reference
Bacterial test (reverse mutation)	*S. typhimurium* TA98, TA100, TA1535, TA1537, TA1538 with and without hamster S9	10-500 ug/plate	Positive in TA1535 with S9	Lijinsky and Andrews, 1980
Bacterial test (reverse mutation)	*S. typhimurium* TA100 with and without S9	without S9 0.05-0.55 umol/2mL incubation volume (equivalent to approx. 2 - 32 ug/2mL) with S9 0.05 - 0.7 umol/2mL incubation volume (equivalent to approx. 2 - 41 ug/2mL)	Positive in TA100 without S9	Lutz et al., 1982
Bacterial test (reverse mutation)	*S. typhimurium* TA97, TA98, TA100, TA1535	0.3-166 ug/plate or 3-333 ug/plate	Negative	NTP, 1995

	with and without rat or hamster S9			
Bacterial test (reverse mutation)	*S. typhimurium* TA98, TA100, TA1535, TA1537, TA1538 with and without S9	Spot test (0.05 uL/plate) or plate incorporation assay (0.025, 0.05, 0.10 uL/plate)	Negative	Principe et al., 1981
Bacterial test (forward mutation)	*Streptomyces coelicolor* Resistance to streptomycin	Spot test (100 uL /plate) or plate incorporation assay (2-100 uL/plate)	Negative	Principe et al., 1981
Fungi test (point mutation)	*Aspergillus nidulans* Resistance to 8-azaguanine	Spot test (20 uL/plate) or plate incorporation assay (10-40 uL/plate)	Negative	Principe et al., 1981
Mammalian cells test (gene mutation)	V79 cell Resistance to 6-thioguanine	1 or 2 uM (equivalent to 58 or 116 ng/mL)	Positive	Smith et al., 1990

Table 12-2 Genotoxicity studies in vivo

Type of test	Test system	Dose	Result	Reference
Micronucleus assay	Rats (Fischer 344) Bone marrow	5-20 mg/kg bw/day, i.p., 3 consecutive days	Negative	NTP,1994
Micronucleus assay	Mouse ($B6C3F_1$) Peripheral blood	3-50 mg/kg bw/day, oral gavage, 13 weeks	Negative	NTP,1995
Dominant lethal test	Rat (Sprague-Dawley)	25 mg/kg bw/day, 33 weeks	Negative	Jenkinson and Anderson, 1990

In vitro Studies

There were four bacterial reverse mutation studies and three other mutation studies. Regarding bacterial reverse mutation studies using *S. typhimurium*, two positive (TA100 without metabolic activation and TA1535 with metabolic activation) and two negative results were obtained, depending on the study. As for the other mutation studies, conflicting results (one positive mammalian gene mutation assay and two negative microbial mutation assays) were reported.

In vivo Studies

Two micronucleus studies and one dominant lethal study *in vivo* are available. In contrast to the *in vitro* studies, all three studies gave negative results. A NTP study showed that 2-propen-1-ol induced no micronuclei in femoral bone marrow up to 20 mg/kg/bw/day after three consecutive days, when administered intraperitoneously in male rats. Another NTP study reported that 2-propen-1-ol induced no micronuclei in peripheral blood, when examined retrospectively, following oral administration of up to 50 mg/bw/day for 13 weeks in male and female mice. In addition, the dominant lethal study showed that 2-propen-1-ol did not induce dominant lethal effects in male SD rats given 25 mg/kg bw.

Conclusion

The *in vitro* studies including reverse mutation studies in bacteria and mammalian cells gave conflicting results (positive or negative), while the *in vivo* studies concerning micronucleus and the

dominant lethal study in rodents gave negative results. Based on the mutagenicity data *in vitro* and *in vivo*, there is equivocal evidence indicating that 2-propen-1-ol may be genotoxic.

3.1.7 Carcinogenicity

Studies in Animals

Oral

A carcinogenicity study was conducted with male and female Fischer 344 rats given 2-propen-1-ol via drinking water (300 mg/L, total dose of 3.2 g) for 106 weeks, followed by observation until natural death (123-132 weeks) [Lijinsky and Reuber, 1987]. The tumours observed in liver, adrenal cortex, pituitary and leukaemia were stated to be of the types commonly seen in untreated F344 rats. The occurrence of specific tumour types increased after treatment with other substances included in this study, the increased occurrence of hepatic nodules/carcinoma in females given 2-propen-1-ol relative to that present in the controls is not the subject of discussion. When evaluated in combination with the liver effects found in other studies, the increased occurrence of hepatic nodules/carcinoma in females is considered to be biologically significant and may indicate equivocal evidence for carcinogenicity.

Conclusion

There is no clear evidence of carcinogenicity in male rats, but there was equivocal evidence of carcinogenicity in the liver of female rats.

3.1.8 Toxicity for Reproduction

Studies in Animals

Effects on Fertility

A reproduction/developmental toxicity screening test was conducted according to the OECD TG 421 [Allyl Alcohol Consortium, 2004b]. SD Rats (12 animals/sex/group) were administered 2-propen-1-ol given by gavage at doses of 0 (vehicle; water for injection), 2, 8 or 40 mg/kg bw/day. Males were dosed for a total of 42 days before mating, and females were dosed from 14 days before mating throughout the mating and pregnancy period to day 3 of lactation. The autopsy was conducted on the day after the last administration.

No deaths were found in any group. Salivation, decreased locomotor activity, irregular respiration in males and females, and lacrimation and loose stools in males were observed at 40 mg/kg bw/day. No effects of 2-propen-1-ol on body weight and food consumption were detected. Necropsy examinations revealed no abnormal findings in offspring at all doses. Evidence of general toxicity in parent animals at 40 mg/kg bw/day included rough surface of the liver, thickening of forestomach and limiting edge of the stomach in males, enlargement, yellowish patches and rough surface of the liver in females. Histopathological examinations revealed atrophy of the thymus and hyperplasia of luteal cells in the ovary in females at 40 mg/kg bw/day. In livers, necrosis, fibrosis, proliferation of bile duct, hypertrophy, and brown pigment deposition in perilobular hepatocytes, and diffuse clear cell changes in males and females, and hyperplasia of squamous epithelium in the forestomach in males dosed at 40 mg/kg bw/day.

In male rats, no changes in histopathological findings or weight of the testes and epididymis were found. In females, extension of mean oestrous cycle length and increase in females with irregular oestrous cycle were observed at 40 mg/kg bw/day group. There were no adverse effects on the

other reproductive parameters (such as the mating index, fertility index, numbers of corpora lutea or implantations, implantation index, delivery index, gestation index, gestation length, parturition or maternal behaviour).

Developmental Toxicity

A reproduction/developmental toxicity screening test was conducted according to OECD TG 421, as described above [Allyl Alcohol Consortium, 2004b]. On examination of offspring, a decrease in viability index on day 4 and total litter loss (from one dam) were observed in the 40 mg/kg bw/day group. External examination of offspring revealed no external morphological abnormalities. Adverse effects on offspring may be influenced by parental toxicity.

In a prenatal developmental study conducted in SD rats, 2-propen-1-ol was administered by gavage at doses of 0, 10, 35, or 50 mg/kg bw/day to pregnant rats on gestation days 9 to19 [OECD TG 414]. At doses of 10 mg/kg bw/day and higher significant toxicity in dams was observed. Maternal toxicities at 35 and 50 mg/kg bw/day were mortalities, clinical findings, reductions in body weight gain and feed consumption, macroscopic liver findings and increased liver weights. One female at 10 mg/kg bw/day also had macroscopic liver findings. An increased frequency of total litter loss was observed at 35 and 50 mg/kg bw/day dose levels. In case of total litter loss, severe toxicities were observed in the dam (loss of body weight, severe decreases in feed consumption, and evidence of significant liver toxicity). Despite the severe maternal toxicity observed, there were no 2-propen-1-ol related increases in malformation rates or incidence of variations. 2-Propen-1-ol had no effects on intrauterine growth or survival in the fetuses from dams that survived to necropsy. Therefore, 10 mg/kg bw/day was considered to be the LOAEL for maternal toxicity, based on liver findings, and 10 mg/kg bw/day was considered to be the NOAEL for developmental toxicity, based on an increased frequency of total litter loss at 35 and 50 mg/kg bw/day, when 2-propen-1-ol was administered orally by gavage to pregnant rats [Lyondell Chemical Company (2005b)].

Conclusion

In a reproduction/developmental toxicity screening test, the NOAEL was considered to be 8 mg/kg bw/day for general toxicity and reproductive/developmental toxicity. Adverse effects on offspring may be influenced by parental toxicity.

In a prenatal developmental study, the LOAEL for maternal toxicity was considered to be 10 mg/kg bw/day, based on liver findings and the NOAEL for developmental toxicity was considered to be 10 mg/kg bw/day based on an increased frequency of total litter loss at higher doses.

3.2 Initial Assessment for Human Health

Animal studies demonstrate that 2-propen-1-ol appears to be oxidised readily in the liver, giving a variety of metabolic products, such as acrolein, acrylic acid, glycidaldehyde, and glyceraldehyde. Among these metabolites, the most reactive metabolite, acrolein may cause hepatotoxicity in the liver.

The inhalation LC_{50} is 140-150 mg/m^3 for 8-hr exposure in rats. The dermal LD_{50} (rabbit) is 89 mg/kg bw. The oral LD_{50} in rats, mice and rabbits are 70, 96 and 71 mg/kg bw, respectively. The intraperitoneal LD_{50} in mice and rats are 60 and 37 mg/kg bw, respectively.

2-Propen-1-ol is considered to be slightly irritating to the skin and irritating to eyes in animals. Moreover, 2-propen-1-ol may cause irritation of the eye and nasal mucosa in humans. 2-Propen-1-ol is considered to not be a skin sensitizer in guinea pigs.

In a repeat dose inhalation toxicity study male rats were exposed to 2-propen-1-ol at nominal concentrations of 0, 2.4, 4.7, 12, 47, 95, 142, 237 or 355 mg/m^3 for 7 hours/day, 5 days/week for 12 weeks. Histopathology showed that there was slight congestion of the lungs and liver at the dose of 355 mg/m^3 (150 ppm). The NOAEL for inhalation toxicity in male rats is 12mg/m^3 (5 ppm) based on retardation of body weight gain.

In a repeat dose oral toxicity study, 2-propen-1-ol administered in the drinking water continuously for 15 weeks at or above a level of 100 ppm (8.3 mg/kg bw/day in males and of 6.9 mg/kg bw/day in females) had adverse effects on kidney tissues in the rats. The NOAEL was 50 ppm of 2-propen-1-ol in drinking water (equivalent to 4.8 mg/kg bw/day in male rats and 6.2 mg/kg bw/day in female rats) based on adverse effects on kidney tissues.

The *in vitro* studies including reverse mutation studies in bacteria and mammalian cells gave conflicting results (positive or negative), while the *in vivo* studies concerning micronucleus and the dominant lethal study in rodents gave negative results. Base on the mutagenicity data *in vitro* and *in vivo*, 2-propen-1-ol has equivocal evidence of genotoxicity.

A carcinogenicity study was conducted with male and female Fischer 344 rats via drinking water (300 mg/mL, total dose of 3.2g) for 106 weeks, followed by observation until natural death (123-132 weeks). There is no clear evidence of carcinogenicity in male F344 rats given 2-propen-1-ol in drinking water for 106 weeks, but equivocal evidence of carcinogenicity was seen in the liver of female rats.

Reproductive/developmental toxicity was studied in SD Rats (12 animals/sex/group), administered 2-propen-1-ol by gavage at doses of 0 (vehicle; water), 2, 8 or 40 mg/kg bw/day. Males were dosed for total of 42 days before mating, and females were dosed from 14 days before mating throughout the mating and pregnancy period to day 3 of lactation. The autopsy was conducted on the day after the final administration.

No deaths were found in any group. Necropsy examinations revealed no abnormal findings in offspring at all doses. Histopathological examinations revealed atrophy of the thymus and hyperplasia of luteal cells in the ovary in females at 40 mg/kg bw/day. Necrosis, fibrosis, proliferation of bile duct, hypertrophy, and brown pigment deposition in perilobular hepatocytes, and diffuse clear cell changes in males and females, and hyperplasia of squamous epithelium in the forestomach in males.

In parental male rats, no changes in histopathological findings or weight of the testes and epididymis were found. In parental females, extension of mean oestrous cycle length and increase in females with irregular oestrous cycle were observed in the 40 mg/kg bw/day group. There were no adverse effects on the other reproductive parameters (such as the mating index, fertility index, numbers of corpora lutea or implantations, implantation index, delivery index, gestation index, gestation length, parturition or maternal behaviour).

On examination of offspring, decrease in viability index on day 4 and total litter loss (from one dam) were observed at 40 mg/kg bw/day group. External examinations of offspring revealed no external morphological abnormalities.

The NOAEL is considered to be 8 mg/kg bw/day for general toxicity and reproductive/developmental toxicity. Adverse effects on offspring may be influenced by parental toxicity.

In a prenatal developmental study conducted in SD rats, 2-propen-1-ol was administered by gavage at doses of 0, 10, 35, or 50 mg/kg bw/day to pregnant rats on gestation days 9 to19 [OECD TG 414]. At doses of 10 mg/kg bw/day and higher, significant toxicity in dams was observed. Maternal

toxicities at 35 and 50 mg/kg bw/day were mortalities, clinical findings, reductions in body weight gain and feed consumption, macroscopic liver findings and increased liver weights. One female at 10 mg/kg bw/day also had macroscopic liver findings. An increased frequency of total litter loss was observed at 35 and 50 mg/kg bw/day dose levels. In case of total litter loss, severe toxicities were observed in the dam (loss of body weight, severe decreases in feed consumption, and evidence of significant liver toxicity). Despite the severe maternal toxicity observed, there were no 2-propen-1-ol related increases in malformation rates or incidence of variations. 2-Propen-1-ol had no effects on intrauterine growth or survival in the fetuses from dams that survived to necropsy. Therefore, 10 mg/kg bw/day was considered to be the LOAEL for maternal toxicity, based on liver findings, and 10 mg/kg bw/day was considered to be the NOAEL for developmental toxicity, based on an increased frequency of total litter loss at 35 and 50 mg/kg bw/day, when 2-propen-1-ol was administered orally by gavage to pregnant rats.

4 HAZARDS TO THE ENVIRONMENT

4.1 Aquatic Effects

The toxicity of 2-propen-1-ol to aquatic organisms has been investigated for the three trophic levels and reliable test results are summarized in the Tables 13 and 14.

Acute Toxicity Test Results

Fish

Two reliable acute fish toxicity studies are available. Medaka (*Oryzias latipes*) were exposed to 2-propen-1-ol under semi-static conditions at nominal concentrations of 0, 0.32, 0.56, 0.68, 0.82, and 1.0 mg/L for 96 hours [OECD TG 203]. Chemical analysis of the old (expired) test solution 24 hours after renewal showed that the measured concentrations ranged from 90 % to 102 % of the nominal test concentrations. The LC_{50} (96-h) for Medaka was 0.59 mg/L [MOE Japan 2003]. In another study, Fathead minnows (*Pimephales promelas*) were exposed to 2-propen-1-ol under flow-through conditions for 96 hours and the LC_{50} (nominal) was 0.32 mg/L [Geiger et al. 1990].

Invertebrates

Two reliable studies are available. In an acute aquatic invertebrate toxicity study [OECD TG 202], *Daphnia magna* were exposed under semi-static conditions to 2-propen-1-ol at measured concentrations of 0, 0.858, 1.69, 2.97, 5.19, and 9.73 mg/L for 48 hours [MOE Japan 2003]. Chemical analysis of the old (expired) test solution 24 hours after renewal showed that the measured concentrations ranged from 83 % to 96 % of the nominal concentrations. The EC_{50} (48-h, immobilization) was 2.1 mg/ L.

Polychaete (*Ophryotrocha diadema*) was exposed to 2-propen-1-ol under static conditions in a test that was not conducted to a standard guideline method. The 48-h toxicity tests were carried out using *O. diadema* at each concentration in duplicated trials. The animals were starved for 2 days prior a test, and no food was provided during the test. For control cultures, the medium used was 49 mL of sterile, filtered sea water. Test cultures were prepared by adding 1 mL of an appropriate dilution in distilled water of the chemical to 49 mL of sea water. For each test a half-logarithmic series of concentrations was used. After the 48-h exposure period, surviving animals were transferred into clean sea water, as were the control animals, where they were allowed one week to recover. Any animals which died during this period were recorded along with the earlier mortalities in the test. The 48-h LC_{50} was 0.33-1.0 mg/ L (nominal) [Parker, 1984].

Algae

Three reliable studies are available. In an algal growth inhibition study [OECD TG 201], green alga (*Pseudokirchneriella subcapitata*) was exposed under static conditions to 2-propen-1-ol at measured concentrations of 0, 0.20, 0.46, 0.94, 2.1, 4.4 and 9.7 mg/L for 72 hours. The 48-h E_rC_{50} from a linear portion of the growth curve and the 72-h E_bC_{50} by biomass were calculated to be 11 mg/L [MOE Japan, 2003]. This study was conducted in a closed system, and therefore daily change in growth rates in the control was significant. Then the toxic value was calculated based on the averaged growth rate between 0 and 48 hours in a closed system. In another guideline study [OECD TG 201], green alga (*Pseudokirchneriella subcapitata*) was exposed to 2-propen-1-ol at measured concentrations of 0, 1.4, 3.0, 6.1, 12.4, 23.4 and 42.5 mg/L for 72 hours. The 72-h E_rC_{50} and the 72-h E_bC_{50} were 7.8 and 2.6 mg/L, respectively [Allyl Alcohol Consortium, 2004a]. In the third guideline study [OECD TG 201], green alga (*Pseudokirchneriella subcapitata*) was exposed to 2-propen-1-ol under static conditions at measured concentrations of 0, 0.34, 0.93, 2.4, 6.0 and 9.1 mg/L for 72 hours. The 72-h E_rC_{50} and the 72-h E_bC_{50} were 5.4 and 2.3 mg/L, respectively [Lyondell Chemical Company, 2005a].

Table 13 Acute toxicity of 2-propen-1-ol to aquatic organisms

Organism	Test duration	Result (mg/L)	Reference
Fish			
Medaka (*Oryzias latipes*)	96-h (semi-static)	LC_{50} = 0.59	MOE Japan, 2003
Fathead minnow (*Pimephales promelas*)	96-h (flow-through)	LC_{50} = 0.32	Geiger et al., 1990
Invertebrates			
Daphnia (*Daphnia magna*)	48-h (semi-static)	LC_{50} = 2.1	MOE Japan, 2003
Polychaete (*Ophryotrocha diadema*)	48-h (static)	LC_{50} = 0.33-1.0	Parker, 1984
Algae			
Green algae (*Pseudokirchneriella subcapitata*)	72-h (static, closed system)	E_rC_{50} = 11 (growth rate) E_bC_{50} = 11 (biomass)	MOE Japan, 2003
Green algae (*Pseudokirchneriella subcapitata*)	72-h (static, open system)	E_rC_{50} = 7.8 (growth rate) E_bC_{50} = 2.6 (biomass)	Allyl Alcohol Consortium, 2004a
Green algae (*Pseudokirchneriella subcapitata*)	72-h (static)	E_rC_{50} = 5.4 (growth rate) E_bC_{50} = 2.3 (biomass)	Lyondell Chemical Company, 2005a

Chronic Toxicity Test Results

Fish

No studies available for this endpoint.

Invertebrates

One reliable study is available. In a chronic toxicity study [OECD TG 211], *Daphnia magna* were exposed under semi-static conditions to 2-propen-1-ol at measured concentrations of 0, 0.031, 0.091, 0.210, 0.436, 0.919, and 2.02 mg/L for 21 days [MOE Japan, 2003]. All parental daphnia died at the highest concentration of 2.02 mg/L. The EC_{50} (reproduction) was not determined because no significant difference was observed at the concentrations of 0.031 to 0.919 mg/L except the highest concentration. The NOEC of 21-d chronic toxicity in *Daphnia magna* was 0.92 mg/L.

Algae

Three reliable studies are available. In an algal growth inhibition study [OECD TG 201] green alga (*Pseudokirchneriella subcapitata*) were exposed for 72 hours under static conditions to 2-propen-1-ol at measured concentrations of 0, 0.20, 0.46, 0.94, 2.1, 4.4, and 9.7 mg/L [MOE Japan, 2003], 0, 1.4, 3.0, 5.8, 12.0, 22.2, and 42.2 mg/L [Allyl Alcohol Consortium, 2004a] and 0, 0.34, 0.93, 2.4, 6.0 and 9.1 mg/L [Lyondell Chemical Company, 2005a]. The NOEC values were 2.2 mg/L (48-h for growth rate and 72-h for biomass) [MOE Japan, 2003], 1.4 mg/L (72-h for growth rate and biomass) [Allyl Alcohol Consortium, 2004a] and 0.93 mg/L (72-h for growth rate and biomass) [Lyondell Chemical Company, 2005a].

Table 14 Chronic toxicity of 2-propen-1-ol to aquatic organisms

Organism	Test duration	Result (mg/L)	Reference
Invertebrates			
Daphnia (*Daphnia magna*)	21-d (semi-static)	NOEC = 0.92	MOE Japan, 2003
Algae			
Green algae (*Pseudokirchneriella subcapitata*)	72-h (static, closed system)	NOEC = 2.2 (biomass) NOEC (48-h) = 2.2 (growth rate)	MOE Japan, 2003
Green algae (*Pseudokirchneriella subcapitata*)	72-h (static, open system)	NOEC = 1.4 (biomass and growth rate)	Allyl Alcohol Consortium, 2004a
Green algae (*Pseudokirchneriella subcapitata*)	72-h (static)	NOEC = 0.93 (biomass and growth rate)	Lyondell Chemical Company, 2005a

Toxicity to Microorganisms

A study using protozoa (*Tetrahymena pyriformis*) for 2 days (growth inhibition) was conducted [Schultz et al., 1994] giving the IC_{50} (50% inhibition concentration) of 4,806 mg/L.

4.2 Terrestrial Effects

A study using a dicotyledon plant (*Lactuca sativa*) for 3 days (germination) was conducted [Reynolds, 1977], giving the EC_{50} (50% germination concentration) of 3.3 mg/L.

4.3 Other Environmental Effects

There is no available information.

4.4 Initial Assessment for the Environment

2-Propen-1-ol is colourless liquid and is miscible with water. Melting point, boiling point, vapour pressure and partition coefficient are -129 °C, 96.9 °C, 25 hPa (20 °C) and log Kow = 0.17, respectively. 2-Propen-1-ol is not expected to be hydrolyzed under normal environmental conditions. Indirect photo-oxidation by hydroxyl radicals in the atmosphere is predicted to occur with a half-life of 4.32 hours. 2-Propen-1-ol is readily biodegradable under aerobic conditions within 14 days (BOD = 86 %). The estimated BCF is 3.2 and there is low potential for bioaccumulation. Fugacity Model Mackay level III calculations [Allyl Alcohol Consortium, 2005a]

indicate that 2-propen-1-ol will be distributed mainly to water (62.1 %) and soil (37.7 %) compartments if released to air, while 2-propen-1-ol will stay exclusively in the water compartment (99.7 %) if released to water. If released to soil, 2-propen-1-ol will be distributed mainly to water (43.0 %) and soil (56.9 %) compartments. If released simultaneously to air, soil and water, 2-propen-1-ol will be distributed mainly to water (75.7 %) and soil (24.1 %) compartments. Henry's Law constant is 4.99 x 10^{-6} atm.m^3/mole.

Acute toxicities to fish (96-h LC_{50}) are 0.589 mg/L (Medaka) and 0.32 mg/L (Fathead minnow). Acute toxicity to *Daphnia magna* (48-h EC_{50}) is 2.05 mg/ L. The 48-h LC_{50} in Polychaete (*Ophryotrocha diadema*) is 0.33-1.0 mg/ L. Acute toxicities to green algae (*Pseudokirchneriella subcapitata*) are 5.4 mg/L (72-h E_rC_{50}) and 2.3 mg/L (72-h E_bC_{50}) [OECD TG 201]. The NOEC of 21-d chronic toxicity in *Daphnia magna* is 0.92 mg/L [OECD TG 211]. The NOEC value in green algae (*Pseudokirchneriella subcapitata*) is 0.93 mg/L (72-h for growth rate and biomass) [OECD TG 201].

Toxicity (2-day IC_{50}) to protozoa (*Tetrahymena pyriformis*) is 4806 mg/L.

5 RECOMMENDATIONS

Human Health:

The chemical is a candidate for further work. The chemical possesses properties indicating a hazard for human health (acute toxicity, repeated dose toxicity, irritation, genotoxicity, carcinogenicity, reproductive/developmental toxicity). 2-Propen-1-ol is manufactured in a closed system in Japan, but is used to produce various products and occupational exposure through inhalation and dermal routes is possible in both production and user sites. Monitoring data provided by the sponsor country indicate that potential indirect exposure to consumers via the environment is anticipated. Therefore, an exposure assessment and, if necessary, risk assessment for workers and consumers should be performed.

Environment:

The chemical is a candidate for further work. The chemical possesses properties indicating a hazard to the environment (acute toxicity in algae, fish and daphnia and chronic toxicity in daphnia). Based on data presented by the sponsor country (relating to production by two producers which account for approx 30-40 % of global production and relating to the use, the total reported releases and the transfers in the sponsor country), potential environmental exposure is anticipated. Therefore, member countries are invited to perform an exposure assessment and, if necessary, a risk assessment for the environment.

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S I D S

D o s s i e r

Existing Chemical	ID: 107-18-6
CAS No.	107-18-6
EINECS Name	allyl alcohol
EC No.	203-470-7
TSCA Name	2-Propen-1-ol
Molecular Formula	C3H6O

Producer Related Part

Company:	Safepharm Laboratories
Creation date:	21-SEP-2005

Substance Related Part

Company:	Safepharm Laboratories
Creation date:	21-SEP-2005

Memo:	ICCA HPV 2-Propen-1-ol
Printing date:	09-MAY-2006
Revision date:	
Date of last Update:	**09-MAY-2006**
Number of Pages:	**236**
Chapter (profile):	Chapter: 1, 2, 3, 4, 5, 6, 7, 8, 10
Reliability (profile):	Reliability: without reliability, 1, 2, 3, 4
Flags (profile):	Flags: without flag, confidential, non confidential, WGK (DE), TA-Luft (DE), Material Safety Dataset, Risk Assessment, Directive 67/548/EEC, SIDS

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26-OCT-2005

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26-OCT-2005

1.0.2 Location of Production Site, Importer or Formulator

1.0.3 Identity of Recipients

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26-OCT-2005

1.0.4 Details on Category/Template

1.1.0 Substance Identification

IUPAC Name: 2-propenol
Smiles Code: OCC=C
Mol. Formula: CH2=CHCH2OH
Mol. Weight: 58.08

26-OCT-2005 (95)

1.1.1 General Substance Information

Substance type: organic
Physical status: liquid
Colour: colorless
Odour: pungent, mustard-like

26-OCT-2005 (95)

Purity type: typical for marketed substance
Substance type: organic
Physical status: liquid
Purity: >= 99 - % w/w
Colour: clear, colorless
Odour: sharp, mustard-like

26-OCT-2005 (159)

Purity type: typical for marketed substance
Substance type: organic
Physical status: liquid
Purity: >= 99 - % w/w
Colour: clear liquid
Odour: pungent odor

03-JAN-2006 (31) (143)

1.1.2 Spectra

1.2 Synonyms and Tradenames

2-propen-1-ol

26-OCT-2005 (58) (95)

2-propene-1-ol-3

26-OCT-2005

2-propene-3-ol

26-OCT-2005 (95)

2-propenol

26-OCT-2005

2-propenyl alcohol

26-OCT-2005

3-hydroxypropene

26-OCT-2005 (58)

alcool allilco (Italian)

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alcool allylique (French)

26-OCT-2005 (31)

allilowy alkohol (Polish)

26-OCT-2005 (31)

Allylalkohol (German)

26-OCT-2005 (31)

Orvinylcarbinol

26-OCT-2005

propenol

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propenyl alcohol

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vinyl carbinol

26-OCT-2005 (58) (95)

1.3 Impurities

Purity type: typical for marketed substance

Remark: PURITY: >= 99.0% (W/W)
ACID IMPURITY (as acetic acid): =<0.01% (W/W)
WATER: =< 0.3% (W/W)

26-OCT-2005 (154)

Purity type: typical for marketed substance

Remark: PURITY:99.0% min
OH IMPURITY:0.8% max
ALDEHYDE IMPURITY:0.2% max
COLOR, APHA:20 max
WATER:0.3% max

03-JAN-2006 (31)

Purity type: typical for marketed substance

Remark: Typical impurities:
n-propanol; 0.75% w/w max.
water ; 0.30% w/w max.
propionaldehyde; 0.01% w/w max.

26-OCT-2005 (159)

1.4 Additives

1.5 Total Quantity

Quantity: = 136100 tonnes produced in 2003

Remark: Worldwide; 136100 t/year
Japan; 56700 t/year
(estimated)
26-OCT-2005 (88)

Quantity: = 45000 tonnes produced in 2001

Remark: Estimated production in Japan
26-OCT-2005 (99)

Quantity: = 27931 tonnes produced in 1996

26-OCT-2005 (98)

1.6.1 Labelling

Labelling: as in Directive 67/548/EEC
Symbols: (T) toxic
(N) dangerous for the environment
Specific limits: no data
R-Phrases: (10) Flammable
(23/24/25) Toxic by inhalation, in contact with skin and if swallowed
(36/37/38) Irritating to eyes, respiratory system and skin
(50) Very toxic to aquatic organisms
S-Phrases: (1/2) Keep locked up and out of reach of children
(36/37/39) Wear suitable protective clothing, gloves and eye/face protection
(38) In case of insufficient ventilation, wear suitable respiratory equipment
(45) In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible)
(61) Avoid release to the environment. Refer to special instructions/Safety data sets

26-OCT-2005

1.6.2 Classification

Classified: as in Directive 67/548/EEC
Class of danger: dangerous for the environment
R-Phrases: (50) Very toxic to aquatic organisms

26-OCT-2005

Classified: as in Directive 67/548/EEC
Class of danger: toxic
R-Phrases: (23/24/25) Toxic by inhalation, in contact with skin and if swallowed

26-OCT-2005

Classified: as in Directive 67/548/EEC
Class of danger: flammable
R-Phrases: (10) Flammable

26-OCT-2005

1.6.3 Packaging

Memo: Transportation

Remark: Packing: 170 kgs in drum
Hazard class: 6.1 (Packing Group: I)
03-JAN-2006 (31)

Memo: Packing

Remark: 170 kg (200 L) in drum
7-10 tonnes in tanker lorry
26-OCT-2005 (154)

1.7 Use Pattern

Type: industrial
Category: Chemical industry: used in synthesis

Remark: Raw material for diallyl phthalate resin, medicine, allyl glycidyl ether, resin, epichlorohydrin, fragrance, and fire retardant
26-OCT-2005 (154)

Type: industrial
Category: Chemical industry: used in synthesis

Remark: 2-Propen-1-ol is an isomer of propylene oxide, and is a bifunctional molecule used by chemical manufacturers for a multitude of purposes by reaction of the alkene functionality, the hydroxy functionality, or both.

2-Propen-1-ol is an important starting material, and is used in the manufacture of a wide range of chemicals, as shown in the table below as well as an intermediate for manufacturing pharmaceuticals, fire retardants and herbicides.

2-Propen-1-ol products and its use:

Product Name	CAS No.	USE
1,4-butanediol	110-63-4	optical resins
2-methyl-1,3-propanediol	2163-42-0	optical resins
diallyl diglycol carbonate	142-22-3	optical resins
diallyl phthalate	131-17-9	plasticizer
diallyl isophthalate	1087-21-4	plasticizer
allyl glycidyl ether	106-92-3	silane coupling agents for a multitude of applications (such

		as water treatment and glass adhesion)
epichlorohydrin	106-89-8	manufacture of epoxy resins and synthetic glycerol and epichlorohydrin elastomer
allyl methacrylate	96-05-9	resins for coating applications
styrene allyl alcohol	25119-62-4	resins for coating applications
allyl hexanoate	123-68-2	flavorings

11-JAN-2006 (159)

Type: industrial
Category: Chemical industry: used in synthesis

Remark: raw material for epichlorohydrin, organic synthesis chemistry
26-OCT-2005 (144)

1.7.1 Detailed Use Pattern

1.7.2 Methods of Manufacture

Orig. of Subst.: Synthesis
Type: Production

Remark: Method I:
Propylene oxide is isomerized to make 2-propen-1-ol.

Method II:
Propylene is oxidized directly to make 2-propen-1-ol.

CH3CH=CH2 + CH3COOH + 1/2 O2 --------->
CH2=CHCH2OH + CH3COOH

[Method II was discovered by SHOWA DENKO K.K..]
05-DEC-2005 (154)

1.8 Regulatory Measures

1.8.1 Occupational Exposure Limit Values

Limit value: .5 other: ppm

Remark: Threshold Limit Values : 8 hr Time Weighted Avg (TWA): 0.5 ppm (1.2 mg/m3), skin.

Excursion Limit Recommendation: Excursions in worker exposure levels may exceed three times the TLV-TWA for no more than a total of 30 min during a work day, and under no circumstances should they exceed five times the TLV-TWA, provided that the TLV-TWA is not exceeded.

A4; Not classifiable as a human carcinogen.

Other Occupational Permissible Levels :
Other recommendations: 2 ppm as reported in 1980 for Australia, Belgium, Netherlands, Sweden and Switzerland (1978), Finland (1975), and in 1984 for West Germany.
26-OCT-2005 (1)

Remark: Immediately Dangerous to Life or Health:
20 ppm

OSHA Standards:
Vacated 1989 OSHA PEL TWA 2 ppm (5 mg/m3); STEL 4 ppm (10 mg/m3), skin designation, is still enforced in some states.

NIOSH Recommendations:
Recommended Exposure Limit: 10 Hr Time-Weighted Avg: 2 ppm (5 mg/m3), Skin.

Recommended Exposure Limit: 15 Min Short-Term Exposure Limit: 4 ppm (10 mg/m3), Skin.
26-OCT-2005 (105)

Remark:

Standards & regulations	Country	Adopted Value
MSHA (STANDARD-air)	USA	TWA 2 ppm (5 mg/m3), Skin
OSHA PEL (general industry, construction, shipyards and federal contractors)	USA	8H TWA 2 ppm (5 mg/ m3), skin
OEL	Arab REPUBLIC OF EGYPT	TWA 2 ppm (5 mg/ m3), skin
OEL	AUSTRALIA	TWA 2 ppm (5 mg/ m3), STEL 4 ppm, Skin
OEL	AUSTRIA	MAK 2 ppm (5 mg/ m3), skin
OEL	BELGIUM	TWA 2 ppm (4.8 mg/ m3), STEL 4 ppm (9.5 mg/ m3), Skin
OEL	DENMARK	TWA 2 ppm (5 mg/ m3), Skin
OEL	FINLAND	TWA 2 ppm (5 mg/ m3), STEL 4 ppm (10 mg/ m3), Skin
OEL	FRANCE	VME 2 ppm (5 mg/ m3), VLE 4 ppm (10 mg/ m3), Skin
OEL	GERMANY	MAK 2 ppm (5 mg/ m3), skin
OEL	THE NETHERLANDS	MAC-TGG 5 mg/ m3, Skin
OEL	JAPAN	OEL 1 ppm (2.4 mg/ m3) Skin
OEL	NORWAY	TWA 2 ppm (5 mg/ m3)
OEL	THE PHILIPPINES	TWA 2 ppm (5 mg/ m3), Skin

OEL	POLAND	MAC(TWA) 2 mg/ m3, MAC(STEL) 10 mg/ m3, Skin
OEL	RUSSIA	TWA 1 ppm
OEL	SWEDEN	NGV 2 ppm (5 mg m3), KTV 6 ppm (14 mg/ m3), Skin
OEL	SWITZERLAND	MAK-W 2 ppm (5 mg/ m3), KZG-W 4 ppm (10 mg/ m3), Skin
OEL	TURKEY	TWA 2 ppm (5 mg/ m3), Skin
OEL	UNITED KINGDOM	TWA 2 ppm (4.8 mg/ m3), STEL 4 ppm (9.7 mg/ m3), Skin

Note:
MSHA: Mine Safety and Health Administration
OEL: Occupational Exposure Limit
OSHA: Occupational Safety and Health Administration
PEL: Permissible Exposure Limit

21-DEC-2005 (124)

Remark: OEL (Occupational Exposure Limit) = 1ppm (2.4 mg/m3), skin absorption

06-JAN-2006 (66)

1.8.2 Acceptable Residues Levels

1.8.3 Water Pollution

1.8.4 Major Accident Hazards

1.8.5 Air Pollution

1.8.6 Listings e.g. Chemical Inventories

1.9.1 Degradation/Transformation Products

1.9.2 Components

1.10 Source of Exposure

Source of exposure: Human: exposure by production
Exposure to the: Substance

Method: Revised ICCA HPV Guidance for initial risk assessment (1998)
Result: The atmospheric concentration was measured at one production site in Japan.

Work place monitoring data for 2-propen-1-ol

Operation	Monitoring Data (Maximum Concentration) (ppm)(mg/m3)	Frequency	Working time (hrs/day)
Drum filling	0.12(0.29)	10-20 times/month	5
Sampling products	1.94(4.61)	1 time/day	0.17
Sampling in operation	0.37(0.88)	1 time/day	0.5
Inspection work	<0.02(0.048)	1 time/day	0.5

[Monitoring method] Air sample was suctioned at the breathing zone of the worker at the suction rate of 0.2 L/min. and adsorbed through a collection can and analyzed by GC. As shown in Table , the monitored exposure concentrations were <0.02 - 1.94 ppm (<0.048 - 4.61 mg/m3) at drum filling,
sampling products, sampling in operation and inspection work.

Reliability: (2) valid with restrictions
21-DEC-2005 (65)

Source of exposure: Human: exposure of the operator by intended use
Exposure to the: Substance

Remark: NIOSH (NOES Survey 1981-1983) has statistically estimated that 3,246 workers (157 of these are female) are potentially exposed to 2-propen-1-ol in the US. The NOES Survey does not include farm workers.

Occupation Description	Total Employees (Male and Female)	Total Female Employees
Chemical Technicians	1006	157
Drillers, Oil Well	1386	
Supervisors, Production Occupations	13	
Misc. Material Moving Equipment Operators	841	
Total	3246	157

Reliability: (2) valid with restrictions
19-DEC-2005 (106)

Source of exposure: Human: indirect exposure
Exposure to the: Substance

Method: Live crabs (Charybdis feriatus) weighing 3.7 kg were purchased three times from a seafood outlet in Hong Kong in the autumn of 1996 and steamed within one hour of purchase. Crabs were steamed for 20 minutes in a large stainless steel pot and cooled overnight (12h) in a refrigerator at 6°C before the meets were picked manually. Three types of meat were picked including the leg meat (LC, walking legs with claws) the body meat (B) and the carapace soft meat (C).

Percentage moisture of each cooked sample was determined by

an oven drying method.

Extraction was carried out with a simultaneous distillation and extraction apparatus. Sample meat (LC or B 500g; C 250g) was mixed with boiled double-distilled water in the ratio of 1 g of sample to 2 g of water. 10 µg of 2,4,6-trimethylpyridine was added as internal standard and redistilled dichloromethane (50 mL) was used as solvent. Extraction was performed for 2 hours from the start of boiling. Qualitative analysis of samples was performed using GC/MS, by comparison with retention times and spectra of standards. Quantification was determined by an internal standard method.

Moisture content and compounds from triplicate samples were analyzed by ANOVA and compared by the Tukey test at the $p<0.05$ level of significance.

Result: 2-Propen-1-ol was detected at concentrations of 0.3 ug/kg dry weight in the legs and claws, and at 0.1 ug/kg dry weight in the body. No 2-propen-1-ol was detected in the carapace meat.

Reliability: (2) valid with restrictions
Well reported published study

30-NOV-2005 (32)

Source of exposure: Human: indirect exposure
Exposure to the: Substance

Method: Volatile components of crushed garlic were obtained by water distillation, steam distillation, and Likens-Nickerson (L-N) distillation/solvent extraction with or without steam, respectively. The volatile components were analyzed and identified by capillary GC and combined GC-MS.

Result: 2-Propen-1-ol was identified in crushed garlic as a volatile component. Concentrations ranged 0.11-121ug/g garlic depending on the method used to remove the garlic oils from the water phase (see table)

Extraction method	Yield (µg/g garlic)
Essential oil obtained by water distillation	0.44
Essential oil obtained by steam distillation	0.11
Water layer of distillate in water distillation	23.09
Water layer of distillate in steam distillation	36.14
Likens-Nickerson water distillation/solvent extraction	121.22
Likens-Nickerson steam distillation/solvent extraction	81.04

Reliability: (2) valid with restrictions
Well reported published study

03-JAN-2006 (168)

Source of exposure: Human: indirect exposure
Exposure to the: Substance

Method: Fresh mussels were collected from the Oarai coast in Ibaraki, Japan, deshelled and homogenised with distilled water. Volatile components were isolated by vacuum distillation in the frozen state and analysed by GC-MS.

The procedure was repeated, but the homogenised mussels were allowed to rot for 10 days at room temperature prior to vacuum distillation.

Result: No 2-propen-1-ol was detected in fresh mussels, but in rotten mussels the level was 1.08 µg/g.

Reliability: (2) valid with restrictions
Well reported published study

30-NOV-2005 (167)

Source of exposure: Human: indirect exposure
Exposure to the: Substance

Result: 2-Propen-1-ol is rapidly formed in the body from the hydrolysis of allyl esters used as flavour agents in food. The estimated intake of 2-propen-1-ol from this route is 18µg/kg bw/day in Europe and 5.8 µg/kg bw/day in the USA

Reliability: (2) valid with restrictions
Report by FAO/WHO Expert Committee on Food Additives

30-NOV-2005 (164)

1.11 Additional Remarks

Memo: Acceptable Daily Intakes

Remark: Acceptable Daily Intakes:
Oral RfD: 0.005 mg/kg/day (UF: 1000, MF: 1)

26-OCT-2005 (158)

Remark: 2-Propen-1-ol vapor is irritating to eyes, nose, lungs, skin and mucous membranes. 2-Propen-1-ol is a potent lachrymator. The onset of eye irritation may be delayed. Noticeable eye irritation occurs at 5 ppm (12 mg/m3) with severe irritation occuring at
25 ppm (59 mg/m3). Above 25 ppm also the upper respiratory organs and
even lung tissue can be affected.

14-DEC-2005

1.12 Last Literature Search

Type of Search: Internal and External
Date of Search: 07-JUL-2005

Remark: ACGIH
AQUIRE (CIS, STN)
BEILSTEIN (STN)
BIOSIS (STN, Dialog)
CHEMCATS (STN)
CHRIS (CIS, CHEM-BANK)
CSCHEM (STN)
ChemFinder
ECDIN
GMELIN (STN)
HODOC(STN)
HSDB (CIS, STN, DataStar, CHEM-BANK)
IARC
INTERNET

IRIS (CIS, CHEM-BANK)
IUCLIDMSDS-CCOHS (STN, Dialog)
MEDLINE (STN, Dialog, Datastar)
MSDS-OHS (STN)
NCI
NIOSHOHMTADS (CIS, CHEM-BANK)
NIOSHTIC(STN, Dialog)
PROMT(STN, Dialog)
REGISTRY (STN, Dialog)
RTECS(STN, CIS, Dialog, CHEM-BANK)
SPECINFO (STN)
SRC PhysPro Database(SRC: Syracuse Research Corporation)
TOXCENTER (STN)
TOXFILE (Dialog, Datastar)
TSCATS (CIS)

26-OCT-2005

1.13 Reviews

2.1 Melting Point

Value: -129 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (58) (73) (159) (162)

Value: -50 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
28-OCT-2005 (95)

Value: -129 degree C

Reliability: (4) not assignable
Manufacturer's safety data sheet
28-OCT-2005 (143)

Value: -129 degree C

Reliability: (4) not assignable
Data reported in the secondary literature
28-OCT-2005 (154)

2.2 Boiling Point

Value: 96.9 degree C at 1033 hPa

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (58) (73)

Value: 96 - 97 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
28-OCT-2005 (95)

Value: 96.9 degree C

Reliability: (4) not assignable
Manufacturer's safety data sheet
28-OCT-2005 (143)

Value: 96.9 degree C

Reliability: (4) not assignable
Data reported in the secondary literature
28-OCT-2005 (154)

Value: 97.2 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
05-DEC-2005 (163)

Value: 97.1 degree C

Year: 1979

Method: Analytical grade 2-propen-1-ol supplied by BDH (India) Ltd. was distilled in a laboratory packed distillation column.
Reliability: (2) valid with restrictions
Well reported published study
07-DEC-2005 (123)

2.3 Density

Value: .854 at 20 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (95)

Value: .842

Reliability: (2) valid with restrictions
Peer reviewed literature data
28-OCT-2005 (73)

Value: .854 at 20 degree C

Reliability: (4) not assignable
Manufacturer's safety data sheet
28-OCT-2005 (143)

Value: .8535 at 20 degree C

Reliability: (4) not assignable
Data reported in the secondary literature
28-OCT-2005 (154)

2.3.1 Granulometry

2.4 Vapour Pressure

Value: 25 at 20 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (58) (73)

Value: 24

Reliability: (4) not assignable
Manufacturer's safety data sheet
28-OCT-2005 (143)

Value: 27 at 20 degree C

Reliability: (4) not assignable
Data reported in the secondary literature
28-OCT-2005 (53)

Value: 43 at 30 degree C

Reliability: (4) not assignable
Secondary data reported in the literature
28-OCT-2005 (53)

Value: 34.7 hPa

Year: 1989

Result: Reported as 26.1 mmHg
Reliability: (2) valid with restrictions
Peer reviewed literature data
05-DEC-2005 (37)

Value: 24.53 hPa at 20 degree C

Year: 1984

Result:

T deg C	P exp kPa	Pcalc kPa
10.000	1.307	1.279
12.000	1.467	1.461
14.000	1.667	1.667
16.000	1.893	1.898
18.000	2.146	2.157
20.000	2.453	2.448
22.000	2.773	2.772
24.000	3.135	3.135
26.000	3.533	3.539
28.000	4.000	3.989
30.000	4.506	4.490

32.000	4.986	5.045
34.000	5.653	5.660
37.000	6.753	6.708
40.000	7.933	7.923

Reliability: (2) valid with restrictions
Peer reviewed literature data
05-DEC-2005 (24)

2.5 Partition Coefficient

Partition Coeff.: octanol-water
log Pow: .17

Year: 1985

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (54) (58)

Partition Coeff.: octanol-water
log Pow: .21

Method: other (calculated): KOWWIN v1.67
Year: 2005

Result: Log Kow(version 1.67 estimate): 0.21

Experimental Database Structure Match:
Name : Allyl alcohol
CAS Num : 000107-18-6
Exp Log P: 0.17
Exp Ref : Hansch,C et al. (1995)

SMILES : OCC=C
CHEM : 2-Propen-1-ol
MOL FOR: C3 H6 O1
MOL WT : 58.08

TYPE	NUM	LOG KOW FRAGMENT DESCRIPTION	COEFF	VALUE
Frag	1	-CH2- [aliphatic carbon]	0.4911	0.4911
Frag	1	=CH2 [olefinic carbon]	0.5184	0.5184
Frag	1	=CH- or =C<[olefinc carbon]	0.3836	0.3836
Frag	1	-OH[hydroxy, aliphatic attach]	-1.4086	-1.4086
Const		Equation Constant		0.2290

Log Kow = 0.2135
Reliability: (2) valid with restrictions
Reliable estimation method
30-NOV-2005 (13)

2.6.1 Solubility in different media

Remark: Miscible with water, alcohol, chloroform, ether, petroleum ether.

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (58) (73) (95) (138)

Solubility in: Water
Value: 1000 g/l at 20 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
28-OCT-2005 (34)

Solubility in: Water
Value: 318 g/l

Method: other: calculated using WSKOW v1.41
Year: 2005

Result: Water Sol from Kow (WSKOW v1.41) Results:
==========

Water Sol: 3.177e+005 mg/L

Experimental Water Solubility Database Match:
Name : ALLYL ALCOHOL
CAS Num : 000107-18-6
Exp WSol : 1E+006 mg/L (20 deg C)
Exp Ref : YALKOWSKY,SH & DANNENFELSER,RM (1992)

SMILES : OCC=C
CHEM : 2-Propen-1-ol
MOL FOR: C3 H6 O1
MOL WT : 58.08
---------- WSKOW v1.41 Results

Log Kow (estimated) : 0.21
Log Kow (experimental): 0.17
Cas No: 000107-18-6
Name : Allyl alcohol
Refer : Hansch,C et al. (1995)
Log Kow used by Water solubility estimates: 0.17

Equation Used to Make Water Sol estimate:
Log S (mol/L) = 0.796 - 0.854 log Kow - 0.00728 MW +
Correction
(used when Melting Point NOT available)

Correction(s):	Value
Alcohol, aliphatic	0.510

Log Water Solubility (in moles/L) : 0.738
Water Solubility at 25 deg C (mg/L): 3.177e+005

Reliability: (2) valid with restrictions
Reliable estimation method
30-NOV-2005 (12)

Remark: Miscible with water, alcohol, chloroform, ether, petroleum ether.
Reliability: (4) not assignable
Manufacturer's safety data sheet
30-NOV-2005 (143)

2.6.2 Surface Tension

Remark: 25.8 dynes/cm at 20 degree C in contact with air or vapor
Reliability: (2) valid with restrictions
Peer reviewed literature data
28-OCT-2005 (161)

2.7 Flash Point

Value: 21.1 degree C
Type: open cup

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (95)

Value: 23.9 degree C
Type: closed cup

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (95)

Value: 21 degree C
Type: closed cup

Reliability: (2) valid with restrictions
Peer reviewed literature data
28-OCT-2005 (58) (73) (138)

Value: 21 degree C
Type: closed cup

Reliability: (4) not assignable
Secondary data reported in the literature
28-OCT-2005 (31)

Value: 21.5 degree C
Type: closed cup

Method: other: see test conditions

Test condition: Temperature 25 degree C, Moisture 45 %. Atmospheric Pressure:760 mm Hg.
Reliability: (4) not assignable

28-OCT-2005 (143)

2.8 Auto Flammability

Value: 378 degree C

Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (58) (73) (95)

Value: 443 degree C

Reliability: (4) not assignable
Data reported in the secondary literature
28-OCT-2005 (43) (154)

2.9 Flammability

Result: flammable

Remark: Liquid which can be ignited under almost normal temperature conditions. Water may be ineffective on fire.
NFPA classification: 3.3
Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (58) (102)

2.10 Explosive Properties

Result: explosive under influence of a flame

Remark: Explosive Limits and Potential Lower 2.5 Vol % ; Upper 18 Vol % (in Air). Explosion hazard : Moderate, when exposed to flame.
Reliability: (2) valid with restrictions
Peer reviewed literature data
Flag: Critical study for SIDS endpoint
28-OCT-2005 (137)

2.11 Oxidizing Properties

2.12 Dissociation Constant

Acid-base Const.: pKa = 15.5 at 25 deg C

Year: 1979

Reliability: (2) valid with restrictions
Peer reviewed literature data

14-DEC-2005 (142)

2.13 Viscosity

Value: 1.34 mPa s (dynamic)

Reliability: (4) not assignable
Manufacturer's safety data sheet
28-OCT-2005 (143)

2.14 Additional Remarks

Memo: Henry's Law Constant

Remark: Experimental Henry's Law Constant: 4.99E-6 atm-m3/mol
Temper: 25 degree C
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
14-DEC-2005 (55)

3.1.1 Photodegradation

Type: air
Light source: Sun light
INDIRECT PHOTOLYSIS
Sensitizer: OH
Conc. of sens.: 1500000 molecule/cm³
Rate constant: .000000000029709 cm³/(molecule * sec)
Degradation: 50 % after 4.3 hour(s)

Method: other (calculated): AOPWIN v1.91
Year: 2005
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (2) valid with restrictions
The value is estimated using the method recommended in the OECD Guidance
Flag: Critical study for SIDS endpoint
30-NOV-2005 (10)

Type: air
Light source: Sun light
INDIRECT PHOTOLYSIS
Sensitizer: O3
Conc. of sens.: 700000000000
Rate constant: .000000000000000012 cm³/(molecule * sec)
Degradation: 50 % after 22.9 hour(s)

Method: other (calculated): AOPWIN v1.91
Year: 2005
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (2) valid with restrictions
The value is estimated using the method recommended in the OECD Guidance
30-NOV-2005 (10)

Type: air
Light source: Sun light
INDIRECT PHOTOLYSIS
Sensitizer: OH
Conc. of sens.: 1000000 molecule/cm³
Rate constant: .0000000000259 cm³/(molecule * sec)
Degradation: 50 % after 4.3

Method: other (calculated)
Year: 1993
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Measured value of rate constant (Atkinson R (1986) Chem. Reviews 86, 69) used to calculate half-life
Reliability: (2) valid with restrictions
Well reported published study
30-NOV-2005 (49)

Type: air
INDIRECT PHOTOLYSIS
Sensitizer: O3
Rate constant: .0000000000000000144 cm³/(molecule * sec)

Degradation: 50 % after 5.5

Method: other (measured)
Year: 1993
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: 99%, Supplier: Aldrich/Fluka

Method: Ozone produced by the built-in generator of a calibrated continuous ozone analyser was introduced in a 3.5 m3 all-Teflon collapsable chamber constructed from 200A FEP film and covered with black plastic. The matrix air, purified by passing ambient air through large sorbent cartridges containing activated carbon, molecular sieves, silica gel and permanganate-coated alumina, contained no detectable concentrations of ozone, oxides of nitrogen, reactive hydrocarbons and carbonyls. The relative humidity was 55+/-10%. Once the desired ozone concentration in purified air was obtained, the ozone generator was turned off and the ozone concentration was monitored to verify the absence of olefins or other ozone-consuming impurities. Cyclohexane (acting as an OH scavenger) was introduced in the chamber. Finally, 2-propen-1-ol was injected into the chamber. The reaction was followed under pseudo first order conditions, with initial 2-propen-1-ol concentrations of 1.75-2 ppm, initial ozone concentrations of 174-200 ppb and cyclohexane concentrations of 200 or 400 ppm. Ozone was monitored continuously by ultraviolet photometry. Plots of the ratio of intial ozone concentration to concentration at time t versus time were made, and rate constants calculated.

In a related series of experiments, the above procedure was followed, but samples of the reaction mixture were taken and passed through C18 cartridges coated with twice recrystallised DNPH to convert carbonyl compounds to their 2,4-dinitrophenylhydrazine derivatives.

The derivatives were separated using reverse-phase HPLC. Confirmation of the presence of monofunctional or difunctional derivatives was obtained by measuring their 430/360 nm absorbtion ratio.

Result: Rate constant = 1.44E-17 cm^3/(molecule*sec)

Degradation products:

Formaldehyde (50+/-3%)
Hydroxyacetaldehyde (30+/-3%)
Acetaldehyde (6.5+/-0.5%)
Acetone (1.7+/-1.0%)
Cyclohexanone (observed but not measured)
Unidentified monofunctional carbonyl (approx 3%)
Well reported published study

30-NOV-2005 (49) (50)

Type: air
Light source: other
Light spect.: 230 - 300 nm

Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Method: A gas reaction flask (4.4 L) was used. Maintained at 25°C in a

water bath. Irradiation took place using a high pressure mercury lamp fitted with a water cooled quartz filter. Before each experiment, the system was flushed with dry CO2-free air. 2-Propen-1-ol was dosed into the system using a Hamilton syringe via a silicone septum. CO and CO2 production were monitored using gas chromatography and an IR gas analyser, respectively.
Result: Approximately one third of an initial concentration of 2-propen-1-ol (100 ppm) was converted to CO2 and CO following 2 hours irradiation at wavelengths of 230-300 nm, indicating some potential for photolytic degradation
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (57)

Type: air
Light source: Sun light
INDIRECT PHOTOLYSIS
Sensitizer: OH
Conc. of sens.: .00005 molecule/cm³
Degradation: 50 % after 14.7 hour(s)

Method: other (calculated)
Year: 1980
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Rate constant: 25.9 cm3/molecule.sec
Reliability: (4) not assignable
Data reported in the secondary literature
30-NOV-2005 (103)

Type: air
Light source: Sun light
INDIRECT PHOTOLYSIS
Sensitizer: OH

Method: other (calculated)
Year: 1986
Test substance: other TS: Name: 2-Propen-1-ol (CAS No. 107-18-6)

Method: Calculated using Fate of Atmospheric Pollutants Portion of GEMS.
Result: A half life of 6.03 hr was estimated. The predominant fate of 2-Propen-1-ol in the atmosphere is expected to be reaction with OH radicals.
Reliability: (4) not assignable
Data reported in the secondary literature
05-DEC-2005 (46)

Type: water
Light source: Sun light
INDIRECT PHOTOLYSIS
Sensitizer: OH
Rate constant: .00000000129 cm³/(molecule * sec)

Method: other (calculated)
Year: 1967
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Water pH 7

Reliability: (4) not assignable
Data reported in the secondary literature
30-NOV-2005 (15)

Type: water
Light source: Sun light
INDIRECT PHOTOLYSIS
Sensitizer: OH
Rate constant: .000000012 cm^3/(molecule * sec)

Method: other (calculated)
Year: 1976
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Concentration of sensitizer: 10 E-17 molar (pseudo first order estimated).
Result: The rate constant translates to a t1/2 of 1.8 years
Reliability: (4) not assignable
30-NOV-2005 (75)

Type: water
Light spect.: > 290 nm
Conc. of subst.: 13.5 mg/l
INDIRECT PHOTOLYSIS
Sensitizer: OH
Degradation: 14.9 % after

Method: other (measured)
Year: 1985
GLP: no data
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Concentration of substance: 13.5 mg/l aqueous solution, pH 5.9.
Method: aqueous solution where OH radicals were generated by the photolysis of hydrogen peroxide, 3 hr exposure.
Reliability: (4) not assignable
30-NOV-2005 (93)

3.1.2 Stability in Water

Type: abiotic

Remark: 2-Propen-1-ol is expected to be stable in water because it contains no functional groups that are susceptible to hydrolysis
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
30-NOV-2005 (87)

3.1.3 Stability in Soil

Type: laboratory
Radiolabel: yes
Concentration: 970 mg/kg
Soil temperature: 22 degree C
Soil humidity: 2.19 g water/100g soil dry weight

Soil classification: other: sand sandy loam
Content of clay: 16.4 - 21.6 %
silt: 30.1 - 37.6 %
sand: 53.5 - 40.8 %
Organ. carbon: 2.9 - 1 %
pH: 6.9 - 6.1
Dissipation time
DT50: < 4 - 8 day(s)
DT90: 7 - 27 day(s)

Method: other
Year: 1981
Test substance: other TS: refer to freetext

Remark: The half-life of residues is longer in the sand than in the sandy loam. This may be explained by the higher organic carbon content. The data presented here suggest that binding site of unextractable residues is not only the organic matter but also inorganic soil constituents at least during the first 30 days of experiment.
Test substance: Name: 2-Propen-1-ol-2,3-[14]C (specific activity 0.46 mCi/mmole)
Supplier: New England Nuclear
Shell Unkrauttod A - (technical formulation a.i. 850 g/L)
Reliability: (2) valid with restrictions
Well reported published study
30-NOV-2005 (139)

3.2.1 Monitoring Data (Environment)

Remark: 2-Propen-1-ol is classified as a Class I compound of PRTR law in Japan and all users and manufacturers have to report the amount of release and transport into the environment.

The newest reported releases and transfers (2003) of 2-propen-1-ol from users and manufacturers throughout Japan are summarised as shown below.

Total Reported Releases (kg)
****
Air	7,280
Water	11,248
Soil	0
Landfill	0
Total	18,528

****

Total Reported Transfers (kg)
****
Waste transfers	157,450
Transfers into sewage	550
Total	157,950

****

Total Reported Releases and Transfers (kg)
176,478
Reliability: (2) valid with restrictions
Data collected by Japanese government

21-DEC-2005 (101)

Remark: An environmental investigation in atmosphere was conducted in 5 points in Japan.
2-Propen-1-ol was detected in one out of 5 points (3 out of 15 samples) as a result of this investigation. The concentration of 2-propen-1-ol ranged from 50-60ng/m3 with a detection limit of 50ng/m3.

Result of environmental investigation in atmosphere of 2-propen-1-ol in Japan (fiscal year 1995)

Monitored point	Detection Frequency	Detection range	Detection limit
20% (1/5)	20% (3/15)	50-60 ng/m3	50 ng/m3

Monitored point : Kanagawa Environmental Research Center
Nagano Environmental Conservation Research Institute
Mt. Norikura
Tokuyama city
Hiroshima city
Japanese Ministry of Environment monitored 2-propen-1-ol concentrations and calculated the EHE as shown in Table

Total daily exposure of 2-propen-1-ol to humans summarised as shown below.

	Concentrations	Average Exposure (ug/kg/day)
Air Environmental air	0.05 ug/m3	0.015
Room air	-	-
Water Drinking water	-	-
Well water	<0.3 ug/L	0.012
Public water	<0.3 ug/L	(0.012)
Food	-	-
Soil	-	-
Total oral exposure	-	0.012
Total exposure	-	0.027

Note: 1) Underlined values show that exposure amounts were regarded as less than minimum detection limit'.
2) A value in brackets is not used for calculation of total oral exposure
3) Calculated by using these parameters, respiratory volume: 15 m3, daily water intake: 2L, daily food intake: 2000kg, body weight: 50kg.

Reliability: (2) valid with restrictions
Well reported published study

06-JAN-2006 (99)

Remark: Chemical profiles by Scorecard ED in US reported the largest releases by transfers of 2-propen-1-ol to EPA Toxics Release Inventory (TRI) and the data are shown in Table.

Table: Total reported releases and transfers of 2-propen-1-ol in USA (2002)

Total Reported Releases	(kg)
Air	65,621
Water	6,320
Land	86
Environment from facilities	301,912
Total	373,939

Total Reported Transfers and Wastes (kg)	
Off-site transfers	1,527,258
Production-related Wastes	4,812,953
Total	6,340,211

Total Reported Releases, Transfers and Wastes (kg)
6,714,150

Reliability: (2) valid with restrictions
Data collected by US government

21-DEC-2005 (160)

Remark: The levels of 2-propen-1-ol in the environment in Kitakyushu-city area of Japan was monitored in 1991-1992. No 2-propen-1-ol was detected in seawater (9 points), river water (10 points), reservoir water (2 points) and sewage treatment plant effluent (2 points) in addition to well water, tap water and rain water (each one point). The detection limit was 0.008 µg/L.

The activated carbon preconcentration method and GC/SIM measurement were used as the analytical method.

Detected Frequency and Analytical data (µg/L) of 2-Propen-1-ol

Sea			River and Reservoir		
Detected Frequency	Detected Range	Mean	Detected Frequency	Detected Range	Mean
0/33	nd	nd	0/48	nd	nd

Effluent of				Detection
Rain Mean	Well Mean	Tap Mean	Sewage Plant Mean	Limit (µg/L)
nd	nd	nd	nd	0.008

nd not detected

Reliability: (2) valid with restrictions
Well reported published study

03-JAN-2006 (69)

Type of measurement: other: Emissions from vehicles
Medium: air

Method: Sampling was conducted at the Allegheny Tunnel, Pennsylvania, USA. The tunnel is a four-lane dual tunnel 1850 m long at an elevation of 707 m above sea level. The roadbed is essentially level and straight. Traffic conditions are representative of highway cruise, at speeds close to the 55 mph speed limit. Strong diurnal and weekly traffic patterns permit sampling in periods dominated by diesels as well as periods dominated by gasoline powered vehicles.

The tunnel-air sampling was conducted in the eastbound tunnel. Traffic composition was determined by visual count. Traffic volume eastbound was determined by a road tube axle counter in combination with the visual counting.

Gas phase emissions were collected by adsorption on Tenax GC. Sampling times were 185-240 minutes, with air sampled at a rate of 900 mL/minute. All samples were analysed by GC/MS with thermal desorption used to recover the sample from the Tenax cartridge.

Result: 2-Propen-1-ol was not detected in exhaust gases from internal combustion engines in this study.

Reliability: (2) valid with restrictions
Well reported published study

30-NOV-2005 (52)

Medium: air

Test condition: 2-Propen-1-ol in air samples was absorbed on activated carbon and extracted with a 5 % solution of isopropyl alcohol in carbon disulfide.
2-Propen-1-ol was measured by gas chromatography.

Reliability: (4) not assignable
Inadequate reporting

30-NOV-2005 (68)

3.2.2 Field Studies

3.3.1 Transport between Environmental Compartments

Type: fugacity model level III
Method: other: Mackay Level III fugacity Model
Year: 2005

Remark: Software:
Chemical Evaluation and Research Institute, Japan (2002): report on generic fugacity model (Mackay level III).

Result:

Compartments	Release 100% to Air (1000kg/h)	Release 100% to Water (1000kg/h)	Release 100% to Soil (1000kg/h)	Equal emission scenario (1:1:1) (333 kg/h to each compartment)
Air	67.6 %	0.1 %	0.1 %	1.0 %

Water	25.1 %	99.7 %	19.4 %	62.1 %
Soil	7.3 %	0.0 %	80.4 %	36.7 %
Sediment	0.1 %	0.3 %	0.1 %	0.2 %

****

Test condition: The distributions were calculated with following factors.
2-Propen-1-ol
Molecular Weight: 58.08
Melting point: -129 °C (measured)
Vapor Pressure: 2.50E3 Pa (measured)
Water Solubility: 317700 g/m3 (estimated)
log Kow: 0.17 (measured)
Half life (hours)
- in air: 4.32
- in water: 336 (default value)
- in soil: 192
- in sediment: 576
Temp.: 25°C
Reliability: (2) valid with restrictions
Reliable estimation method
Flag: Critical study for SIDS endpoint
21-DEC-2005 (8)

Type: adsorption
Media: water - soil
Method: other
Year: 1980

Remark: The value of the soil adsorption coefficient indicates that 2-propen-1-ol may display high mobility in soil.
Result: Calculated soil adsorption coefficient: 4.37.
Reliability: (4) not assignable
30-NOV-2005 (71)

Type: volatility
Media: soil - air
Year: 1984

Remark: Based on a vapour pressure value of 28.1 mmHg measured at 25 degree celsius, 2-propen-1-ol is not highly volatile.
Reliability: (4) not assignable
30-NOV-2005 (24)

Type: volatility
Media: water - air
Method: other
Year: 1975

Remark: Based on a measured Henry's Law Constant of 4.9 E-6 atm. m3/mole at 25 degree celsius, 2-propen-1-ol will tend to migrate only slowly from water to air.
Reliability: (4) not assignable
30-NOV-2005 (55)

3.3.2 Distribution

Media: water - soil
Method: other (calculation): PCKOCWIN v1.66
Year: 2005

Result: Koc = 1.32
Test condition: SMILES : OCC=C
CHEM : 2-Propen-1-ol
MOL FOR: C3 H6 O1
MOL WT : 58.08
---------- PCKOCWIN v1.66 Results --------

First Order Molecular Connectivity Index .....: 1.914
Non-Corrected Log Koc.....: 1.6414
Fragment Correction(s):
1 Aliphatic Alcohol (-C-OH) .....: -1.5193
Corrected Log Koc .....: 0.1221

Estimated Koc: 1.32
Reliability: (2) valid with restrictions
The value is estimated using the method recommended in the OECD Guidance
21-DEC-2005 (11)

3.4 Mode of Degradation in Actual Use

3.5 Biodegradation

Type: aerobic
Inoculum: activated sludge
Concentration: 100 mg/l related to Test substance
Degradation: 86 % after 14 day(s)
Result: readily biodegradable

Method: other: MITI method
Year: 1975
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6) (Lot K-115)

Method: MITI method (similar to OECD TG301C)
Test solutions (duplicate) were centrifuged at 1500 x g for 10 min and the resulting supernatant was analyzed by GC and TOC meter.

Analytical conditions:
TOC meter
Flow rate: 200 mL/mim
Temperature: 850 °C
Solvent: H2O
Sample volume: 20 uL
GC
Detector: FID
Carrier gas: N2
Column: 20% PEG 20M/chromosorb W (2 mm diameter x 2 m length, Glass column)
Column temperature: 56 °C
Injection volume: 3 uL
Result: Degradation after 14 days by Coulometer 86%, TOC 95.5%, and GC 100%
Test condition: Concentration
test substance: 100 mg/L
activated sludge: 30 mg/L

Reliability: (1) valid without restriction
MITI Guideline study
Flag: Critical study for SIDS endpoint
30-NOV-2005 (97)

Year: 1988
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Biodegradability of 2-Propen-1-ol in river and sea water was tested according to the cultivation method. 2-Propen-1-ol was judged to be easily degradable.

Biodegradability of Chemical by Cultivation method

River	Sea	Institute	Concentration	Judgement
60	100	KGU	25	E (0)

Values in columns of River and Sea are degradation percentage of chemical after 3 day-cultivation.
River water from Mino River
Sea water from Akashi Beach
KGU: Kobe Gaku-In University
Concentration of test chemical, ppm
E: Easy of degradability (Judgement of degradability by cultivation method)
0: Easy of degradability (Judgement by MITI method)
Reliability: (2) valid with restrictions
Well reported published study
21-DEC-2005 (77)

3.6 BOD5, COD or BOD5/COD Ratio

Method: other
Year: 1952
Year:
Method:
Remark: Readily biodegradable
Result: % BODT: after 5 days ---> 9.1 %
after 10 days --> 55 %
after 15 days --> 78.2 %
after 20 days --> 81.8 %
Test condition: BOD measured at 20 deg C
Reliability: (4) not assignable
31-OCT-2005 (80)
Method: other
Year: 1954
Year:
Method:
Remark: Readily biodegradable
Result: BOD5: 1.6 ppm O2 / ppm substance,
BODT: 2.2 ppm O2 / ppm substance.
Test condition: BOD measured at 20 deg C
Reliability: (4) not assignable
31-OCT-2005 (96)

Method: other: APHA Standard Method

Concentration: 100 mg/l related to Test substance

C O D
Year:
COD: 2120 mg/g substance

R A T I O B O D 5 / C O D

BOD5/COD: .84
Method:
Result: Theoretical oxygen demand (THOD) 2.21 g/g
BOD measurement 1.79 g/g ----> 81 % of THOD
COD measurement 2.12 g/g ----> 96 % of THOD
Reliability: (4) not assignable
31-OCT-2005 (15)

3.7 Bioaccumulation

BCF: .5

Method: other: calculated
Year: 2005
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Calculated using: BCFWIN version 2.14 - 2000 U.S. Environmental Protection Agency
Log Kow used by BCF estimates: 0.17
Result: Log BCF = 0.500, BCF = 3.162
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
30-NOV-2005 (9)

3.8 Additional Remarks

Remark: 2-Propen-1-ol is used as an intermediate in the preparation of a variety of substances and has been used as a contact herbicide.
When released to water 2-propen-1-ol is not expected to volatilize, photooxidize or directly photolyze.
Biodegradation is expected to be the predominant fate of 2-propen-1-ol in water.
Release of 2-propen-1-ol to soil is expected to result in biodegradation and possible migration to groundwater.
Volatilization from wet soil, direct photolysis and bioconcentration are not expected to be significant.
Volatilization from dry surfaces or soil should be significant.
Release of 2-propen-1-ol to the atmosphere is expected to result mainly in reaction with photochemically generated hydroxyl radicals with estimated half-life of 6.03-14.7 hr.
Direct photolysis is not expected to be significant. Due to the high water solubility of 2-propen-1-ol, rainout may also occur.
Reliability: (4) not assignable
05-DEC-2005

AQUATIC ORGANISMS

4.1 Acute/Prolonged Toxicity to Fish

Type: semistatic
Species: Oryzias latipes (Fish, fresh water)
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** yes
LC50: .589

Method: OECD Guide-line 203 "Fish, Acute Toxicity Test"
Year: 2003
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No 107-18-6), Supplier: Wako Pure Chemical Industries, Ltd, Purity: >=99.0%, Lot No. WAN5739

Method: -Test Organisms
a) Size (length and weight): 2.23 cm (2.12 - 2.35 cm) in length; 0.16 g (0.10 - 0.25 g) in weight
b) Age: 11 month
c) Pretreatment: Acclimated for eleven months and not fed for 24 hours prior to the test. The mortality for 7 days before test was 1.1%.
d) Supplier/Source: Kureha Special Laboratory (breeding in the test lab.)

-Test Conditions
a) Dilution Water Source: Dechlorinated Tap Water
b) Dilution Water Chemistry:
Hardness: 28 mg/L (as CaCO3)
PH: 7.7
c) Exposure Vessel Type: a 5-liter screw capped glass vessel (closed system) with 3-liter test solution
d) Nominal Concentrations (mg/L): 0, 0.32, 0.56, 0.68, 0.82 and 1.0
e) Solvent and Concentrations: Not used
f) Stock and test solution preparation: One hundred mg of the test substance was dissolved in 100 mL dilution water to produce a 1,000 mg/L stock solution and test solution was prepared by adding the appropriate amount of the stock solution into the dilution water.
g) Number of Replicates: One vessel per treatment
h) Fish per Replicates: Ten fish per replicate
i) Renewal Rate of Test Solution: The test solution was renewed every 24 h.
J) Water Temperature: 23.9 - 24.0 deg C
k) Light Condition: 16h:8h, light-darkness cycle
l) Feeding: No

-Analytical monitoring
Portions of the test solutions were withdrawn at the beginning of the test and after 24 hours. Concentrations of the test substance were determined by GC-MS.
-Statistical Method
a) Data Analysis: Probit method for LC50
b) Measured Concentrations : Geometric mean concentrations

Result: -Measured Concentrations
All of the measured concentrations were between 88 and 112% of

the nominal concentrations (Table 1).

Table 1. Measured Concentrations of the Test Substance in the Test Water.
(Semi-static Condition)

Nominal conc. (mg/L)	Measured conc. (mg/L) (Percent of Nominal)		Mean* Measured Concentration (mg/L)
	O Hour New	24 Hours old	
Control	<0.01	<0.01	----
0.32	0.283 (88)	0.289 (90)	0.286
0.56	0.509 (91)	0.498 (89)	0.503
0.68	0.664 (98)	0.624 (92)	0.644
0.82	0.831 (101)	0.839 (102)	0.835
1.0	1.21 (112)	0.987 (99)	1.05

*: Geometric mean
new: Freshly prepared test solutions
old: Test solutions after 24 hours exposure

-Element value
LC50 (96 hr) : 0.589 mg/l (Table 3)
LC0 (96 hr) : 0.286 mg/l (Table 4)
LC100 (96 hr) : 0.835 mg/l (Table 4)

-Biological observations: No abnormal response at the test concentration and control during the exposure.

-Cumulative mortality

Table 2. The Numbers of Dead Fish (Mortality)

Nominal conc. (mg/L)	Mean* Measured Conc. (mg/L)	Cumulative Mortality (Percent Mortality) 24hr	48 hr	72 hr	96 hr
Control	---	0(0)	0(0)	0(0)	0(0)
0.32	0.286	0(0)	0(0)	0(0)	0(0)
0.56	0.503	0(0)	1(10)	2(20)	3(30)
0.68	0.644	1(10)	2(20)	3(30)	5(50)
0.82	0.835	4(40)	9(90)	10(100)	0(100)
1.0	1.05	5(50)	10(100)	10(100)	10(100)

*: Geometric mean

Table 3. Calculated LC50 values

Exposure period (hours)	LC50 (mg/l)	95% confidence limit (mg/l)	Statistical Method
24	0.994	0.842-1.71	Probit
48	0.685	0.610-0.769	Probit
72	0.634	0.529-0.756	Probit
96	0.589	0.487-0.678	Probit

Table 4. Observation of the highest concentration in 0% mortality and the lowest concentration in 100% mortality.

Exposure Period (Hours)	Highest Conc. in 0% Mortality (mg/L)	Lowest Conc. in 100% Mortality (mg/L)
24	0.503	>1.05
48	0.286	1.05
72	0.286	0.835
96	0.286	0.835

Conclusion: 96h-LC50 of 2-propen-1-ol was 0.589 mg/L
Reliability: (1) valid without restriction
Flag: Critical study for SIDS endpoint
21-DEC-2005 (100)

Type: static
Species: Pimephales promelas (Fish, fresh water)
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no
LC50: .32

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: Reagent grade

Remark: -Test species:
Common name; Fathead minnow
Genus and species; Pimephales promelas
Phylum; Chordata
Approximate size; 0.2 - 0.5 g
Result: -1st result.
LC50 = 0.32 mg/L

-2nd result.
LC50 = 0.32 mg/L

LC50(median lethal concentration)
Endpoint: mortality
Test condition: -Test vessel and diluent water
The static bioassays were performed in seamless glass, 30.5-cm cuboidal, Pyrex chromatography jars to which 20 liters of test solution was added. Water quality was routinely monitored to characterize the diluent water and ensure its suitability. Activated carbon-filtered, dechlorinated and tempered industrial service water from Lake Ontario was used in each test.

-Test solutions
Based on a recommended expected environmental concentration cutoff level, the maximum concentration used in these bioassays was 100 mg/L. Chemical known to be readily soluble at 100 mg/L were added directly to the diluent water in each aquarium in the appropriate amounts to give nominal concentrations of 100, 10, 1 and 0.1 mg/L. The chemical concentrations were not analyzed. Once the test solutions were prepared, the starting temperature, dissolved oxygen

and pH values were determined for each exposure concentration and the control. When the starting pH of the test solution fell outside the extremes of 6.5 to 8.5, the pH was adjusted to 7.0 by addition of 10 % (v/v) NaOH or 10% (v/v) H2SO4.

-The dilution water
Total dissolved solids: 180 mg/L
Total hardness (CaCO3): 130 mg/L
Noncarbonate hardness (as CaCO3): 38 mg/L
Alkalinity (as CaCO3): 93 mg/L
Soluble sulfate (SO4--): 31 mg/L
Chloride by I.C. (as Cl-): 26 mg/L
Total NH3 (as N): 0.06 mg/L
Organic nitrogen: 0.21 mg/L
NO3- (as N): 0.29 mg/L
NO2- (as N): <0.01 mg/L
Molybdenum-reactive dissolved SiO2: 0.35 mg/L
Total phosphorus (P): 0.02 mg/L
Total cyanide (CN-): <0.005 mg/L
Soluble fluoride (F-): 0.35 mg/L
pH: 7.4 pH units
Conductivity: 260 micro-mhos/cm
Total organic carbon: 1.8 mg/L
Total residual Cl2: 6.6 micro-g/L

-Test organisms
Each organism was acclimated to the control diluent water in the breeding/rearing tanks. Food was withheld for 24 h preceding start of the test. Juveniles of each species, as uniform in size as possible, were collected from the colonies. Ten juvenile organisms of each species were exposed to the test chemical in each treatment. Stratified randomization was accomplished by proportioning out no more than 20% of any one species into an aquarium at any one time. Biological loading was kept below 0.5 gram wet weight per liter of test solution.
-Physical/chemical parameter
Determinations of the temperature, dissolved oxygen and pH of each test solution were made in conjunction with the daily biological observations. Test temperature target was 20 plus or minus 1 degree C. If the dissolved oxygen concentration in a test chamber fell below 40% of the starting level in a test, the test was repeated with 0.05 L/min glass-sparger aeration. Each test was conducted within the extremes of 6.5 to 8.5 pH units. The photoperiod duration was 16 h of light. The air-water interface of each tank received approximately 50 ft-c of cool-white fluorescent light.

-Biological parameters
Biological observations were made daily. Survival, condition and behavioral information were recorded. Dead organisms were removed when observed. A test organism was considered dead if it appeared motionless and exhibited no response to gentle prodding. If more than one-half of the population of a species exposed in any treatment was determined to be dead, additional aquaria containing lower concentrations of test solution were set up. At any time during the test when each organism of a species were considered dead, these

biological parameters were determined and recorded.
Each species in these tests were exposed for the same time period 96 h. As in any bioassay that determines a dose response, the LC50 value can be archived at any time during the exposure.

-Data analysis
-Estimations of LC50 values. The LC50 values were estimated by an interpolation method using a computer program written for aquatic toxicity studies. The linear interpolation uses the logarithm transformation of the concentration and the angle transformation of the percent dead between the two doses that bracket 50 % response (i.e., proportion killed). The moving average and probit methods could not be used because the required minimum of two partial mortalities was rarely obtained.

-Number of times to test
The compound was tested twice.

Reliability: (3) invalid
This study is regarded as a range finding test but not a definitive test because the exposure concentrations were only 100, 10, 1 and 0.1 mg/L. Therefore, the reliability of the estimated toxic value is not high. However this study shows that this substance is highly toxic to fish as the 96-h LC50 is less than 1 mg/L.

14-DEC-2005 (41)

Type: static
Species: Carassius auratus (Fish, fresh water)
Exposure period: 24 hour(s)
Unit: mg/l **Analytical monitoring:** yes
LC50: ca. 1

Method: other: APHA for static tank acute toxicity tests
Year: 1979
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Test condition: Goldfish (carassius auratus) of uniform length (average 6.2 plus or minus 0.7 cm) and weight (average 3.3 plus or minus 1.0 g) and in good health were used as test fish.

This chemical was tested at a series of concentrations. In this test 10 fish exposed at 20 plus or minus 1 degree C in 25 L of solution contained in all-glass tanks measuring 42 X 28 X 28 cm. The duration of the test was 24 h and the solutions were aerated throughout the test period.

The concentrations of the test solutions tested was determined before and after the test, either with a total organic carbon analyzer or extraction and subsequent GC analysis.

Where the pH of test solutions was outside the range 6 - 8 it was adjusted to 7.0 with NaOH or H2SO4.
Solutions were made in local tap-water. The result of analysis was, in mg/L: Cl- = 65, NO2- = 0, NO3- = 4, SO4-- = 35, PO4--- = 0.15, HCO3- = 25, SiO2 = 25, NH4+ = 0, Fe = 0.05, Mn = 0, Ca++ = 100, Mg++ = 8, alkali as Na+ = 30 and pH = 7.8.

The LC50 is the concentration at 50% of the fish survive a test of a given duration. The LC50 was obtained by interpolation from a graph of logarithm of the concentration versus the percentage mortality.

Reliability: (3) invalid
This study was conducted for 24 hours, therefore the reliability of the toxic values should be low.

30-NOV-2005 (25)

Type: flow through
Species: Pimephales promelas (Fish, fresh water)
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** yes
LC50: .32
LC50 (range) : .28 - .37

Year: 1990
Test substance: other TS: Name: 2-Propen-1-ol (CAS No. 107-18-6), Purity >99%

Result: LC50 = 0.32 mg/L (mean)
LC50 = 0.28 mg/L (minimum)
LC50 = 0.37 mg/L (maximum)

LC50(median lethal concentration)
Endpoint: mortality

Cumulative Mortality:

Mean Concentration (mg/L)	0	0.26	0.37	0.65	1.00	1.50
0 hour	0	0	0	0	0	0
24	0	1	1	8	20	20
48	0	3	12	20	20	20
72	0	4	13	20	20	20
96	0	4	13	20	20	20

Percent Mortality (%):

Mean Concentration (mg/L)	0	0.26	0.37	0.65	1.00	1.50
0 hour	0	0	0	0	0	0
24	0	5	5	40	100	100
48	0	15	60	100	100	100
72	0	20	65	100	100	100
96	0	20	65	100	100	100

Test condition: -Test species: 28 days, 18.6 mm, 0.098 g

-Water parameter
temperature: 24.6 degree C (mean value)
hardness: 46.4 mg/L (as CaCO3, mean value)
alkalinity: 44.4 mg/L (as CaCO3, mean value)
dissolved O2: 7.7 mg/L (mean value)
pH: 7.5 (mean value)

Reliability: (2) valid with restrictions

21-DEC-2005 (45)

4.2 Acute Toxicity to Aquatic Invertebrates

Type: semistatic
Species: Daphnia magna (Crustacea)
Exposure period: 48 hour(s)
Unit: mg/l **Analytical monitoring:** yes
EC50: 2.05
EC50 (24 h) : 3.2

Method: OECD Guide-line 202
Year: 2003
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Supplier: Wako Pure Chemical Industries, Ltd., Purity: 99.0%, Lot No. WAN5739

Method: -Test Organisms
a) Age: < 24 hours old
b) Supplier/Source: National Institute for Environmental Studies (JAPAN)

-Test Conditions
a) Dilution Water Source: Laboratory supply water (dechlorinated)
b) Dilution Water Chemistry:
Hardness: 34 mg/L (as CaCO3)
pH: 7.7
c) Exposure Vessel Type: 100mL test solution in a 100 mL screw-capped glass vessel
d) Nominal Concentrations (as mg/L): 0, 1.0, 1.8, 3.2, 5.6 and 10
e) Vehicle/Solvent and Concentrations: Not used
f) Stock Solutions and Stability: 100 mg of the chemical was dissolved in 100 mL dilution water to produce a stock solution of 1,000 mg/L and the test solution was prepared by adding the appropriate amount of the stock solution into the dilution water.
g) Number of Replicates: 4
h) Individuals per Replicates: 5
i) Renewal Rate of Test Water: Total solution in a vessel was renewed every 24 hours.
j) Water Temperature: 20.2 - 20.5 deg C
k) Light Condition: 16:8 hours, light-darkness cycle
l) Feeding: No

-Analytical Procedure
Portions of the test solutions were withdrawn at 0 hour and 24 hours and concentrations of the test substance were determined by GC-MS.

-Statistical Method
a) Data Analysis: Probit method for EiC50
b) Method of Calculating Mean Measured Concentrations: Geometric mean concentrations

Result: -Measured Concentrations
All of the measured concentrations were between 83 and 99% of the nominal concentrations (Table 1).

Table 1. Measured Concentrations of the Test Substances in the Test Water (Semi-Static Condition)

Nominal conc. (mg/L)	Measured concentration (mg/L)				Geometric Mean (24h)
	0 Hours new	Percent of Nominal	24 Hours old	Percent of Nominal	
Control	< 0.01	-	<0.01	-	-
1.0	0.887	89	0.829	83	0.858
1.8	1.71	95	1.67	93	1.69
3.2	2.88	90	3.06	96	2.97
5.6	5.40	96	4.99	89	5.19
10	9.93	99	9.54	95	9.73

new: Freshly prepared test solutions
old: Test solutions after 24 hours exposure

-Water Chemistry
Temperature : 20.2 -20.5 deg C
pH : 7.6 - 7.8
Dissolved oxygen (mg/L) : 8.7
Total hardness (mg/L as CaCO3) : 32 - 34

-Element value
EiC50 (24 hr) : 3.20 mg/L
(95% confidence limits: 2.80 - 3.67 mg/L) (Table 2 & 3)
EiC50 (48 hr) : 2.05 mg/L
(95% confidence limits: 1.74 - 2.39 mg/l) (Table 2 & 3)
EiC0 (48 hr) : 0.858 mg/L (Table 2 & 4)
EiC100 (48 hr) : 5.19 mg/l (Table 2 & 4)

Table 2. The Numbers of Immobile Daphnia (Percent Immobility)

Nominal conc. (mg/L)	Mean* Measured Conc.(mg/L)	Cumulative Numbers of Immobilized Daphnia (percent Immobility)	
		24 Hours	48 Hours
Control	---	0(0)	0(0)
1.0	0.858	0(0)	0(0)
1.8	1.69	0(0)	5(25)
3.2	2.97	7(35)	18(90)
5.6	5.19	20(100)	20(100)
10	9.73	20(100)	20(100)

* : Geometric Mean

Table 3. Calculated EiC50 Values

Exposure Period (Hours)	EiC50 (mg/L)	95% Confidence Limits (mg/L)	Statistical Method
24	3.20	2.80-3.67	Probit
48	2.05	1.74-2.39	Probit

Table 4. Highest Concentration in 0% Immobility and Lowest Concentraton in 100% Immobility

Exposure Period (Hours)	Highest Concentration in 0% Immobility (mg/L)	Lowest Concentration in 100% Immobility (mg/L)
24	1.69	5.19
48	0.858	5.19

Conclusion: 48h-EiC50 of 2-propen-1-ol was 2.05 mg/L
Reliability: (1) valid without restriction
OECD TG study
Flag: Critical study for SIDS endpoint
06-JAN-2006 (100)

Type: static
Species: Daphnia magna (Crustacea)
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: .25 - .4

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: Reagent grade

Remark: -Test species:
Common name; Water flea
Genus and species; Daphnia magna
Phylum; Arthropoda
Approximate age; First and second larval instar
Result: -1st result.
LC50 = 0.25 mg/L

-2nd result.
LC50 = 0.40 mg/L

LC50(median lethal concentration)
Endpoint: mortality
Test condition: -Test vessel and diluent water
The static bioassays were performed in seamless glass, 30.5-cm cuboidal, Pyrex chromatography jars to which 20 liters of test solution was added. Water quality was routinely monitored to characterize the diluent water and ensure its suitability. Activated carbon-filtered, dechlorinated and tempered industrial service water from Lake Ontario was used in each test.

-Test solutions
Based on a recommended expected environmental concentration cutoff level, the maximum concentration used in these bioassays was 100 mg/L. Chemical known to be readily soluble at 100 mg/L were added directly to the diluent water in each aquarium in the appropriate amounts to give nominal concentrations of 100, 10, 1 and 0.1 mg/L. The chemical concentrations were not analyzed. Once the test solutions were prepared, the starting temperature, dissolved oxygen and pH values were determined for each exposure concentration and the control. When the starting pH of the test solution fell outside the extremes of 6.5 to 8.5, the

pH was adjusted to 7.0 by addition of 10 % (v/v) NaOH or 10% (v/v) H2SO4.

-The dilution water
Total dissolved solids: 180 mg/L
Total hardness (CaCO3): 130 mg/L
Noncarbonate hardness (as CaCO3): 38 mg/L
Alkalinity (as CaCO3): 93 mg/L
Soluble sulfate (SO4--): 31 mg/L
Chloride by I.C. (as Cl-): 26 mg/L
Total NH3 (as N): 0.06 mg/L
Organic nitrogen: 0.21 mg/L
NO3- (as N): 0.29 mg/L
NO2- (as N): <0.01 mg/L
Molybdenum-reactive dissolved SiO2: 0.35 mg/L
Total phosphorus (P): 0.02 mg/L
Total cyanide (CN-): <0.005 mg/L
Soluble fluoride (F-): 0.35 mg/L
pH: 7.4 pH units
Conductivity: 260 micro-mhos/cm
Total organic carbon: 1.8 mg/L
Total residual Cl2: 6.6 micro-g/L

-Test organisms
Each organism was acclimated to the control diluent water in the breeding/rearing tanks. Food was withheld for 24 h preceding start of the test. Juveniles of each species, as uniform in size as possible, were collected from the colonies. Ten juvenile organisms of each species were exposed to the test chemical in each treatment. Stratified randomization was accomplished by proportioning out no more than 20% of any one species into an aquarium at any one time. Biological loading was kept below 0.5 gram wet weight per liter of test solution.

-Physical/chemical parameter
Determinations of the temperature, dissolved oxygen and pH of each test solution were made in conjunction with the daily biological observations. Test temperature target was 20 plus or minus 1 degree C. If the dissolved oxygen concentration in a test chamber fell below 40% of the starting level in a test, the test was repeated with 0.05 L/min glass-sparger aeration. Each test was conducted within the extremes of 6.5 to 8.5 pH units. The photoperiod duration was 16 h of light. The air-water interface of each tank received approximately 50 ft-c of cool-white fluorescent light.

-Biological parameters
Biological observations were made daily. Survival, condition and behavioral information were recorded. Dead organisms were removed when observed. A test organism was considered dead if it appeared motionless and exhibited no response to gentle prodding. If more than one-half of the population of a species exposed in any treatment was determined to be dead, additional aquaria containing lower concentrations of test solution were set up. At any time during the test when each organism of a species were considered dead, these biological parameters were determined and recorded.
Each species in these tests were exposed for the same time

period 96 h. As in any bioassay that determines a dose response, the LC50 value can be archived at any time during the exposure.

-Data analysis
-Estimations of LC50 values. The LC50 values were estimated by an interpolation method using a computer program written for aquatic toxicity studies. The linear interpolation uses the logarithm transformation of the concentration and the angle transformation of the percent dead between the two doses that bracket 50 % response (i.e., proportion killed). The moving average and probit methods could not be used because the required minimum of two partial mortalities was rarely obtained.

-Number of times to test
The compound tested twice.

Reliability: (3) invalid
This study is regarded as a range finding test but not a definitive test because the exposure concentrations were only 100, 10, 1 and 0.1 mg/L. Therefore, the reliability of the estimated toxic value is not high. However this study shows that this substance is highly toxic to daphnia as the 96-h LC50 is less than 1 mg/L.

14-DEC-2005 (41)

Type: static
Species: Asellus intermedius (Crustacea)
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: .32 - 1

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: Reagent Grade

Remark: -Test species:
Common name; Pillbug
Genus and species; Asellus intermedius
Phylum; Arthropoda
Approximate size; 0.012 g

Result: -1st result.
LC50 = 0.32 mg/L

-2nd result.
LC50 = 1.00 mg/L

LC50(median lethal concentration)
Endpoint: mortality

Test condition: -Test vessel and diluent water
The static bioassays were performed in seamless glass, 30.5-cm cuboidal, Pyrex chromatography jars to which 20 liters of test solution was added. Water quality was routinely monitored to characterize the diluent water and ensure its suitability. Activated carbon-filtered, dechlorinated and tempered industrial service water from Lake Ontario was used in each test.

-Test solutions

Based on a recommended expected environmental concentration cutoff level, the maximum concentration used in these bioassays was 100 mg/L. Chemical known to be readily soluble at 100 mg/L were added directly to the diluent water in each aquarium in the appropriate amounts to give nominal concentrations of 100, 10, 1 and 0.1 mg/L. The chemical concentrations were not analyzed. Once the test solutions were prepared, the starting temperature, dissolved oxygen and pH values were determined for each exposure concentration and the control. When the starting pH pf the test solution fell outside the extremes of 6.5 to 8.5, the pH was adjusted to 7.0 by addition of 10 % (v/v) NaOH or 10% (v/v) H2SO4.

-The dilution water
Total dissolved solids: 180 mg/L
Total hardness (CaCO3): 130 mg/L
Noncarbonate hardness (as CaCO3): 38 mg/L
Alkalinity (as CaCO3): 93 mg/L
Soluble sulfate (SO4--): 31 mg/L
Chloride by I.C. (as Cl-): 26 mg/L
Total NH3 (as N): 0.06 mg/L
Organic nitrogen: 0.21 mg/L
NO3- (as N): 0.29 mg/L
NO2- (as N): <0.01 mg/L
Molybdenum-reactive dissolved SiO2: 0.35 mg/L
Total phosphorus (P): 0.02 mg/L
Total cyanide (CN-): <0.005 mg/L
Soluble fluoride (F-): 0.35 mg/L
pH: 7.4 pH units
Conductivity: 260 micro-mhos/cm
Total organic carbon: 1.8 mg/L
Total residual Cl2: 6.6 micro-g/L

-Test organisms
Each organism was acclimated to the control diluent water in the breeding/rearing tanks. Food was withheld for 24 h preceding start of the test. Juveniles of each species, as uniform in size as possible, were collected from the colonies. Ten juvenile organisms of each species were exposed to the test chemical in each treatment. Stratified randomization was accomplished by proportioning out no more than 20% of any one species into an aquarium at any one time. Biological loading was kept below 0.5 gram wet weight per liter of test solution.

-Physical/chemical parameter
Determinations of the temperature, dissolved oxygen and pH of each test solution were made in conjunction with the daily biological observations. Test temperature target was 20 plus or minus 1 degree C. If the dissolved oxygen concentration in a test chamber fell below 40% of the starting level in a test, the test was repeated with 0.05 L/min glass-sparger aeration. Each test was conducted within the extremes of 6.5 to 8.5 pH units. The photoperiod duration was 16 h of light. The air-water interface of each tank received approximately 50 ft-c of cool-white fluorescent light.

-Biological parameters

Biological observations were made daily. Survival, condition and behavioral information were recorded. Dead organisms were removed when observed. A test organism was considered dead if it appeared motionless and exhibited no response to gentle prodding. If more than one-half of the population of a species exposed in any treatment was determined to be dead, additional aquaria containing lower concentrations of test solution were set up. At any time during the test when each organism of a species were considered dead, these biological parameters were determined and recorded.
Each species in these tests were exposed for the same time period 96 h. As in any bioassay that determines a dose response, the LC50 value can be archived at any time during the exposure.
-Data analysis
-Estimations of LC50 values. The LC50 values were estimated by an interpolation method using a computer program written for aquatic toxicity studies. The linear interpolation uses the logarithm transformation of the concentration and the angle transformation of the percent dead between the two doses that bracket 50 % response (i.e., proportion killed). The moving average and probit methods could not be used because the required minimum of two partial mortalities was rarely obtained.

-Number of times to test
The compound was tested twice.

Reliability: (3) invalid
This study is regarded as a range finding test but not a definitive test because the exposure concentrations were only 100, 10, 1 and 0.1 mg/L. Therefore, the reliability of the estimated toxic value is not high. However this study shows that this substance is highly toxic to crustacea (Asellus intermedius) as the 96-h LC50 is less than 1 mg/L.

30-NOV-2005 (41)

Type: static
Species: Gammarus fasciatus (Crustacea)
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: Reagent Grade

Remark: -Test species:
Common name; Sideswimmer
Genus and species; Gammarus fasiatus
Phylum; Arthropoda
Approximate size; 0.007 g

Result: -1st result.
LC50 = 21 mg/L

-2nd result.
LC50 = 4.9 mg/L

LC50(median lethal concentration)
Endpoint: mortality

Test condition: -Test vessel and diluent water
The static bioassays were performed in seamless glass,

30.5-cm cuboidal, Pyrex chromatography jars to which 20 liters of test solution was added. Water quality was routinely monitored to characterize the diluent water and ensure its suitability. Activated carbon-filtered, dechlorinated and tempered industrial service water from Lake Ontario was used in each test.

-Test solutions
Based on a recommended expected environmental concentration cutoff level, the maximum concentration used in these bioassays was 100 mg/L. Chemical known to be readily soluble at 100 mg/L were added directly to the diluent water in each aquarium in the appropriate amounts to give nominal concentrations of 100, 10, 1 and 0.1 mg/L. The chemical concentrations were not analyzed. Once the test solutions were prepared, the starting temperature, dissolved oxygen and pH values were determined for each exposure concentration and the control. When the starting pH of the test solution fell outside the extremes of 6.5 to 8.5, the pH was adjusted to 7.0 by addition of 10 % (v/v) NaOH or 10% (v/v) H2SO4.

-The dilution water
Total dissolved solids: 180 mg/L
Total hardness (CaCO3): 130 mg/L
Noncarbonate hardness (as CaCO3): 38 mg/L
Alkalinity (as CaCO3): 93 mg/L
Soluble sulfate (SO4--): 31 mg/L
Chloride by I.C. (as Cl-): 26 mg/L
Total NH3 (as N): 0.06 mg/L
Organic nitrogen: 0.21 mg/L
NO3- (as N): 0.29 mg/L
NO2- (as N): <0.01 mg/L
Molybdenum-reactive dissolved SiO2: 0.35 mg/L
Total phosphorus (P): 0.02 mg/L
Total cyanide (CN-): <0.005 mg/L
Soluble fluoride (F-): 0.35 mg/L
pH: 7.4 pH units
Conductivity: 260 micro-mhos/cm
Total organic carbon: 1.8 mg/L
Total residual Cl2: 6.6 micro-g/L

-Test organisms
Each organism was acclimated to the control diluent water in the breeding/rearing tanks. Food was withheld for 24 h preceding start of the test. Juveniles of each species, as uniform in size as possible, were collected from the colonies. Ten juvenile organisms of each species were exposed to the test chemical in each treatment. Stratified randomization was accomplished by proportioning out no more than 20% of any one species into an aquarium at any one time. Biological loading was kept below 0.5 gram wet weight per liter of test solution.

-Physical/chemical parameter
Determinations of the temperature, dissolved oxygen and pH of each test solution were made in conjunction with the daily biological observations. Test temperature target was 20 plus or minus 1 degree C. If the dissolved oxygen concentration in a test chamber fell below 40% of the

starting level in a test, the test was repeated with 0.05 L/min glass-sparger aeration. Each test was conducted within the extremes of 6.5 to 8.5 pH units. The photoperiod duration was 16 h of light. The air-water interface of each tank received approximately 50 ft-c of cool-white fluorescent light.

-Biological parameters
Biological observations were made daily. Survival, condition and behavioral information were recorded. Dead organisms were removed when observed. A test organism was considered dead if it appeared motionless and exhibited no response to gentle prodding. If more than one-half of the population of a species exposed in any treatment was determined to be dead, additional aquaria containing lower concentrations of test solution were set up. At any time during the test when each organism of a species were considered dead, these biological parameters were determined and recorded.
Each species in these tests were exposed for the same time period 96 h. As in any bioassay that determines a dose response, the LC50 value can be archived at any time during the exposure.

-Data analysis
-Estimations of LC50 values. The LC50 values were estimated by an interpolation method using a computer program written for aquatic toxicity studies. The linear interpolation uses the logarithm transformation of the concentration and the angle transformation of the percent dead between the two doses that bracket 50 % response (i.e., proportion killed). The moving average and probit methods could not be used because the required minimum of two partial mortalities was rarely obtained.

-Number of times to test
The compound was tested twice.

Reliability: (3) invalid
This study should be regarded as a range-finding test

06-JAN-2006 (41)

Type: static
Species: other: Dugesia tigrina
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: 1 - 4

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: Reagent Grade

Remark: -Test species:
Common name; Flatworm
Genus and species; Dugesia tigrina
Phylum; Platyhelminthes
Approximate size; 0.006 g

Result: -1st result.
LC50 = 1.0 mg/L

-2nd result.

LC50 = 4.0 mg/L

LC50(median lethal concentration)
Endpoint: mortality

Test condition: -Test vessel and diluent water
The static bioassays were performed in seamless glass, 30.5-cm cuboidal, Pyrex chromatography jars to which 20 liters of test solution was added. Water quality was routinely monitored to characterize the diluent water and ensure its suitability. Activated carbon-filtered, dechlorinated and tempered industrial service water from Lake Ontario was used in each test.

-Test solutions
Based on a recommended expected environmental concentration cutoff level, the maximum concentration used in these bioassays was 100 mg/L. Chemical known to be readily soluble at 100 mg/L were added directly to the diluent water in each aquarium in the appropriate amounts to give nominal concentrations of 100, 10, 1 and 0.1 mg/L. The chemical concentrations were not analyzed. Once the test solutions were prepared, the starting temperature, dissolved oxygen and pH values were determined for each exposure concentration and the control. When the starting pH of the test solution fell outside the extremes of 6.5 to 8.5, the pH was adjusted to 7.0 by addition of 10 % (v/v) NaOH or 10% (v/v) H2SO4.

-The dilution water
Total dissolved solids: 180 mg/L
Total hardness (CaCO3): 130 mg/L
Noncarbonate hardness (as CaCO3): 38 mg/L
Alkalinity (as CaCO3): 93 mg/L
Soluble sulfate (SO4--): 31 mg/L
Chloride by I.C. (as Cl-): 26 mg/L
Total NH3 (as N): 0.06 mg/L
Organic nitrogen: 0.21 mg/L
NO3- (as N): 0.29 mg/L
NO2- (as N): <0.01 mg/L
Molybdenum-reactive dissolved SiO2: 0.35 mg/L
Total phosphorus (P): 0.02 mg/L
Total cyanide (CN-): <0.005 mg/L
Soluble fluoride (F-): 0.35 mg/L
pH: 7.4 pH units
Conductivity: 260 micro-mhos/cm
Total organic carbon: 1.8 mg/L
Total residual Cl2: 6.6 micro-g/L

-Test organisms
Each organism was acclimated to the control diluent water in the breeding/rearing tanks. Food was withheld for 24 h preceding start of the test. Juveniles of each species, as uniform in size as possible, were collected from the colonies. Ten juvenile organisms of each species were exposed to the test chemical in each treatment. Stratified randomization was accomplished by proportioning out no more than 20% of any one species into an aquarium at any one time. Biological loading was kept below 0.5 gram wet weight per liter of test solution.

-Physical/chemical parameter
Determinations of the temperature, dissolved oxygen and pH of each test solution were made in conjunction with the daily biological observations. Test temperature target was 20 plus or minus 1 degree C. If the dissolved oxygen concentration in a test chamber fell below 40% of the starting level in a test, the test was repeated with 0.05 L/min glass-sparger aeration. Each test was conducted within the extremes of 6.5 to 8.5 pH units. The photoperiod duration was 16 h of light. The air-water interface of each tank received approximately 50 ft-c of cool-white fluorescent light.

-Biological parameters
Biological observations were made daily. Survival, condition and behavioral information were recorded. Dead organisms were removed when observed. A test organism was considered dead if it appeared motionless and exhibited no response to gentle prodding. If more than one-half of the population of a species exposed in any treatment was determined to be dead, additional aquaria containing lower concentrations of test solution were set up. At any time during the test when each organism of a species were considered dead, these biological parameters were determined and recorded.
Each species in these tests were exposed for the same time period 96 h. As in any bioassay that determines a dose response, the LC50 value can be archived at any time during the exposure.
-Data analysis
-Estimations of LC50 values. The LC50 values were estimated by an interpolation method using a computer program written for aquatic toxicity studies. The linear interpolation uses the logarithm transformation of the concentration and the angle transformation of the percent dead between the two doses that bracket 50 % response (i.e., proportion killed). The moving average and probit methods could not be used because the required minimum of two partial mortalities was rarely obtained.

-Number of times to test
The compound was tested twice.

Reliability: (3) invalid
This study should be regarded as a range-finding test

14-DEC-2005 (41)

Type: static
Species: other: Helisoma trivolvis
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: 4.8

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: Reagent Grade

Remark: -Test species:
Common name; Snail
Genus and species; Helisoma trivolvis
Phylum; Mollusca
Approximate size; 0.180 g

Result: -1st result.
LC50 = 4.8 mg/L

-2nd result.
LC50 = 4.8 mg/L

LC50(median lethal concentration)
Endpoint: mortality

Test condition: -Test vessel and diluent water
The static bioassays were performed in seamless glass, 30.5-cm cuboidal, Pyrex chromatography jars to which 20 liters of test solution was added. Water quality was routinely monitored to characterize the diluent water and ensure its suitability. Activated carbon-filtered, dechlorinated and tempered industrial service water from Lake Ontario was used in each test.

-Test solutions
Based on a recommended expected environmental concentration cutoff level, the maximum concentration used in these bioassays was 100 mg/L. Chemical known to be readily soluble at 100 mg/L were added directly to the diluent water in each aquarium in the appropriate amounts to give nominal concentrations of 100, 10, 1 and 0.1 mg/L. The chemical concentrations were not analyzed. Once the test solutions were prepared, the starting temperature, dissolved oxygen and pH values were determined for each exposure concentration and the control. When the starting pH of the test solution fell outside the extremes of 6.5 to 8.5, the pH was adjusted to 7.0 by addition of 10 % (v/v) NaOH or 10% (v/v) H2SO4.

-The dilution water
Total dissolved solids: 180 mg/L
Total hardness (CaCO3): 130 mg/L
Noncarbonate hardness (as CaCO3): 38 mg/L
Alkalinity (as CaCO3): 93 mg/L
Soluble sulfate (SO4--): 31 mg/L
Chloride by I.C. (as Cl-): 26 mg/L
Total NH3 (as N): 0.06 mg/L
Organic nitrogen: 0.21 mg/L
NO3- (as N): 0.29 mg/L
NO2- (as N): <0.01 mg/L
Molybdenum-reactive dissolved SiO2: 0.35 mg/L
Total phosphorus (P): 0.02 mg/L
Total cyanide (CN-): <0.005 mg/L
Soluble fluoride (F-): 0.35 mg/L
pH: 7.4 pH units
Conductivity: 260 micro-mhos/cm
Total organic carbon: 1.8 mg/L
Total residual Cl2: 6.6 micro-g/L

-Test organisms
Each organism was acclimated to the control diluent water in the breeding/rearing tanks. Food was withheld for 24 h preceding start of the test. Juveniles of each species, as uniform in size as possible, were collected from the colonies. Ten juvenile organisms of each species were exposed to the test chemical in each treatment. Stratified randomization was accomplished by proportioning out no more

than 20% of any one species into an aquarium at any one time. Biological loading was kept below 0.5 gram wet weight per liter of test solution.

-Physical/chemical parameter
Determinations of the temperature, dissolved oxygen and pH of each test solution were made in conjunction with the daily biological observations. Test temperature target was 20 plus or minus 1 degree C. If the dissolved oxygen concentration in a test chamber fell below 40% of the starting level in a test, the test was repeated with 0.05 L/min glass-sparger aeration. Each test was conducted within the extremes of 6.5 to 8.5 pH units. The photoperiod duration was 16 h of light. The air-water interface of each tank received approximately 50 ft-c of cool-white fluorescent light.
-Biological parameters
Biological observations were made daily. Survival, condition and behavioral information were recorded. Dead organisms were removed when observed. A test organism was considered dead if it appeared motionless and exhibited no response to gentle prodding. If more than one-half of the population of a species exposed in any treatment was determined to be dead, additional aquaria containing lower concentrations of test solution were set up. At any time during the test when each organism of a species were considered dead, these biological parameters were determined and recorded.
Each species in these tests were exposed for the same time period 96 h. As in any bioassay that determines a dose response, the LC50 value can be archived at any time during the exposure.

-Data analysis
-Estimations of LC50 values. The LC50 values were estimated by an interpolation method using a computer program written for aquatic toxicity studies. The linear interpolation uses the logarithm transformation of the concentration and the angle transformation of the percent dead between the two doses that bracket 50 % response (i.e., proportion killed). The moving average and probit methods could not be used because the required minimum of two partial mortalities was rarely obtained.

-Number of times to test
The compound was tested twice.

Reliability: (3) invalid
This study should be regarded as a range-finding test

14-DEC-2005 (41)

Type: static
Species: other: Lumbriculus variegatus
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: .1 - .32

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: Reagent Grade

Remark: -Test species:

Common name; Segmented worm
Genus and species; Lumbriculus variegatus
Phylum; Annelida
Approximate size; 0.006 g

Result: -1st result.
LC50 = 0.32 mg/L

-2nd result.
LC50 = 0.10 mg/L
LC50(median lethal concentration)
Endpoint: mortality

Test condition: -Test vessel and diluent water
The static bioassays were performed in seamless glass, 30.5-cm cuboidal, Pyrex chromatography jars to which 20 liters of test solution was added. Water quality was routinely monitored to characterize the diluent water and ensure its suitability. Activated carbon-filtered, dechlorinated and tempered industrial service water from Lake Ontario was used in each test.

-Test solutions
Based on a recommended expected environmental concentration cutoff level, the maximum concentration used in these bioassays was 100 mg/L. Chemical known to be readily soluble at 100 mg/L were added directly to the diluent water in each aquarium in the appropriate amounts to give nominal concentrations of 100, 10, 1 and 0.1 mg/L. The chemical concentrations were not analyzed. Once the test solutions were prepared, the starting temperature, dissolved oxygen and pH values were determined for each exposure concentration and the control. When the starting pH of the test solution fell outside the extremes of 6.5 to 8.5, the pH was adjusted to 7.0 by addition of 10 % (v/v) NaOH or 10% (v/v) H2SO4.

-The dilution water
Total dissolved solids: 180 mg/L
Total hardness (CaCO3): 130 mg/L
Noncarbonate hardness (as CaCO3): 38 mg/L
Alkalinity (as CaCO3): 93 mg/L
Soluble sulfate (SO4--): 31 mg/L
Chloride by I.C. (as Cl-): 26 mg/L
Total NH3 (as N): 0.06 mg/L
Organic nitrogen: 0.21 mg/L
NO3- (as N): 0.29 mg/L
NO2- (as N): <0.01 mg/L
Molybdenum-reactive dissolved SiO2: 0.35 mg/L
Total phosphorus (P): 0.02 mg/L
Total cyanide (CN-): <0.005 mg/L
Soluble fluoride (F-): 0.35 mg/L
pH: 7.4 pH units
Conductivity: 260 micro-mhos/cm
Total organic carbon: 1.8 mg/L
Total residual Cl2: 6.6 micro-g/L

-Test organisms
Each organism was acclimated to the control diluent water in the breeding/rearing tanks. Food was withheld for 24 h preceding start of the test. Juveniles of each species, as uniform in size as possible, were collected from the

colonies. Ten juvenile organisms of each species were exposed to the test chemical in each treatment. Stratified randomization was accomplished by proportioning out no more than 20% of any one species into an aquarium at any one time. Biological loading was kept below 0.5 gram wet weight per liter of test solution.

-Physical/chemical parameter
Determinations of the temperature, dissolved oxygen and pH of each test solution were made in conjunction with the daily biological observations. Test temperature target was 20 plus or minus 1 degree C. If the dissolved oxygen concentration in a test chamber fell below 40% of the starting level in a test, the test was repeated with 0.05 L/min glass-sparger aeration. Each test was conducted within the extremes of 6.5 to 8.5 pH units. The photoperiod duration was 16 h of light. The air-water interface of each tank received approximately 50 ft-c of cool-white fluorescent light.

-Biological parameters
Biological observations were made daily. Survival, condition and behavioral information were recorded. Dead organisms were removed when observed. A test organism was considered dead if it appeared motionless and exhibited no response to gentle prodding. If more than one-half of the population of a species exposed in any treatment was determined to be dead, additional aquaria containing lower concentrations of test solution were set up. At any time during the test when each organism of a species were considered dead, these biological parameters were determined and recorded.
Each species in these tests were exposed for the same time period 96 h. As in any bioassay that determines a dose response, the LC50 value can be archived at any time during the exposure.

-Data analysis
-Estimations of LC50 values. The LC50 values were estimated by an interpolation method using a computer program written for aquatic toxicity studies. The linear interpolation uses the logarithm transformation of the concentration and the angle transformation of the percent dead between the two doses that bracket 50 % response (i.e., proportion killed). The moving average and probit methods could not be used because the required minimum of two partial mortalities was rarely obtained.

-Number of times to test
The compound was tested twice.

Reliability: (3) invalid
This study should be regarded as a range-finding test

14-DEC-2005 (41)

Type: static
Species: other: Ophryotrocha diadema
Exposure period: 96 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: .33 - 1

Year: 1984
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Species: Ophryotrocha diadema,
Common name: Polychaete, Annelida
Test condition: -Water parameter
Salinity: 32 ppt (dilution water) (mean value)

-Effect
mortality

-Endpoint
LC50: lethal concentration for 50% of test organisms

-Stock cultures
Cultures of Ophryotrocha diadema were supplied by Professor Bertil Akesson, Department of Zoology, University of Gothenburg, Sweden. The biology of O. diadema in the wild is as yet undescribed; in cultures it is normally vagile but forms tube-like structures when mating and producing eggs. Stock cultures were kept in glass bowls and were prepared using filtered sea water adjusted to a salinity of 32 o/oo by the addition of distilled water. The cultures were fed using fragmented spinach and maintained at 21 degree C. The water was changed weekly and fresh subcultures prepared every 2 months.

-Acute toxicity tests
The 48-h LC50 tests were carried out using O. diadema at each concentration in duplicated trials. The animals were starved for 2 days before a test, and no food was provided during the test. For control cultures, the medium use was 49 mL of sterile, filtered sea water. Test cultures were prepared by adding 1 mL of an appropriate dilution in distilled water of the chemical to 49 mL of sea water. For each test a half-logarithmic series of concentrations was used following the convention of Portman.
After the 48 h exposure period, surviving animals were transferred into clean sea water, as were the control animals, where they were allowed one week to recover. Any animals which died during this period were recorded along with the earlier mortalities in the test.
Reliability: (2) valid with restrictions
30-NOV-2005 (112)

Type: static
Species: other: Mercenaria mercenaria
Exposure period: 48 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: 1.03

Year: 1979
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Species: Mercenaria mercenaria.
Common name: Northern quahog or hard clam
Test condition: -Life stage: egg

-Water parameter

Temperature: 24 degree C (mean value)

-Effect
development change, general

-Endpoint
EC50: Effect concentration for 50% of test organisms
Reliability: (4) not assignable
30-NOV-2005 (38)

Type: semistatic
Species: other: Mercenaria mercenaria
Exposure period: 12 day(s)
Unit: mg/l **Analytical monitoring:** no
EC50: < .25
NR-LETH : = .25

Year: 1979
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Species: Mercenaria mercenaria.
Common name: Northern quahog or hard clam
Result: LC50 < 0.25 mg/L (12 days)
NR-LRTH = 0.25 mg/L (<= 12 days)
Test condition: -Age: 2 day, larvae

-Water parameter
Temperature: 24 degree C (mean value)

-Effect
mortality

-Endpoint
LC50: lethal concentration for 50% of test organisms
NR-LETH: 100% mortality or 0% survival
Reliability: (4) not assignable
30-NOV-2005 (38)

Type: static
Species: Crangon crangon (Crustacea)
Exposure period: 48 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: 1 - 10

Year: 1972
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Common name: Common shrimp, sand shrimp
Test condition: Water temperature: 15 degree C
Reliability: (4) not assignable
Data reported in the secondary literature
30-NOV-2005 (116)

Type: static
Species: other: Cerastoderma edule (cockle)
Exposure period: 48 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: > 100

Year: 1972
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Test condition: Water temperature: 15 degree C
Reliability: (4) not assignable
Data reported in the secondary literature
30-NOV-2005 (116)

Type: semistatic
Species: Crangon crangon (Crustacea)
Exposure period: 48 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: 1 - 10

Year: 1971
Test substance: other TS:2-Propen-1-ol (CAS No. 107-18-6)

Test condition: Water temperature: 15 deg C
Reliability: (4) not assignable
Data reported in the secondary literature
30-NOV-2005 (117)

Type: semistatic
Species: other: Cerastoderma edule (cockle)
Exposure period: 48 hour(s)
Unit: mg/l **Analytical monitoring:** no
EC50: > 100

Year: 1971
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Test condition: Water temperature: 15 deg C
Reliability: (4) not assignable
Data reported in the secondary literature
30-NOV-2005 (117)

Type: static
Species: Daphnia magna (Crustacea)
Exposure period: 24 hour(s)
Unit: mg/l **Analytical monitoring:** no data
EC50: 4.9

Year: 1982
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Test condition: Effect - behavior; quantifiable change in activity, including trained behavior
Reliability: (4) not assignable
Data reported in the secondary literature
30-NOV-2005 (157)

4.3 Toxicity to Aquatic Plants e.g. Algae

Species: other algae: Pseudokirchneriella subcapitata
Endpoint: other: biomass and growth rate
Exposure period: 72 hour(s)
Unit: mg/l **Analytical monitoring:** yes
EC50 (biomass) : 2.25

EC50 (growth rate).5.38
NOEC (biomass & gr.93h rate) :.93
Limit Test: no

Method: OECD Guide-line 201 "Algae, Growth Inhibition Test"
Year: 2005
GLP: yes
Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), purity 99.38%, Lot No. CX30609214, Sigma-Aldrich

Result: Analytical results presented as means over the test period, adjusted means (corrected for mean recovery of 2-propen-1-ol from spiked samples; 110.7%) and geometric means (uncorrected and corrected for mean recovery of 2-propen-1-ol from spiked samples; 110.7%) are shown in Table 1.

The adjusted geometric mean concentrations of 2-propen-1-ol (considered by the authors to give the best representation of actual test concentrations) were 0, 0.343, 0.930, 2.41, 6.03 and 9.12 mg/L.

Table 1: Measured concentrations of 2-propen-1-ol in test solutions during the 72-hr toxicity test.

Measured Concentration (% nominal)				
Nominal Conc (mg/L)	Mean 0-72h (mg/L)	Adjusted Mean (a) (mg/L)	Adjusted Geometric Mean (mg/L)	Geometric Mean (a) (mg/L)
Control	-	-	-	-
0.65	0.383 (59)	0.346 (53)	0.380 (58)	0.343 (53)
1.3	1.06 (82)	0.958 (74)	1.03 (79)	0.930 (72)
2.5	2.83 (113)	2.56 (102)	2.67 (107)	2.41 (96)
5.0	6.98 (140)	6.31 (126)	6.67 (133)	6.03 (120)
10	10.4 (104)	9.39 (94)	10.1 (101)	9.12 (91)

(a) Results corrected for mean 2-propen-1-ol recovery as determined in duplicate spiked samples of test medium.

Table 2: Mean Cell Density

Nominal Concentration (mg/L)	Mean Cell Density (x 10000 cells/mL)(a) 24h	48h	72h	Percent Difference (b)
Control	4.6	22	118	-
0.65	4.5	23	124	+5

1.3	3.8*	22	116	-2
2.5	1.7*	9.1	54*	-54
5.0	0.81*	1.7*	3.2*	-97
10	0.56*	0.67*	1.0*	-99

(a) Values are means of triplicate test chambers
(b) Percent difference at 72h compared to control
* Significant reduction as compared to the control (Dunnett's test, p<0.05)

Table 3: Mean area under the growth curve

Nominal Concentration (mg/L)	Mean Area Under Growth Curve (a) 0-24h	0-48h	0-72h	Percent Difference (b)
Control	43	330	2000	-
0.65	42	350	2100	+5
1.3	34*	320	2000	0
2.5	8.4*	110*	850*	-58
5.0	-2.3*	4.2*	39*	-98
10	-5.3*	-15*	-18*	-100

(a) Values are means of triplicate test chambers and rounded to two significant figures
(b) Percent difference at 72h compared to control
* Significant reduction as compared to the control (Dunnett's test, p<0.05)

Table 4: Mean growth rate values

Nominal Concentration (mg/L)	Mean Growth Rate (cells/mL/h) (a) 0-24h	0-48h	0-72h	Percent Difference (b)
Control	0.064	0.064	0.066	-
0.65	0.063	0.065	0.067	+2
1.3	0.055	0.065	0.066	0
2.5	0.022*	0.046*	0.055*	-17
5.0	-0.011*	0.011*	0.016*	-76
10	-0.025*	-0.0085*	0.00014*	-100

(a) Values are means of triplicate test chambers and rounded to two significant figures
(b) Percent difference at 72h compared to control
* Significant reduction as compared to the control (Dunnett's test, p<0.05)

RESULTS BASED ON NOMINAL CONCENTRATIONS:

Hour	EC Type	EC Value (mg total product/L)	95% CI (mg total product/L)	NOEC (mg total product/L)

Hour				
24	EbC50	2.2	2.0-2.3	0.65
	ErC50	2.3	2.0-2.7	1.3
48	EbC50	2.2	2.1-2.4	1.3
	ErC50	3.3	2.7-3.8	1.3
72	EbC50	2.4	2.3-2.4	1.3
	ErC50	3.8	3.5-4.0	1.3

RESULTS BASED ON ADJUSTED GEOMETRIC MEAN CONCENTRATIONS:

Hour	EC Type	EC Value (mg/L)	95% CI (mg/L)	NOEC (mg/L)
24	EbC50	2.09	1.95-2.23	0.343
	ErC50	2.26	1.90-2.61	0.930
48	EbC50	2.11	1.77-2.46	0.930
	ErC50	5.14	4.79-5.50	0.930
72	EbC50	2.25	2.21-2.30	0.930
	ErC50	5.38	5.28-5.47	0.930

Test condition: - Test Organisms:Pseudokirchneriella subcapitata (former name; Selenastrum capricornutum)
a) Supplier/Source: Department of Botany, Culture Collection of Algae, University of Texas, Austin, Texas, USA
b) Method of Cultivation: The prepared cultures were maintained in a temperature-controlled environmental chamber under continuous light. Periodically, new cultures were cloned from an existing culture derived from the parent stock
c) Any pretreatment: All cultures were maintained under the same conditions as those used for testing. The algal culture used for this test was four days old at test initiation.

- Test Conditions:
a) Medium: Freshwater algal nutrient medium (ASTM E1218-97a).
b) Exposure Vessel Type: 259 mL Erlenmeyer flasks with foam stoppers containing 100 mL of solution.
c) Nominal Concentrations: 0 (control), 0.65, 1.3, 2.5, 5.0, 10 mg/L
d) Vehicle/Solvent and Concentrations: Test medium
e) Stock Solution: A 10 mg/L primary standard was prepared by diluting 0.0102 g of 2-propen-1-ol to 1L volume with test medium. The primary standard was used at the highest treatment and the four lower treatments were prepared individually by diluting appropriate volumes of the primary standard with test medium.
f) Number of Replicates: 3
g) Initial Cell Number: 10,000 cells/mL
h) Water Temperature: 24+/-2 deg C
i) pH: 7.5 (0h) - 9.2 (72h)
j) Light Condition: Continuous cool-white fluorescent lighting at 8,561 - 8,679 lux.
k) Shaking: 100 rpm

- Methods of Analysis: Samples were collected from the control

and each test substance treatment at 0 and 72 hours of the test. The 0 hour samples were collected from the parent solutions. The 72 hour saomples were collected from pooled solutions after combining replicate solutions by treatment. Sampling began with the control and continued up to the highest test substance treatment. Each sample volume was approximately 50 mL. Each sample was acidified to a pH <2.0 with HCl then transferred into a 40 mL glass vial, filling the vial completely and then sealed leaving no headspace. The samples were analyzed in accordance with EPA Method 8620 using GC/MS. Sample introduction into the GC/MS was accomplished using the heated purge and trap Method 5030.

- Statistical Method:

NOEC estimates: Estimated using a one-way analysis of variance (ANOVA) procedure and a two-tailed Dunnett's test. Prior to the Dunnett's test, a Shapiro-Wilk's test and a Levene's test were conducted to test for normality and homogeneity of variance, respectively, over treatments at each time point. If the results from the Shapiro-Wilk's and Levene's tests indicated normality and insignificant heterogeneity, the analysis was performed on the non-transformed raw data. In instances of non-normality or heterogeneity, a square root transformation was performed. If both the non-transformed raw data and the transformed data exhibited non-normality or inequality of variance, a non-parametric analysis of variance was performed on the ranks of the raw data values. Non-parametric analyses were performed on the 48- and 72-hour growth rate data. Parametric analyses were performed on the 24-, 48-, and 72-hour area under the growth curve and the 24-hour growth rate data.

EC estimates: ErC50 and EbC50 estimates were calculated using a logistic (sigmoid-shaped) model fit to the data with percent inhibition as the dependent variable and concentration as the independent variable.

Method of Calculating Mean Measured Concentrations: Adjusted Geometric mean

Reliability: (1) valid without restriction
OECD TG study

06-JAN-2006 (90)

Species: other algae: Pseudokirchneriella subcapitata
Endpoint: other: biomass and growth rate
Exposure period: 72 hour(s)
Unit: mg/l **Analytical monitoring:** yes
NOEC (growth rate 1.4 biomass) :1.4
EbC50 (biomass) : 2.58
ErC50 (growth rate787.8

Method: OECD Guide-line 201 "Algae, Growth Inhibition Test"
Year: 2004
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6) Supplier: SHOWA DENKO K.K., Lot No.: 040420, Purity: 99.9%

Remark: The EC50 and NOEC were determined using measured concentrations at the start.

Result: - Measured Concentrations : The concentrations of 2-propen-1-ol were measured at the start and after 72 hours of exposure.

The percent of the measured concentrations to the nominal values was 70 - 91% at the start and 70 - 86% after 72 hours of exposure, showing that all deviations from the nominal were not less than +/- 20%. Therefore, the measured concentrations at the start of exposure were used for each calculation.

Nominal Conc. (mg/L)	Measured Concentration (mg/L) 0hr	Percent of Nominal	72 hrs	Percent of Nominal
Control	<0.3 ---		<0.3	---
2.0	1.4	70	1.4	70
3.8	3.0	79	3.0	79
7.2	6.1	85	5.8	81
14.0	12.4	89	12.0	86
26.0	23.6	91	22.2	85
50.0	45.5	91	42.4	85

- Water chemistry (pH) and temperature in test:
The pH at 0 and 72 h was 7.9 and 7.8 - 8.8, respectively.
The temperature at 0, 24, 48 and 72 h was 23.4, 21.9, 22.5 and 22.8 degree C, respectively.

-Effect Data:
Area Method (growth curve)
EC50 (biomass; 0-72hr) = 2.58 mg/L
NOEC (biomass; 0-72hr) = 1.4 mg/L

Rate Method
ErC50 (growth rate; 0-72hr) = 7.80 mg/L (95% C. I.: 5.10 - 11.9 mg/L)
NOEC (growth rate; 0-72hr) = 1.4 mg/L

Growth Inhibition (%)of Pseudokirchneriella subcapitata (Area Method)

Nominal Conc. [Measured Conc. at 0hr] mg/L	Area under the growth curves Area A (0-72h)	Inhibition (%)*1 IA (0-72h)
Control	46744000	---
2.0 [1.4]	48657000	-4.1
3.8 [3.0]	17676000	62.2**
7.2 [6.1]	7691000	83.5**
14.0 [12.4]	761000	98.4++
26.0 [23.6]	191000	99.6++
50.0 [45.5]	120000	99.7++

++
*1 Values are the growth inhibition (%) relative to the control.
** Indicates a significant difference (alpha = 0.01) from the control.
++ Statistical comparison test could not be performed for these concentrations since data including these concentrations did not show homogeneity of variances. However, it was concluded that these concentration levels showed adverse effect on algal growth judging from IA values.

Growth Inhibition (%) of Pseudokirchneriella subcapitata (Rate Method)

Nominal Conc.[Measured Conc. at 0hr] mg/L	Growth rates and percent inhibition (Average) Rate u (0-72h)	Inhibition (%)*1 Im (0-72hr)
Control	0.0792	---
2.0 [1.4]	0.0800	-1.0
3.8 [3.0]	0.0660	16.7**
7.2 [6.1]	0.0541	31.7**
14.0 [12.4]	0.0212	73.2**
26.0 [23.6]	0.0057	92.8**
50.0 [45.5]	0.0028	96.5**

++
*1 Values are the growth inhibition (%) relative to the control.
** Indicates a significant difference (alpha = 0.01) from the control.
++ Statistical comparison test could not be performed for these concentrations since data including these concentrations did not show homogeneity of variances. However, it was concluded that these concentration levels showed adverse effect on algal growth judging from IA values.

- Growth Curves: Log phase during the test period

- Calculation of toxic value: Measured concentrations were used.

The test period (ex. 0-72hr), which was suitable, was used for the calculations.

Test condition: - Test Organisms:Pseudokirchneriella subcapitata
a) Supplier/Source: Obtained from American Type Culture Collection
b) Method of Cultivation: Sterile
c) Stain Number: ATCC22662
d) Any pretreatment: Acclimated for 3 days before testing, any groups observed abnormal cells or cellular deformation

were not used for testing.

- Test Conditions:
a) Medium: OECD medium in TG201
b) Exposure Vessel Type: 100 mL Medium in a 300mL flask , static closed system)
c) Nominal Concentrations: control, solvent control, 2.0 3.8, 7.2, 14.0, 26.0 and 50.0 mg/L
d) Vehicle/Solvent and Concentrations: not used
e) Stock Solution: preparation for use
f) Number of Replicates: 3
g) Initial Cell Number: 10,000 cells/mL
h) Water Temperature: 23 plus or minus 2 degree C
i) Light Condition: 4,000 lux plus or minus 20 %, continuously
j) Shaking: 100 rpm

- Methods of Analysis: Test concentrations were measured at the start and the 72 hour. Start of exposure: extraction of the sample (100 mL). End of test: proper quantity extraction of the sample was carried out from the examination tank (3/concentration division) of each concentration division, and equivalent mixture was carried out and it considered as the liquid for analysis of 100mL(s). It measured after centrifugal separation to remove the algae. It measured by GC/MS. It computed from the peak area ratio.

- Statistical Method:
a) Data Analysis: linear regression method (method of least squares) for EC50, 1-way ANOVA and Dunnett method for NOEC
b) Method of Calculating Mean Measured Concentrations (i.e. arithmetic mean, geometric mean, etc.): method of least squares with standard curve

Reliability: (1) valid without restriction
OECD TG study

03-JAN-2006 (5)

Species: other algae: Pseudokirchneriella subcapitata
Endpoint: other: biomass and growth rate
Exposure period: 72 hour(s)
Unit: mg/l **Analytical monitoring:** yes
NOECr (0-48h) : 2.2
NOECb (0-72h) : 6.09
Eb,rC50 (0-48h) : 11

Method: OECD Guide-line 201 "Algae, Growth Inhibition Test"
Year: 2003
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Supplier: Wako Pure Chemical Industries, Ltd., Purity: >=99.0%, Lot No. WAN5739

Method: -Test Organisms
a) Strain Number: ATCC22662
b) Supplier/Source: American Type Culture Collection
c) Pretreatment: Precultured for 3 days in OECD medium before use.

-Test Conditions

a) Medium: OECD medium
b) Exposure Vessel Type: 100-ml Medium in a 300-ml Conical Flask with a glass stopper (closed system)
c) Nominal Concentrations (as mg/L): 0, 0.22, 0.46, 1.0, 2.2, 4.6 and 10
d) Vehicle/Solvent and Concentrations: Not used
e) Number of Replicates: 3
f) Initial Cell Number: 10,000 cells/ml
g) Water Temperature: 23±2 deg C
h) Light Condition: 4,000 - 5,000 lux, continuous

-Analytical Procedure
Portions of the test solutions were withdrawn at 0 hour and 72 hours and concentrations of the test substance were determined by GC-MS.

-Statistical Method
a) Data Analysis: Probit method for EC50 and Dunnett's multicomparison method for NOEC
b) Method of Calculating Mean Measured Concentrations: arithimetic mean

Result: -Measured Concentrations
Measured concentrations at the beginning (0 hr) and the end (72 hr) of the test were ranged within 92 - 100% and 85 - 106% of the nominal concentrations, respectively.

Table 1. Measured Concentration of the Test Substance in Test Water

Nominal Conc. (mg/L)	Measured Concentration (mg/L) 0 Hour	Percent of Nominal	72 Hours Conc(mg/L)	Percent of Nominal	Mean Measured
Control	<0.01	-	<0.01	-	-
0.22	0.219	100	0.188	85	0.204
0.46	0.436	95	0.486	106	0.461
1.0	0.918	92	0.957	96	0.938
2.2	2.13	97	2.04	93	2.09
4.6	4.27	93	4.55	99	4.41
10	9.51	95	9.87	99	9.69

-Water Chemistry
Temperature : 23.0 deg C
pH : 8.9 - 9.2 (0 hr), 9.0 - 10.3 (72 hr)

-Element Value
Area method:
EbC50 (0 - 72 hr) = 11 mg/L
NOEC (0 - 72 hr) = 2.2 mg/L
Rate method:
ErC50 (0 - 24 hr) = 6.9 mg/L
ErC50 (0 - 48 hr) = 11mg/L
NOEC (0 - 48 hr) = 2.2 mg/L
Table 2. Cell Densities (average) of Pseudokirchneriella subcapitata
during the 72-hours Exposure

Nominal Conc. mg/L	Cell Density (x10000 cells/mL) 0 hour	24 hours	48 hours	72 hours
Control	1.00	7.33	21.58	34.42
0.22	1.00	6.67	21.58	32.25
0.46	1.00	7.42	20.52	33.25
1.0	1.00	8.17	26.75	33.33
2.2	1.00	6.00	20.58	34.00
4.6	1.00	4.00	14.75	27.17
10	1.00	1.92	5.67	11.42

Table 3-1. Percent Growth Inhibition of Pseudokirchneriella subcapitata
(Area method)

Nominal Conc. (Mean Measured Conc) (mg/L)	Area under the growth curve Area A(0-72) (average)	Inhibition (%)*1 IA(0-72)
Control	1047.0	-
0.22 (0.204)	1005.0	4.01
0.46 (0.461)	1009.0	3.63
1.0 (0.938)	1178.0	-12.51NS
2.2 (2.09)	986.0	5.83NS
4.6 (4.41)	716.0	31.61NS
10 (9.69)	259.0	75.26**

*1: Values are the percent inhibition relative to the control.
**: Significant difference p<0.01
NS: No significant difference

Table 3-2. Percent Growth Inhibition of Pseudokirchneriella subcapitata
(Rate Method)

Nominal	Growth Rate

Conc. (Mean Measured Conc.) (mg/L)	r(0-24)	Inhibition (%)*1 Im(0-24)	r(0-48)	Inhibition (%)*1 Im(0-48)	r(0-72)	Inhibition (%)*1 Im(0-72)
Control	1.99	-	1.54	-	1.18	-
0.22 (0.204)	1.90	4.5	1.54	0	1.16	1.1
0.46 (0.461)	2.00	-0.5	1.51	1.9	1.17	0.8
1.0 (0.938)	2.10	-5.5	1.64	6.5	1.17	0.8
2.2 (2.09)	1.79	10.1	1.51	1.9	1.18	0
4.6 (4.41)	1.39	30.2	1.35	12.3	1.10	6.8
10 (9.69)	0.65	67.3	0.87	43.5	0.81	31.4

*1: Values are the percent inhibition relative to the control.
NS: No significant difference

Table 4. Calculated EC50 and NOEC

	EC50 (mg/L)	95% Confidence Limits (mg/L)	NOEC (mg/L)
EbC50 (0-72)*1	11	9.2 - 13	NOEC(Area 0-72) 2.2
ErC50 (0-24)*2	6.9	6.2 - 7.7	-
ErC50 (0-48)*3	11	9.8 - 13	NOEC (Rate 0-48) 2.2
ErC50 (0-72)*4	41	24 - 100	-

*1: Based on Ia(0-72h) value (Areas under the growth curve)
*2: Based on Im(0-24h) value (Growth rates)
*3: Based on Im (0-48h) value (Growth rates)
*4: Based on Im (0-72h) value (Growth rates)

Pseudokirchneriella subcapitata was exposed under static conditions to 2-propen-1-ol at measured concentrations of 0, 0.20, 0.46, 0.94, 2.1, 4.4 and 9.7 mg/L for 72 hours. The 48-h ErC50 from a linear portion of the growth curve and the 72-h EbC50 by biomass were calculated to be 11 mg/L.

Conclusion: EC50 (0-48) of 2-propen-1-ol was 11 mg/L and NOEC (biomass) was 2.2 mg/L.

Reliability: (1) valid without restriction
OECD TG study

06-JAN-2006 (100)

Species: Scenedesmus subspicatus (Algae)
Exposure period: 7 day(s)
Unit: mg/l **Analytical monitoring:** no
EC03 : .6

Year: 1982
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Test condition: Effect: histology: presence of physical damage to tissues
Reliability: (4) not assignable
03-JAN-2006 (157)

4.4 Toxicity to Microorganisms e.g. Bacteria

Species: Tetrahymena pyriformis (Protozoa)
Exposure period: 2 day(s)
Unit: mg/l **Analytical monitoring:**
EC50: 4806

Year: 1994
GLP: no data
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity >= 95%

Result: IC50 = 82.755 mmol/L

converted value
IC50 = 4806 mg/L
Test condition: Effect: population, change in number of species groups in community

IC50: Median Inhibition Concentration (50% inhibition of biological process)

This 2-d assay uses cell density as its endpoint. Population levels were measured spectrophotometrically as absorbance at 540 nm. Each chemical was assayed in a range-finder, followed by definitive testing as duplicates for three or more replicates. Each replicate was at minimum at a six-step arithmetic concentration series using freshly prepared stock solutions. Stock solutions were prepared in dimethyl sulfoxide (DMSO) at concentration of 5, 10, 25, or 50 g/liter. In every case, the volume of stock solution added to each flask was limited so the final DMSO concentration did not exceed 0.75%, an amount that dose not alter Tetraphymena reproduction. Only replicates with control absorbance values from 0.6 to 0.9 were used in the analyses. The 50% growth inhibitory concentration, IC50, was determined for the alcohol using Probit Analysis of Statistical Analysis System software with Y as the absorbance normalized as percentage of control and X as the toxicant concentration in ppm. This 50% effect concentration was adjusted from pipetted amount to weight concentration by multiplying by density.
Reliability: (2) valid with restrictions
Flag: Critical study for SIDS endpoint
30-NOV-2005 (140)

Species: other bacteria
Unit: mg/l **Analytical monitoring:**

EC10: 1285

Year: 1982
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (4) not assignable
03-JAN-2006 (157)

4.5 Chronic Toxicity to Aquatic Organisms

4.5.1 Chronic Toxicity to Fish

4.5.2 Chronic Toxicity to Aquatic Invertebrates

Species: Daphnia magna (Crustacea)
Endpoint: reproduction rate
Exposure period: 21 day(s)
Unit: mg/l **Analytical monitoring:** yes
NOEC: .919
EC50: > .919
LC50 : 1.36

Method: OECD Guide-line 211
Year: 2003
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Supplier: WaKo Pure Chemical Industries, Ltd., Purity >=99.0%, Lot No.: WAN5739

Method: -Test Organisms
a) Age: < 24 hours old
b) Pretreatment: The group of parent showing less than 5 % mortality for 14 days before testing was used.
c) Supplier / Source: National Institute for Environmental Studies (JAPAN)

-Test Conditions
a) Dilution Water Source: Laboratory supply water (dechlorinated)
b) Dilution Water Chemistry:
Hardness: 29 - 36 mg/L (as CaCO3)
pH: 7.3 - 7.8
c) Exposure Vessel Type: 80-ml test solution in a 100-ml screw-capped glass bottle (closed system)
d) Nominal Concentrations (as mg/L):
0, 0.046, 0.10, 0.22, 0.46, 1.0 and 2.2
e) Vehicle/Solvent and Concentrations: Not used
f) Number of Replicates: 10
g) Individuals per Replicates: 1
h) Renewal Rate of Test Water: Total solution in a vessel was renewed every 24 hours.
I) Water Temperature: 20.2 - 20.8 deg C
J) Light Condition: 16:8 hours, light-darkness cycle
k) Feeding: Fed on Chlorella vulgaris at 0.15 - 0.20 mg carbon/day/individual.

-Analytical Procedure
Portions of the test solutions were withdrawn at 0 hour, on the 1st day before renewal, on the 10th day after renewal, on the 11th day before renewal, on the 21st day after renewal and on the 22nd day before renewal. The withdrawn samples were determined by GC-MS.

-Statistical Method
a) Data Analysis: Binominal method for LC50, and one-way analysis of variance (ANOVA) for NOEC and LOEC.
b) Method of Calculating Mean Measured Concentrations: Time-weighted mean concentrations.

Result: -Measured Concentrations
Measured concentrations of the test solutions just after renewal (fresh preparation) and those 24 hour after renewal ranged from 93 - 111% and 37 - 97% of the nominal concentrations, respectively. The time-weighted concentrations were used to calculate the effect values (Table 1-1).

Table 1-1. Measured Concentrations of the test Substance in Test Water during a 21-day Exposure Period. (Semi-static Test Condition)

Nominal Conc.	Measured Conc. (mg/L)						TWM*	%of
(mg/L)	Date 0 New	1 old	10 new	11 old	20 new	21 old	(mg/L)	Nominal
Control	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	-	-
0.046	0.046	0.017	0.047	0.026	0.044	0.019	0.031	67
0.10	0.099	0.084	0.097	0.097	0.097	0.075	0.091	91
0.22	0.218	0.183	0.208	0.213	0.244	0.194	0.210	95
0.46	0.450	0.428	0.438	0.437	0.464	0.398	0.436	95
1.0	0.927	0.867	0.973	0.882	0.949	0.917	0.919	92
2.2	2.05	2.05	2.05	1.92	-	-	2.02	92

Table 1-2. Measured Concentrations as a Percentage of Nominal.

Nominal Conc.	Measured Conc. (mg/L)					
(mg/L)	Date 0 New	1 old	10 new	11 old	20 new	21 old
Control	-	-	-	-	-	-
0.046	100	37	102	57	96	41
0.10	99	84	97	97	97	75
0.22	99	83	95	97	111	88
0.46	98	93	95	95	101	87
1.0	93	87	97	88	95	92
2.2	93	93	93	87	-	-

new: Freshly prepared test solution
old: Old test solution before renewal
* :Time-weighted mean measured concentration during 21 days.

-Water Chemistry
Temperature : 20.2 -20.8 deg C
pH : 7.3 - 7.8

Dissolved oxygen (mg/L) : 7.8 - 9.1
Total hardness (mg/L as CaCO3) : 29 - 36

-Effect Data (reproduction)
All of the following effect values were calculated with the measured concentrations.

LC50 (21 days) = 1.36 mg/L (parental mortality) (Table 2-1, 2-2)
(95% Confidence limits: 0.919 - 2.02 mg/L)
EC50 (21 days) = 0.919 mg/L (Table 2-2, 2-2, 3, 4)
NOEC (21 days) = 0.919 mg/l
LOEC (21 days) > 0.919 mg/l

Table 2-1. Cumulative Numbers of Dead Parental Daphnia

Nominal conc. (mg/L)	Days									
	0	1	2	3	4	5	6	7	8	9
Control	0	0	0	0	0	0	0	0	0	0
0.046	0	0	0	0	0	0	0	0	0	0
0.10	0	0	0	0	0	0	0	0	0	0
0.22	0	0	0	0	0	0	0	0	0	0
0.46	0	0	0	0	0	0	0	0	0	0
1.0	0	0	0	0	0	0	0	0	0	0
2.2	0	0	0	0	0	0	0	0	0	0

Table 2-1(continued).

Nominal conc. (mg/L)	Days									
	10	11	12	13	14	15	16	17	18	19
Control	0	0	0	0	0	0	0	0	0	0
0.046	0	0	0	0	0	0	0	0	0	0
0.10	0	0	0	0	0	0	0	0	0	0
0.22	0	0	0	0	0	0	0	0	0	0
0.46	0	0	0	0	0	0	0	0	0	0
1.0	0	0	0	0	0	0	0	0	0	0
2.2	0	1	3	3	3	9	9	10	10	10

Table 2-1(continued).

Nominal conc. (mg/L)	Days	
	20	21
Control	0	0
0.046	0	0
0.10	0	0
0.22	0	0
0.46	0	0
1.0	0	0
2.2	10	10

Table 2-2 Mortality (%) of Parental Daphnia

Nominal conc.	Days					
(mg/L)	1	2	4	7	14	21
Control	0	0	0	0	0	0
0.046	0	0	0	0	0	0
0.10	0	0	0	0	0	0
0.22	0	0	0	0	0	0
0.46	0	0	0	0	0	0
1.0	0	0	0	0	0	0
2.2	0	0	0	0	30	100

Table 3. Time (Days) to First Brood Production.

Vessel No.	Nominal Conc., mg/L (Measured Conc., mg/L*) Control	0.046 (0.031)	0.10 (0.091)	0.22 (0.210)	0.46 (0.436)	1.0 (0.919)	2.2 (2.02)
1	7	8	8	7	7	7	7
2	7	7	8	7	8	7	8
3	7	8	8	8	7	7	8
4	7	7	8	8	7	7	9
5	7	7	7	8	7	7	7
6	7	8	8	8	7	7	8
7	7	8	8	8	8	8	7
8	7	8	8	8	8	8	12
9	9	8	8	8	7	7	8
10	9	7	8	8	9	8	8
Min	7	7	7	7	7	7	7
Max	9	8	8	8	9	8	12

*: Time-weighted mean measured concentration.

Table 4. Mean Cumulative Numbers of Juveniles Produced per Adult Alive for 21 Days (Sigma F1/P)

Nominal conc. (mg/L)	Days						
	7	8	9	10	11	12	13
Control	11.0	11.1	12.9	24.7	33.3	36.8	54.2
0.046	3.6	11.6	11.6	23.3	38.5	38.5	47.6
0.10	1.6	10.0	10.1	12.8	36.9	37.0	41.6
0.22	1.2	11.2	11.2	12.5	37.6	37.6	37.6
0.46	8.3	11.7	13.1	28.9	35.4	37.9	62.6
1.0	6.8	11.9	11.9	26.7	37.4	37.4	56.7
2.2	-	-	-	-	-	-	-

Table 4. (continued)

Nominal conc. (mg/L)	Days							
	14	15	16	17	18	19	20	21
Control	70.3	74.5	85.8	113.1	117.2	117.4	150.0	153.4
0.046	75.0	75.0	80.0	113.1	114.8	114.8	147.8	151.5
0.10	75.9	75.9	80.7	116.4	116.4	116.4	152.6	152.6
0.22	75.0	75.0	75.0	112.3	112.3	112.3	146.7	147.8
0.46	71.3	74.6	91.4	111.2	114.6	118.7	144.8	148.4
1.0	72.7	72.7	87.1	109.8	109.8	119.9	143.1	143.1
2.2	-	-	-	-	-	-	-	-

Table 5. Cumulative Numbers of Juveniles Produced per Adult Alive for 21 Days in Each Test Vessel and Results of Statistical Comparison of the Mean Values (by ANOVA test).

Vessel No.	Nominal Conc., mg/L (Measured Conc., mg/L*) Control	0.046 (0.031)	0.10 (0.091)	0.22 (0.210)	0.46 (0.436)	1.0 (0.919)	2.2 (2.02)
1	152	160	156	138	160	156	D
2	144	163	149	152	145	153	D
3	150	156	151	152	151	152	D
4	166	151	159	155	160	154	D
5	169	160	180	167	155	151	D
6	150	142	147	136	151	136	D
7	142	133	143	132	136	123	D
8	132	153	148	156	137	134	D
9	159	149	148	137	147	131	D
10	170	148	145	153	142	141	D
Mean	153.4	151.5	152.6	147.8	148.4	143.1	-
SD	12.5	9.1	10.8	11.3	8.6	11.6	-
Inhibition rate(%)		1.2	0.5	3.7	3.3	6.7	-
Sig difference		N.S	N.S	N.S	N.S	N.S	-

*: Time-weighted mean measured concentration
D: Were not included for calculation because the parental Daphnia was dead during a 21-days testing period.
N.S: Indicate no-significant difference

Conclusion: All of the group exposed to the highest concentration died, and mean cumulative numbers of juveniles in 1.0 mg/L was more than 50% of control. Thus EC50(21 days) was greater than 1.0 mg/L (time-weighted mean measured concentration: 0.919 mg/L). Because no significant difference was observed in 1.0 mg/L (time-weighted mean measured concentration: 0.919 mg/L), NOEC(21 days) was determined 0.919 mg/L and LOAEL(21 days) was determined greater than 0.919 mg/L.

Reliability: (1) valid without restriction
OECD TG study

Flag: Critical study for SIDS endpoint

06-JAN-2006 (100)

TERRESTRIAL ORGANISMS

4.6.1 Toxicity to Sediment Dwelling Organisms

4.6.2 Toxicity to Terrestrial Plants

Species: Lactuca sativa (Dicotyledon)
Endpoint: other: Germination
Expos. period: 3 day(s)
Unit: mg/l
EC50: 3.3

Year: 1977
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6). The test compound was used as received from the manufacturer without further purification

Result: Concentrations of 2-propen-1-ol at which percentage germination of lettuce fruits (cv. Great Lakes) at 30 degree C is 50 per cent of the control value.

Concentration (mmol/L) for 50 per cent germination (confidence limits for P=0.05) = 0.056 +/- 0.019 mmol/L
Converted value is 3.3 mg/L.
Test condition: Inhibitory activity was expressed at the millimolar concentration of the chemical producing 50 per cent inhibition at the given temperature (30 degree C).
Reliability: (2) valid with restrictions
30-NOV-2005 (126)

4.6.3 Toxicity to Soil Dwelling Organisms

4.6.4 Toxicity to other Non-Mamm. Terrestrial Species

4.7 Biological Effects Monitoring

4.8 Biotransformation and Kinetics

4.9 Additional Remarks

Memo: Acute toxicity to Frog

Remark: Year: 1935

Route: parenteral
Dose: 51 mg/kg
Effects: Behavioural (excitement), Gastrointestinal (nausea or vomiting)
Test substance: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
14-DEC-2005 (3)

5.0 Toxicokinetics, Metabolism and Distribution

Species: other: rat/dog

Method: other: Mechanistic Study
Year: 1958
GLP: no
Test substance: other TS: refer to freetext

Method: Oxygen uptake of tissues of intoxicated rats: Male rats (Long Evans, 90-150 g) were fasted overnight and given single doses of 60 or 120 mg/kg of 2-propen-1-ol intragastrically as a 10% w/v solution in distilled water. The rats were decapitated one or two hours later and the brain, liver and kidneys removed and sliced in a moistened cold chamber for standard manometric measurement of oxygen uptake with the Warburg apparatus. Oxygen uptake was measured every 15 minutes at 37.2°C in a gas phase of 100% oxygen and the incubation period was arbitrarily set at 120 minutes. Mean rates of oxygen uptake and their standard error were calculated, based on 6-12 observations and 3-4 rats.

Inhibition in vitro of oxygen uptake of rat tissues: The same technique was used and the oxygen uptake of normal rat liver and kidney slices was measured every 15 minutes for 150 minutes. Immediately after the 30 minute reading, 2-propen-1-ol was added to the incubation medium, producing final concentrations of 1E-02, 1E-03, 1E-04 and 1E-05 moles/L. Five or six observations were made at each concentration. Mean rates of oxygen uptake were calculated.

Protection studies with dibenzyline and hexamethonium: Twelve male rats (Long Evans, 132-190 g) were randomised into four groups and fasted overnight. The first group was used as controls and the over groups received 120 mg/kg 2-propen-1-ol intragastrically. One group received only the alcohol, the second group received an intramuscular injection of 10 mg/kg dibenzyline hydrochloride three hours before the alcohol and the last group received 10 mg/kg of hexamethonium chloride intramuscularly one hour before the alcohol. The rats were decapitated one hour after the 2-propen-1-ol was administered. The livers were removed, sliced and quadruplicate measurements of oxygen uptake made for each liver. In order to estimate the degree of swelling of liver slices, the percentage of water in the slices was also measured in quadruplicate.

In an acute study, 20 fasted rats which had been injected intramuscularly with 10 mg/kg of dibenxyline hydrochloride three hours previously and 20 untreated rats, were given 120 mg/kg of 2-propen-1-ol intragastrically. Gross signs of toxicity, time of death and number of deaths were observed in both groups for five days.
Quantitation of blood levels of 2-propen-1-ol following intragastric or intravenous administration : Male rats (Long Evans, 280-390 g) were given 2-propen-1-ol (10% w/v in physiological saline) in single doses of 120 mg/kg (oral) or 30 mg/kg (i.v.). Only the rats receiving oral administration

were fasted overnight. Other rats were given the same solution by constant intravenous infusion at the rate of 42, 68 and 100 mg/kg/hr or a 3.9% solution at 23 and 24 mg/kg/hr. The total volume of solution infused never exceeded 0.75 mL. The solution was infused into the femoral vein.

The rats were anesthetised with sodium pentobarbital and light hypnosis maintained throughout the experiment. After laparotomy, a control sample of 0.1 or 0.2 mL was taken. Serial samples were taken from the portal vein after oral administration and from the inferior vena cava after single i.v. injection and during the constant infusion. The amount of 2-propen-1-ol in the blood was determined using a method developed by the authors. When the rats were killed for necropsy, sections of liver were taken for histologic study.

Miscellaneous studies: Tissue swelling in vitro; effect of 2-propen-1-ol on blood pressure, respiration and plasma histamine levels (dog); dehydrogenase inhibition in vitro

Result: Oral administration of 2-propen-1-ol to rats resulted in severe inhibition of oxygen uptake by the liver, moderate stimulation of oxygen uptake by the kidneys, and no alteration in oxygen uptake by cerebral cortex, one and two hours after administration.

Studies of oxygen uptake in vitro showed that rat liver was about 4 times as sensitive to 2-propen-1-ol as kidney, and although kidney oxygen consumption is presumably increased in vitro.

Administration of either Dibenzyline or hexamethonium prior to 2-propen-1-ol provided good protection against both tissue swelling and decrease in oxygen uptake of liver slices. The swelling was well correlated with the oxygen uptake. On the other hand, prophylactic treatment with
Dibenzyline did not significantly alter the lethal effects of ally alcohol.

Intravenous administration produced profound depression of a dog's blood pressure and respiration, and hemoconcentration, signs characteristic of histamine release. However, there was no evidence that 2-propen-1-ol caused histamine release.

Liver dehydrogenases were not inhibited by 2-propen-1-ol or propyl alcohol at levels possible in vivo. Inhibition by acrolein was 400 to 1000 times greater.

The mean levels of 2-propen-1-ol in the portal venous blood of rats given a single oral dose of 120 mg/kg were found to range between 9 and 15 ug/mL during the 15 to 120 minutes after administration. Although individual values varied considerably, the maximum appeared at about 30 to 60 minutes.

The mean levels in vena caval blood after intravenous dosing with 30 mg/kg 2-propen-1-ol indicated rapid disappearance within 15 minutes and almost complete disappearance in an hour.

Respiration, oxygenation of blood and circulation throughout the larger abdominal vessels appeared satisfactory during the

first 60 to 90 minutes of infusion with 23 or 24 mg/kg/hr. Subsequently, there was increasing indication of circulatory insufficiency. The flow through the larger vessels seemed to decrease although respiration still appeared adequate. The blood levels in the rats rose only slowly, indicating that metabolism was proceeding at about the rate of infusion.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (2) valid with restrictions
Well reported published study
09-MAY-2006 (76)

Year: 1989
Test substance: other TS: refer to freetext

Remark: The major site of 2-propen-1-ol metabolism is the liver. Three metabolic pathways have been linked to its hepatic metabolism. The main route of 2-propen-1-ol metabolism is by cytosolic alcohol dehydrogenase (ADH) while catalase and microsomal cytochrome-P450 dependent mixed-function-oxidase (MFO) may be involved in the biotransformation.
Test substance: Name: 2-Propen-1-ol(CAS No. 107-18-6)
Conclusion: 2-Propen-1-ol toxicity is related to its biotransformation into acrolein, a primary toxic metabolite, through the cytosolic enzyme alcohol dehydrogenase (ADH).
Reliability: (2) valid with restrictions
Well reported published study
30-NOV-2005 (18)

In Vitro/in vivo: In vivo
Species: rat

Method: other: Mechanistic Study
Year: 1980
Test substance: other TS: refer to freetext

Method: Animals: male rats (Wistar, 250-300 g) fed on a semisynthetic diet.
2-Propen-1-ol was administered by intraperitoneal injection at a dose of 25 µL/kg. The animals were sacrificed 24 and 48 hours after dosing. Immediately after death, the GSH level in the liver was determined. Histological examination was performed on liver samples.
Result: Within 24 hours after administration of 2-propen-1-ol the livers showed periportal necrosis and elevation of hepatic glutathione. After 48 hours no necrosis was observed in spite of high level of hepatic glutathione.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (19)

Year: 1972
Test substance: other TS: refer to freetext

Remark: The formation of acrolein from 2-propen-1-ol in rat liver was investigated using whole liver homogenates and subcellular fractions from male albino rats incubated with 2-propen-1-ol in the presence or absence of nicotinamide dinucleotide (NAD+). Acrolein was formed in the microsomal preparations and post microsomal supernatants only in the presence of NAD+. In homogenates and mitochondria, acrolein was formed without NAD+, but addition of NAD+ significantly enhanced acrolein formation. The conversion rate to acrolein was 5%.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
Insufficient detail in published study
30-NOV-2005 (141)

In Vitro/in vivo: In vivo
Type: Metabolism
Species: rat

Year: 1972
GLP: no
Test substance: other TS: refer to freetext

Method: ANIMALS AND TREATMENTS
Male SD rats (200g) were pretreated with either saline (0.5 mL, i.p.) or pyrazole (375 mg/kg bw, i.p.; inhibitor of hepatic alcohol dehydrogenase) 2 hr prior to administration of 2-propen-1-ol (0.05 ml/kg bw, i.p.; 2.46 mCi/mmole).

Comment: Assuming a density of 0.85, this regime was equivalent to approx. 42.5 mg 2-propen-1-ol/kg bw.

AUTORADIOGRAPHY
Paraffin sections from liver, lung and kidney were coated with Kodak NTB-2 emulsion, developed for 4 wk and later stained with hemotoxylin and eosin. Comment: since no steps were taken to prevent extraction of unbound radiolabel from the tissue into the organic solvents used to embed the tissues, the author assumed that most of the exposed grains of the emulsion were indicative of label covalently bound to tissue sections.

COVALENT BINDING
Animals were sacrificed 6, 8 or 24 hr post-treatment with 2-propen-1-ol. Samples of liver, lung and kidney were homogenized in 4 volumes of water, and protein/nucleic acid precipitated with an equal volume ice cold 20% trichloroacetic acid. The precipitate was extracted 5 times with 10 ml methanol (60 degrees C) to remove radioactivity (further extractions ineffective at removing any additional label). The pellet was dissolved in NaOH (1.0 N) and aliquots taken for liquid scintillation counting.
Result: Photomicrographs included in the publication show that extensive periportal necrosis was present 24 hr after administration of 2-propen-1-ol, whereas no microscopic changes were visible in lungs or kidney (photomicrographs not presented).

Covalent binding studies demonstrated a time-dependent decrease in amount of label bound in liver, whereas little radioactivity was present in lung and kidney. Autoradiograms (included in report) demonstrated that most of the binding occurred in the periportal zone, with little present in the centrilobular region.

Inhibition of alcohol dehydrogenase fully prevented hepatic necrosis, and decreased the amount of label bound to liver by approx. 80%. Autoradiograms confirmed that binding of 14C-2-propen-1-ol was markedly reduced in periportal hepatocytes. Covalent binding in lung was also decreased by pretreatment with pyrazole, whereas renal binding was unaffected.

Covalent binding data:

Treatment (n)	Time (hr)	pmol 14C-2-propen-1-ol /mg protein		
		Liver	Lung	Kidney
Control (5)	8	119.5	16.1	10.7
Pyrazole (2)	8	21.6*	11.2	4.2
Control (2)	24	80.2	10.5	3.6
Pyrazole (2)	24	12.9*	4.9	2.4

* P<0.05, test not stated

Test substance: Name: 14C-2-propen-1-ol, specific activity 1-46 mCi/mmol
Conclusion: Results from these studies indicate that both binding of 14C-2-propen-1-ol to liver macromolecules and subsequent periportal hepatic necrosis are mediated by a metabolite of 2-propen-1-ol. This conclusion is compatible with the hypothesis that the toxic metabolite is acrolein.
Reliability: (2) valid with restrictions
Well reported published study
03-JAN-2006 (125)

In Vitro/in vivo: In vivo
Type: Metabolism
Species: rat

Year: 1983
GLP: no data
Test substance: other TS: refer to freetext

Method: ANIMALS AND TREATMENTS
Male SD rats (200-220g; n=4) were treated (0.05 ml, i.p.) with either 2-propen-1-ol (AA) or deuterated 2-propen-1-ol (d2-AA). After 24 hr, surviving animals were killed by exsanguination (blood collected in heparinised beakers) and the liver excised.

Comment: Assuming a density of 0.85, 0.05 ml is equivalent to approx. 42.5 mg/kg bw.

ASSESSMENT OF LIVER DAMAGE

Samples of liver were fixed (buffered formalin), paraffin sections prepared and stained with hemotoxylin and eosin.

Cellular damage was assessed by the method of Mitchell et al. (1973) J Pharmacol Exp Ther, 187, 185. Glutamyl-pyruvate transferase levels (GPT) in plasma were determined by a external laboratory (Pathologists Central Laboratory, Seattle).

COVALENT BINDING
The extent of covalent binding was determined in rats given 0.05 ml (i.p.) 14C-2-propen-1-ol or d2-14C-2-propen-1-ol as described by Reid (1972): see preceding record.

IN VITRO METABOLISM
Metabolism of 2-propen-1-ol and d2-2-propen-1-ol by hepatic 9000 g supernatant (+/- pyrazole; alcohol dehydrogenase inhibitor), 104,000 g cytosol (+/- disulruram; inhibitor of aldehyde dehydrogenase) and microsomal fraction (+/- NADPH; epoxidation of 2-propen-1-ol to glycidol) was followed using the semicarbizide reaction (formation chromophore absorbing at 257 nm). Standards containing known amounts of acrolein were run in parallel.

Result: Hepatic necrosis (score: 1.75) and plasma GTP levels (2540 mU/ml) were 7-8 fold greater at 24 hr post-treatment in rats given 2-propen-1-ol compared to rats given an equivalent dose of d2-2-propen-1-ol (necrosis score: 0.25; GPT 341 mU/ml).

Covalent binding 8 hr post-treatment with 14C-2-propen-1-ol was 155 pmol/mg protein compared with 48 pmol/mg protein in animals given d2-14C-2-propen-1-ol (3 fold difference).

NADH-dependent formation of acrolein and acrylic acid was 51-96% greater when 2-propen-1-ol was used as substrate compared to when d2-2-propen-1-ol was present:

	Acrolein nmol/min/mg protein			
	AA	d2-AA	AA	d2-AA
None	0.0	0.0	0.0	0.0
NADH	48.0	31.2***	46.2	30.5***
NADH/pyrazole	16.6	11.1***	14.8	10.2**

	Acrylic acid nmol/min/mg protein			
	AA	d2-AA	AA	d2-AA
None	0.0	0.0	0.0	0.0
NAD+	5.0	3.1***	5.5	2.8***
NAD+/disulfiram	1.8	0.0***	1.7	0.0***

** P<0.01; *** P<0.001 (test not stated)

There was no difference in the rate of appearance or disappearance of glycidol during microsomal incubation with 2-propen-1-ol or d2-2-propen-1-ol.

Test substance: Deuterated (d2) 2-propen-1-ol was prepared by custom synthesis and purified by preparative gas chromatography

(>99.5% pure).

14C-2-propen-1-ol was purchased from ICN Chemical and Radioisotope Division (Irvine, CA) with a specific activity of 10.8 mCi/mmol.

Deuterated 14C-2-propen-1-ol (0.75 mCi/mmol) was prepared by micro custom synthesis from 1-14C-acrylic acid.

Conclusion: Rats given a single i.p. treatment of 2-propen-1-ol exhibited greater hepatic necrosis, elevated levels of plasma GPT and greater covalent binding to liver protein than rats given an equivalent dose of deuterated 2-propen-1-ol. These differences correlated with significantly greater formation of acrolein and acrylic acid by liver fractions in vitro when 2-propen-1-ol was substrate compared to that seen with deuterated 2-propen-1-ol. These NADH-dependent reactions were sensitive to inhibition by pyrazole and disulfiram, indicating a role for alcohol- and aldehyde dehydrogenases in the hepatic metabolism of 2-propen-1-ol.

Reliability: (2) valid with restrictions
Well reported published study

06-JAN-2006 (113)

In Vitro/in vivo: In vivo
Species: mouse

Method: other: Mechanistic Study
Year: 1987
GLP: no data
Test substance: other TS: refer to freetext

Method: Animals: Male albino mice (average weight 15 g)

Animals were fed for at least 4 weeks with a low vitamin E diet (Altromin, Lage, FRG; 0.55 mg vit. E/kg). The pellet diet was withdrawn 48 hr prior to 2-propen-1-ol administration to decrease the liver glutathione content and to level off its diurnal variation. Animals received a sucrose solution (10% w/w) during this period.

All experiments were started at 5 pm by intraperitoneal application of 2-propen-1-ol (40 - 100 uL/kg, corresponding to 0.60-1.5 mmol/kg). Various pretreatments of animals were performed as follows: glutathione (0.53 mmol/kg) was injected intravenously two hours prior to 2-propen-1-ol; phorone (300 mg/kg) was injected i.p. and the animals were killed two hours later; diethylmaleate was injected i.p. in doses of 400 mg/kg 40 min, 20 min and at the start of the ethane measurement, respectively; the mice were killed one hour later. The monooxygenase inhibitors metyrapone (150 mg/kg), diethyldithiocarbamate (100 mg/kg) and alpha-naphthoflavone (100 mg/kg) were injected i.p. 30 minutes prior to 2-propen-1-ol; benz(a)pyrene was injected i.p. in doses of 20 mg/kg on day 3, 2 and 1 prior to 2-propen-1-ol; pyrazole (375 mg/kg) or cyanamide (50 mg/kg) were injected i.p. one hour prior to 2-propen-1-ol.

Desferoxaminemethanesulfonate was injected in two daily doses of 0.6 mmol/kg (s.c.) for three days. The animals received only sucrose solution in distilled water during the pretreatment. The last dose of Desferal was administered two hours prior to ally alcohol. Phenobarbital induction was performed by feeding as a solution in water (1 mg/mL) for five days. Phenobarbital was withdrawn 24 hours prior to 2-propen-1-ol administration.

Parameters measured:
Hydrocarbon exhalation of single animals measured over a period of one or four hours or until death

Animals were killed after one or four hours and blood taken by heart puncture and the liver was part homogenised in metaphosphoric acid and total soluble glutathione determined. TBA-reactive material was measured and protein determined. Another part of the liver was homogenised in 100 mM potassium phosphate buffer and hepatic P-450 content determined in the homogenate. Alcohol dehydrogenase and aldehyde dehydrogenase activities in the postmitochondrial supernatant was measured, as were serum transaminase activities in heparinised serum.

In some experiments, liver lipid fatty acids were identified.

In vitro studies were performed using soy bean lecithin liposomes and mouse liver microsomes. The lipsomes (0.75 mg phospholipid/mL) or microsomes (1 mg protein/mL) were incubated in potassium phospahte buffer (100 mM; pH 7.4) and in the presence of various concentrations of acrolein (0, 0.3, 1.0, 3.0 or 10.0 mM) at 25°C and normoxic conditions for one hour in gas tight syringes. After one hour, 7 mL of headspace gas were withdrawn isobarically and analyzed by gas chromatography. The concentration of free acrolein in the suspension was determined.

Result: After administration of 2-propen-1-ol (1.1 mmol/kg) hepatic glutathione (24.3+/-7.0 nmol GSH/mg protein) was almost totally lost within the first 15 minutes (<0.5 nmol GSH/mg protein). Subsequently a very large lipid peroxidation was observed, i.e. the animals exhaled 414+/-186 nmol ethane/kg/hr compared to 0.9+/-0.8 of controls, and the hepatic TBA-reactive compounds had increased from 55+/-16 pmol/mg protein in controls to 317+/-163 after one hour. Concomitantly, a 40-45% loss of the polyunsaturated fatty acids (arachidonic and docosahexaenoic acid) in the liver lipids was observed. About 80% of the cytosolic alcohol dehydrogenase activity and about 50% of the microsomal P450 content were destroyed.

In vivo inhibition of alcohol dehydrogenase by pyrazole or induction of aldehyde dehydrogenase by phenobarbital abolished 2-propen-1-ol-induced liver damage as well as glutathione depletion and lipid peroxidation, while inhibition of aldehyde dehydrogenase by cyanamide made a subtoxic dose of 2-propen-1-ol (0.6 mmol/kg) highly toxic. These results strongly favour the importance of acrylic acid formation as an additional detoxification pathway. Enhanced hepatic levels of glutathione content by phorone or diethylmaleate alone caused marginally enhanced lipid peroxidation (phorone) but no liver cell damage. Monooxygenase inhibitors (metyrapone,

diethyldithiocarbamate or alpha-naphthoflavone) or an inducer (benz(a)pyrene) did not affect 2-propen-1-ol-induced toxicity. The ferric iron chelator desferoxaminemethanesulfonate prevented 2-propen-1-ol-induced lipid peroxidation and liver cell damage in vivo.

In vitro, acrolein alone failed to initiate lipid peroxidation in soy bean phospholipid liposomes or in mouse liver microsomes. Thus, acrolein not only impairs the glutathione defense system but also directly destroys cellular proteins and evokes lipid peroxidation by an indirect iron-dependant mechanism.

Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Supplier: Fluka AG (Buchs, Switzerland)

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (62)

In Vitro/in vivo: In vitro
Type: Metabolism
Species: rat

Year: 1980
GLP: no
Test substance: other TS: refer to freetext

Method: Preparations: Male Holtzman rats (120-130 g) were used after pretreament for 3 days with sodium phenobarbital (75 mg/kg/day). Tissue preparations and reconstitution of lyophilized liver 9000g supernatant fraction was done as previously described (Leibman 1971). Liver cytosol and microsomes were isolated from reconstituted lyophilized 9000g supernatant fraction by centrifugation at 105,000g for 45 minutes in a Beckman model L ultracentrifuge with a type 40 rotor. Lung 9000g supernatant preparation was also lyophilized similarly to that of liver. Isolation of cytosol and microsomes from reconstituted lyophilized lung 9000g supernatant fraction was carried out as described by Hook et al.

Assays:

Metabolism of 2-propen-1-ol to acrolein was studied in a reaction mixture containing 1.0 mM NAD+, 5.0 mM $MgSO_4$, 5.0 mM 2-propen-1-ol and reconstituted lyophilized liver or lung 9000g supernatant fraction equivalent to 45 mg of protein or to 25 or 15 mg of cytosolic or microsomal protein (separated from lyophilized 9000g supernatant fraction), respectively, in 3.0 mL of 0.1 M Tris-HCl buffer (pH 7.5). For alcohol dehydrogenase inhibition, 0.5 mM pyrazole was used. After incubation at 37°C for 45 min, each reaction flask was placed in an ice bath and 1.0 mL of 70% $HClO_4$ was added. After centrifugation at 1400 rpm for 10 minutes the supernatant fluid was mixed with 5.0 mL of a solution containing 0.1% 2,4-dinitrophenylhydrazine (DNPH) in 0.2N HCl and kept overnight. The acrolein-DNPH derivative formed was extracted into 10.0 mL of HPLC-grade chloroform. The extract was washed with two 10.0 mL portions of 2N HCl to remove excess DNPH then washed with three 10.0 mL portions of distilled water and

finally concentrated to dryness under nitrogen. The dried samples were dissolved in 1.0 mL of methylene chloride and analyzed by HPLC. The recovery of acrolein-DNPH was >90%.

The conversion of 2-propen-1-ol to acrylic acid was studied in a reaction mixture containing 1.0 mM NAD+, 5.0 mM MgSO4, 5.0 mM 2-propen-1-ol and reconstitiuted lyophilized lung or liver 9000g supernatant fraction equivalent to 45 mg of protein or cytosol equivalent to 25 mg of protein.

Similarly, the conversion of acrolein to acrylic acid was studied with 1.0 mM NAD+ or NADP+, 5.0 mM Mg2+, 5.0 mM acrolein and lung or liver reconstituted lyophilized 9000g supernatant fraction equivalent to 45 mg of protein or 25 or 15 mg of cytosolic or microsomal protein, respectively, in 3.0 mL of 0.1 M Tris-HCl (pH 7.5). Disulfiram (0.5 mM) was used in aldehyde dehydrogenase inhibition studies; a stable turbid solution could be prepared by warming and shaking. After incubation at 37°C for 45 minutes the reaction was terminated by adding 1.5 mL of 6N HCl. After centrifugations, acrylic acid in the supernatant was extracted into ether and derivatised with PNBDI.

The formation of glycidol and glycidaldehyde, microsomal oxidation products of 2-propen-1-ol and acrolein, respectively, was demonstrated by incubation of 3.0 mM 2-propen-1-ol or acrolein with 15 or 10 mg of lung or liver microsomal protein, respectively, in a 3.0 mL incubation mixture described previously (Leibman and Ortiz, 1968).

The hydration of glycidol and glycidaldehyde by liver microsomal epoxide hydrase was investigated by incubation of 2-propen-1-ol and acrolein as well as epoxides with microsomes.

The formation of glycerol from 2-propen-1-ol or glycidol by liver microsomes was also investigated.

Result: Metabolic conversion of 2-propen-1-ol to acrolein and then to acrylic acid has been demonstrated in liver 9000g supernatant fraction and in cytosol in the presence of NAD+. The conversion of 2-propen-1-ol to acrolein was not observed when NAD+ was replaced with NADP+ or NADPH or in liver microsomes with NAD+. Similarly, 2-propen-1-ol was not metabolized to acrolein when lung fractions were used. Liver 9000g supernatant and cytosolic fractions metabolized 80% of added 2-propen-1-ol to acrolein. Pyrazole treatment significantly inhibited (70%) the formation of acrolein in both 9000g and cytosolic fractions. Acrolein formed in 2-propen-1-ol metabolism was further metabolised to acrylic acid in liver 9000g and cytosolic fractions; only 15% of the acrolein was metabolised to acrylic acid. In the presence of pyrazole, acrylic acid was not detectable even though some acrolein was formed from 2-propen-1-ol. Addition of 0.5mM disulfiram did not significantly inhibit formation of acrolein. However, metabolism of acrolein to acrylic acid was inhibited by 62%. Total inhibition of acrylic acid formation was observed when 1.0 mM disulfiram was present.
In vitro metabolism of acrolein to acrylic acid has been shown in liver 9000g supernatant, cytosolic and microsomal preparations. In all of these fractions acrylic acid was

detected when acrolein was incubated either in the presence of NAD+ or NADP+. Acrylic acid was not formed when lung fractions were used or when NAD+ and NADP+ were replaced with NADPH in the liver incubation mixtures. About 20 annd 7% of added acrolein was metabolized to acrylic acid in these NAD+ and NADP+ dependent aldehyde dehydrogenase reactions, respectively, in liver 9000g supernatant fraction. Disulfiram inhibited the NAD+ and NADP+ dependent reactions by 60 and 80% in 9000g supernatant and 71 and 100% in cytosolic fractions, respectively. In microsomes, both reactions were totally inhibited. Complete inhibition of aldehyde dehydrogenase in 9000g supernatant fraction and cytosol was achieved at 1.0 mM disulfiram concentration.
After incubation of 2-propen-1-ol and acrolein with liver and lung microsomes in the presence of NADPH, glycidol and glycidaldehyde, respectively, were formed. Glycidaldehyde recovered in lung and liver microsomal incubations reached a maximum level at 6 minutes and after that gradually declined and was no longer detectable after 15-16 minutes. The rate of glycidol formation was slightly slower than that of glycidaldehyde; it was maximal at 11-12 minutes in both lung and liver and was not detectable after 16-18 minutes.
The hydration of the epoxides, glycidol and glycidaldehyde, either added directly or formed in situ from 2-propen-1-ol and acrolein, respectively, by liver and lung microsomal epoxide hydrase was studied by estimation of glycerol and glyceraldehyde. When boiled microsomes were used epoxides were not converted to either glycerol or glyceraldehyde. Enzymatic conversion of glycidol to glycerol was lower than that of glycidaldehyde to glyceraldehyde by both lung and liver microsomes.
Glycidol and glycidaldehyde were also found to be substrates for lung and liver cytosolic GSH-S-transferases. The rate of GSH conjugation with glycidaldehyde was twice that of glycidol conjugation in both lung and liver cytosol. Also, the rate of glycidol and glycidaldehyde conjugation in liver was double that in lung cytosol. When boiled lung or liver cytosol was used, no conjugation reaction was observed.

Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Reliability: (2) valid with restrictions
Well reported published study
30-NOV-2005 (114)

In Vitro/in vivo: In vivo
Species: rat

Method: other: Mechanistic Study
Year: 1978
GLP: no
Test substance: other TS: refer to freetext

Method: Rats were dosed orally with 1 mL/100g bw of a 1% solution of 2-propen-1-ol. Livers were removed and analysed for malonic aldehyde and conjugated dienes at 0, 1, 3, 24 and 72 hours after administration.
Result: 0, 1, 3, 24, and 72 hr after oral administration livers contained 1.05, 1.088, 1.37, 1.21, and 1.51 (arbitrary) units of malonic aldehyde and 4.30, 5.68, 10.97, 6.67, and 7.13

(arbitrary) units of conjugated dienes.
Alanine aminotransferase activity was very high during this period. Necrosis in the hepatocyte and Kupffer cell cytoplasm was observed from the 4th hr after administration which shows that the onset of lipid peroxidation precedes necrosis.

Test substance: name: 2-propen-1-ol (CAS No. 107-18-6)
Reliability: (2) valid with restrictions
Well reported published study
30-NOV-2005 (78)

In Vitro/in vivo: In vivo
Species: rat

Method: other: Mechanistic Study
Year: 1986
GLP: no data
Test substance: other TS: refer to Freetext

Method: Animals: Female Sprague Dawley rats, 250 - 350 g

Liver perfusion: Rats were treated with sodium phenobarbital (1 mg/mL) in drinking water for at least one week before perfusion experiments to facilitate identification of periportal and pericentral regions of the liver lobule. Where indicated, animals were treated with diethylmaleate (0.7g/kg i.p.) one hour before perfusion. Livers were perfused with Krebs-Henseleit bicarbonate buffer (pH 7.4, 37°C) saturated with an oxygen-carbon dioxide mixture (95:5) in a non-recirculating system. The fluid was pumped into the liver via a cannula placed in the portal vein and flowed past a Teflon-shielded oxygen electrode before being discarded. 2-Propen-1-ol was measured enzymatically in samples of effluent perfusate. Rates were calculated from the influent minus the effluent concentration differences, the flow rate and the liver wet weight. Bile flow was monitored during perfusion. Livers were perfused for one hour with either 2-propen-1-ol (350 uM), acrolein (200 uM), acrylic acid (350 uM) or potassium cyanide (2mM). 4-methylpyrazole (80 uM) was infused during the addition of acrolein to prevent the metabolism of acrolein to 2-propen-1-ol by alcohol dehydrogenase. All chemicals were diluted with Krebs-Henseleit bicarbonate buffer.

Analysis: The left lateral lobule of the liver was frozen rapidly in liquid nitrogen during perfusion. Blocks of tissue were dissected in the frozen state and then sliced. The frozen sections were lyophilised at -40°C to maintain histological structure and facilitate visualisation of periportal and pericentral regions. Samples (0.1-0.3 ug) from both zones were microdissected and weighed. ADP was converted to ATP using creatine phosphate and creating phosphokinese and ATP was determined using a firefly luciferin-luciferase assay. Reduced glutathione and GSSG were determined in powdered extracts of frozen liver. Total glutathione content in periportal and pericentral regions was determined in microdissected samples by enzymatic cycling. Samples of effluent perfusate and bile were collected every four minutes for the enzymatic determination of GSSG in the bile and lactate dehydrogenase in the effluent perfusate. Rates of efflux of GSSG were

determined from the flow rate of bile and the liver wet weight.

Histology: Trypan blue (0.2mM, Sigma) was infused into livers for 10 minutes after exposure to 2-propen-1-ol, acrolein or acrylic acid. Livers were then perfused with 1% paraformaldehyde for 6 minutes and fixed tissue was embedded in parafin and processed for light microscopy. Sections were stained only with eosin, a cytoplasmic stain, so that trypan blue could be identified readily in the nuclei of damaged cells.

Statistics: Statistical significance was assessed using Student's t test.

Result: It has previously been shown that treatment with phenobarbital does not affect rates of 2-propen-1-ol uptake by the liver or 2-propen-1-ol toxicity. Moreover, treatment with phenobarbital does not affect the the time course of 2-propen-1-ol induced damage as indicated by the inhibition of 2-propen-1-ol metabolism and oxygen uptake, release of lactate dehydrogenase and uptake of trypan blue. These data indicate that studies with normal and phenobarbital-treated rats are equivalent.

Infusion of 2-propen-1-ol (350 uM) for 20 min depleted hepatic glutathione content by 95% in both regions of the liver lobule yet damage was undetectable as indexed by release of lactate dehydrogenase or uptake of trypan blue. Perfusion for an additional 40 min in the absence of 2-propen-1-ol resulted in lactate dehydrogenase release (2400 U/L) and uptake of trypan blue by 75% of hepatocytes in periportal regions of the liver lobule; however dye was not taken up by cells in pericentral areas. Because the content was depleted in the undamaged pericentral area, it was concluded that thiol depletion alone cannot explain local toxicity to periportal regions by 2-propen-1-ol.

Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Supplier: Aldrich Chemical Co. Inc (Milwaukee, WI)

Reliability: (2) valid with restrictions
Well reported published study

30-NOV-2005 (20)

In Vitro/in vivo:	In vivo
Species:	rat
No. of animals, males:	30
No. of animals, females:	30
Doses, males:	0, 50 mg/kg bw
Doses, females:	0, 50 mg/kg bw
Vehicle:	other: sunflower oil

Method: other: Mechanistic Study
Year: 1979
GLP: no
Test substance: other TS: refer to Freetext

Method: Animals: CFY rats (male and female, 200-300 g bw)

Dosing: Gavage

Animals per group: 5 treated animals, 3 sunflower oil-treated

and 2 untreated

Groups were killed by bleeding in ether narcosis 6, 12, 24, 48, 72 and 96 hours after dosing.

At autopsy the rat organs were studied for macroscopic changes. Liver tissue was removed for histologic and histochemical examination and the entire pancreas for histologic, histochemical and ultrastructural examination.

Result: Controls: In the rats given a single application of sunflower oil, neither macroscopic nor significant microscopic changes could be found in liver and pancrease compared to untreated controls. Histochemically, a very intensive activity of alcohol dehydrogenase was found in the periphery of the liver lobules around the periportal fields. A low activity of this enzyme was focally demonstrable in the cytoplasm of pancreatic acinar cells.

2-Propen-1-ol-treated animals:

Hepatic findings: The livers of almost all the rats treated with 2-propen-1-ol showed large necroses 12 h and mainly 24 h and later after administration of the chemical. Histologically, the necroses proved as periportal, partially confluent liver cell necrosis. Histochemically, the alcohol dehydrogenase activity in the non-necrotic liver tissue was nearly of the same degree as in the control animal livers.

Pancreatic findings: Generally, the histologic and ultrastructural findings in the exocrine pancreas after a sinle 2-propen-1-ol intake varied from animal to animal in the degree of damage as well as in the quantity of the pancreatic involvement. As a rule, the findings were distributed focally rather than diffusely in the rat pancreas and they did not show a close correlation with the degree of concomitant liver injury in every case.

Histologically, an increasing acidophilia of the pancreatic acinar cells in connection with a diminished basophilic ergastoplasm of the basal parts of the cells was observed 6-24 h after dosing. Focal acinar cell necrosis could be detected 24 and 48 h after the peroral application. Patchy vacuolization of acinar cell groups could be observed in an increasing degree from 24-96 h after dosing. Histochemically, a low alcohol dehydrogenase activity could focally be demonstrated in the cytoplasm of pancreatic acinar cells. The activity was nearly of the same degree as in the pancreas of control animals. The pattern of alcohol dehydrogenase activity in the cryostat section of the pancreas of both the 2-propen-1-ol treated and control animals resembled the ditribution of the acinar cell changes found histologically in the paraffin section of the pancreas after 2-propen-1-ol administration. By transmission electron microscopy, changes of the exocrine pancreas could already be detected 6 h after dosing. Lipid droplets, mostly aggregated in groups, were found in the basal parts of the acinar cells in a high number between 6 and 24 h after dosing. Mitochondrial degenerative changes occurred in the acinar cells in a marked degree 12 and 24 h after dosing. They consisted of mitochondrial swelling and degradation with formation of fingerprint-like pictures.

Focal cytoplasmic degeneration, mostly found in the acinar cells, but also in centro-acinar and ductular cells at all the intervals under study, appeared in a great number between 12 and 48 h after dosing. Single acinar cell necrosis occurred 12 h after dosing, most often after 24 and 48 h but in some cases it could also be seen still after 72 and 96 h. Cytoplasmic vacuolation was focally observed not only in the acinar cells but also in the centroacinar and ductular epithelium cells in an increasing degree from 24 up to 96 h after dosing. The vacuoles were membrane-lined and empty or they contained cytoplasmic debris as seen in autolysomes.

Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Purity: Analytical grade
Supplier: Chinoin, Budapest

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (107)

In Vitro/in vivo: In vitro
Species: rat
Doses, males: 0.1 - 0.8 mM

Method: other: Mechanistic Study
Year: 1986
Test substance: other TS: refer to Freetext

Method: Animals: Male Fischer 344 rats
Source: National Institute on Aging by Harlan Sprague-Dawley, Inc (Indianapolis, Indiana)

Hepatocyte isolation: Carried out using 0.05% collagenase in the 2-step perfusion procedure of Seglen (1976) except that cells were washed with Waymouth MB 752/1 medium. Hepatocytes isolated from rats of different ages did not differ significantly in average yield (30E10+06 cells/g liver) or viability (80-90% excluded typan blue).

Hepatocytes were suspended in Waymouth medium and initial viability estimated. Incubations were initiated immediately thereafter. 2-Propen-1-ol and pyrazole were dissolved in Waymouth medium and added to the incubation mixutes at 0 hr and samples of the cell suspension were removed at 0, 1, 2, 3 and 4 hr of incubation.

The cytotoxicity of 2-propen-1-ol was assessed by measuring lactate dehydrogenase (LDH) leakage and trypsan blue uptake by hepatocytes.

Glutathione (GSH) was determined as nonprotein sulfhydryl groups .

Statistical analysis: Significant differences were determined by analysis of variance (ANOVA) and calculation of the F ratio. Differences with $p<0.05$ were considered significant.

Result: Incubation with 0.1-0.8 mM 2-propen-1-ol resulted in a dose-dependent and time-dependent loss of viability. Dose effect and time effect curves were displaced to the left with hepatocytes isolated from old rats compared with those from

young rats. Intermediate values were found for hepatocytes of middle aged rats. Inhibition by pyrazole of 2-propen-1-ol-induced LDH release from hepatocytes was also affected by age. Total protection was observed for cells from young rats, whereas no protection was found for those of old rats. Cells from middle aged rats were between the extremes.

Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Supplier: Eastman Kodak Company

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (129)

In Vitro/in vivo: In vivo
Species: rat
No. of animals, males: 40
Doses, males: 0 or 0.62 mmol/kg

Method: other: Mechanistic Study
Year: 2000
Test substance: other TS: refer to Freetext

Method: Animals: Male Sprague-Dawley rats
Age/weight: Adults, 200-250 g

30 rats were injected with 0.62 mmol/kg of 2-propen-1-ol intraperitoneally twice a week, the remaining 10 with normal saline as controls. Ten rats were killed at each of 4, 8, and 16 weeks later. The rats were killed 72 h after the last injection. Under ether anesthesia the abdomen was opened and the whole liver excised. Part of the liver was used for histologic examination and the remainder was frozen for RNA extraction. Sera was separated from whole blood obtained by cardiac puncture and kept at -20°C for enzyme assay.

The activities of serum ALAT were measured.
The extent of liver fibrosis was evaluated according to the portal-portal extent and existence of regenerating nodules. Also analysed the number, length and thickness of fibrous septa.

Collagen content was measured using a dye-binding procedure.

TGFbeta1 mRNA in liver tissues was determined.

Statistical analysis: The Mann-Whitney U test, Wilcoxon test, Mantel-Haenzel chi-squared test and Spearman rank correlation coefficient were used to analyse the results. Results were considered significant when $p<0.05$.

Result: All rats except one in the 8-week group survived until the end of the experiment. The dead rat showed only a mild degree of hepatic necrosis and no abnormal features in the other organs, and cause of death could not be determined. Bodyweight in the 8-week group was not significantly different from that of controls sacrificed at the same time.

After 4 weeks, periportal fibrosis was produced in only 6 out of 10 rats, and was mild in extent. After 8 weeks, 8 out of 9

survivors showed moderate to severe fibrosis, which corresponded to a score of 7 or more. The extent of fibrosis correlated significantly with the amount of collagen and TGFbeta1 mRNA expression in liver tissues. The collagen content and expression of TGFbeta1 mRNA were also upregulated significantly in liver tissues with a fibrosis score of 7 or more.

Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)

Conclusion: Hepatic fibrosis can be sufficiently induced by repetitive injection of 0.62 mmol/kg 2-propen-1-ol twice a week for 8 weeks.

Reliability: (2) valid with restrictions
Well reported published study

30-NOV-2005 (67)

In Vitro/in vivo: In vitro

Species: rat

Method: other: Mechanistic Study

Year: 1984

Test substance: other TS: refer to Freetext

Method: Female Sprague-Dawley rats (200-300 g) were treated with sodium phenobarbital (1 mg/mL) in drinking water for at least one week prior to perfusion experiments to facilitate identification of periportal and pericentral regions.

Liver perfusion: Livers were perfused with Krebs-Henseleit bicarbonate buffer in a non-recirculating system. Rates of oxygen uptake were calculated from the influent minus the effluent oxygen concentration difference, the flow rate and the liver wet weight. 2-Propen-1-ol, acrolein and 4-methylpyrazole were diluted with buffer and infused into the liver. Samples of effluent perfusate were collected every two minutes for enzymatic determination of 2-propen-1-ol with alcohol dehydrogenase.

Determination of 2-propen-1-ol concentration in blood: Rats received 2-propen-1-ol (42 mg/kg) by i.p. injection of a 300 mM solution in normal saline. After 30 minutes, rats were anesthetised and 1.5 mL of blood withdrawn from the portal vein and vena cava. After centrifugation, 2-propen-1-ol was determined in the supernatant by a standard enzymatic procedure using alcohol dehydrogenase.

Micro-light detection of NADH fluorescence: Anterograde (via the portal vein) and retrograde (via the vena cava) perfusions of the liver with India ink identified lightly pigmented areas as periportal areas and darkly pigmented spots as pericentral regions. Micro-lightguides were then positioned on periportal and pericentral regions. The liver was illuminated with a 366 nm mercury arc line and the NADH fluorescence (450 nm) of the tissue was detected with a photomultiplier.

Rates of oxygen uptake in periportal and pericentral regions of the liver lobule: Measured using a miniature oxygen electrode which could be placed into periportal and pericentral regions . Rates of oxygen uptake were determined by measuring the rate of decrease of oxygen concentration.

Result: A linear increase in NADH fluorescence was observed when 25-150 µM 2-propen-1-ol was infused; however, when 2-propen-1-ol exceeded 200 µM, oxygen uptake by the liver was inhibited 30-40% and a large increase in NADH fluorescence occurred. 2-Propen-1-ol (350 µM) or acrolein (200 µM) inhibited oxygen uptake only in the periportal regions. The maximal increase in NADH fluorescence due to 2-propen-1-ol infusion (100 µM) was greater in pericentral than in periportal regions. 4-Methylpyrazole (80 µM), an inhibitor of alcohol dehydrogenase, prevented the fluorescence increase due to 2-propen-1-ol in both regions, indicating that the changes were due entirely to NADH generated from alcohol dehydrogenase-dependent 2-propen-1-ol metabolism. Using the correlation (r=0.91) between rates of 2-propen-1-ol uptake and the increase in NADH fluorescence established for the whole organ, local rates of 2-propen-1-ol metabolism were 23 and 31 µmoles/g/hr in periportal and pericentral regions, respectively. These results indicate that metabolism of 2-propen-1-ol occurs at slightly greater rates in pericentral than in periportal regions of the liver lobule. Thirty minutes after i.p. injection of a necrogenic dose of 2-propen-1-ol in vivo, the concentrations of 2-propen-1-ol in the portal vein and vena cava were 1210 and 530 µM, respectively. Thus, both periportal and pericentral regions of the liver lobule were exposed to concentrations of 2-propen-1-ol (e.g. >200 µM) which were metabolized in the perfused liver. Since 2-propen-1-ol is metabolised in both regions of the liver lobule, the hypothesis that the zone-specific hepatotoxicity results from its exclusive metabolism to acrolein in periportal regions seems unlikely.
Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Supplier:Aldrich Chemical Co.
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (21)

In Vitro/in vivo: In vitro
Species: rat
Doses, males: 0.35 or 2 mM

Method: other: Mechanistic Study
Year: 1991
Test substance: other TS: refer to Freetext

Method: Rat livers perfused with 0.35 or 2 mM 2-propen-1-ol. Xanthine dehydrogenase in lyophilised tissue determined. Xanthine oxidase and xanthine dehydrogenase in whole liver assayed.
Result: Perfusion of the liver with 2-propen-1-ol (350 mM) increased xanthine oxidase and decreased xanthine dehydrogenase in the whole liver consistent with the phypthesis that 2-propen-1-ol enhanced calcium-dependent proteolytic conversion of the NAD+-dependent to the O2-dependent form. Xanthine dehydrogenase was higher in pericentral than in periportal regions of the liver lobile and tended to decrease selectively in periportal zones of livers exposed to 2-propen-1-ol. O2 uptake was stimulated transiently by 2-propen-1-ol (2mM) followed by subsequent inhibition of respiration. These results are consistent with the idea that conversion of NAD+-dependent xanthine dehydrogenase to xanthine oxidase is

involved in the zone specific hepatotoxicity of 2-propen-1-ol.
Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Reliability: (2) valid with restrictions
Well reported published study
30-NOV-2005 (26)

In Vitro/in vivo: In vitro
Species: rat

Method: other: Mechanistic Study
Year: 1986
Test substance: other TS: refer to Freetext

Method: Animals: male Wistar rats (350-450 g)
Source: Winkelmann, Borchen.

Livers removed from anesthetised rats and perfused using a recirculating system.

2-Propen-1-ol was added directly to the perfusion medium to yield the appropriate final concentration after a 30 min equilibration period. Its availability was assayed by measurement of its concentration in the perfusate at various time points. Inhibition of alcohol dehydrogenase was accomplished by addition of 4-methylpyrazole (0.5 mmol/L) to the perfusate 30 min prior to application of 2-propen-1-ol. Three different levels of Ca2+ ions were used in the perfusate.
Result: 4-methylpyrazole inhibited the disappearance of 2-propen-1-ol from the perfusate as well as its hepatotoxic activity, demonstrating that the effect of 2-propen-1-ol was preserved in the in vitro preparations to the same extent as under in vivo conditions.

High extracellular Ca2+ (5 mmol/L) led to a threefold increase of liver calcium but produced only marginal hepatotoxicity and only slightly enhanced the hepatotoxic effects of 2-propen-1-ol (1.17 mmol/L).
Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Conclusion: Primary 2-propen-1-ol-induced hepatic injury does not appear to depend on an influx of extracellular calcium
Reliability: (2) valid with restrictions
Well reported published study
30-NOV-2005 (153)

In Vitro/in vivo: In vivo
Species: rat
Doses, males: 0 (vehicle control), 10, 25 or 50 mg/kg
Vehicle: physiol. saline

Method: other: Mechanistic Study
Year: 1994
Test substance: other TS: refer to Freetext

Method: Weanling male Sprague-Dawley rats were randomly assigned to receive either a normal pelleted diet or an obesity-producing energy dense formulation.

In the first experiment, animals were fed on one of the two diets for 72 weeks. Animals from both diet groups were then randomly assigned to receive 0, 10 or 25 mg/kg bw 2-propen-1-ol (administered i.p. in 0.9% saline solution). Six hours after administration the animals were killed and a sample of heparinised plasma taken for determination of alanine aminotransferase (ALT) activity.

In the second experiment, animals were fed one of the two diets for 48 weeks. Animals then received 0, 25 or 50 mg/kg 2-propen-1-ol (i.p. in saline solution). As before, animals were killed six hours after administration and plasma samples were obtained. In addition, the livers were removed for analysis.

Result: ALT activity in plasma was ten-fold elevated in obese animals than in non-obese animals given the 25 mg/kg dose. The minimum dose required to produce elevated ALT was 50% lower for obese animals. Obesity exacerbated periportal injury by 2-propen-1-ol after removal of potential dosing biases, suggesting that potentiating effects of obesity on toxicity occurred at the cellular level. Increased toxicity in obese animals was not attributable to a rise in alcohol dehydrogenase activity, because bioactivation of 2-propen-1-ol to acrolein was similar for hepatic cytosol from untreated obese and non-obese rats. However, hepatic glutathione concentrations in untreated obese animals were 25% below those of untreated non-obese animals.

Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Supplier: Sigma Chemical Co. (St. Louis, MO)

Reliability: (2) valid with restrictions
Well reported published study

30-NOV-2005 (135)

In Vitro/in vivo: In vivo
Species: mouse

Method: other: Mechanistic Study
Year: 1986
Test substance: other TS: refer to Freetext

Method: Male CD-1 mice were treated with carbon tetrachloride (1 mL/kg, i.p.) or 2-propen-1-ol (0.05 mL/kg, i.p.) or both 24 hours prior to sacrifice. The livers were removed, homogenised and mitochondrial preparations were fractionated. Electron micrographs were taken.

Result: Electron micrographs from the livers of mice treated with carbon tetrachloride showed lysed and fragmented mitochondria as well as a few condensed mitochondria. Electron micrographs from the livers of mice treated with 2-propen-1-ol or a combination of 2-propen-1-ol and carbon tetrachloride showed normal mitochondrial morphology.

Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)

Reliability: (4) not assignable
Insufficient detail in published study

30-NOV-2005 (134)

In Vitro/in vivo: In vitro
Doses, males: 0.5 mM

Vehicle: other: Krebs-Henseleit buffer

Method: other: Mechanistic Study
Year: 1994
Test substance: other TS: refer to Freetext

Method: Male Fischer 344 rats (200-250 g) were used. Isolated hepatocytes were prepared using the collagenase perfusion technique. 0.5 mM 2-propen-1-ol solution added to hepatocyte suspensions at t=0. Dithiothreitol (DTT) solution added at t=15 min up to t=2 hr. Samples were removed at various time intervals for measurement of GSH, glutathione disulfide, protein sulfhydryls, cell blebbing, and viability.
Result: 2-Propen-1-ol-induced protein sulfhydryl loss, bleb formation and cell death were prevented by dithiothreitol (DTT) when it was added to hepatocytes 30 minutes after 2-propen-1-ol. The protective effect of DTT was also demonstrated in cells that were washed after 30 minutes of exposure to 2-propen-1-ol, indicating that protection was not related to inhibition of 2-propen-1-ol metabolism or inactivation of acrolein.
Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
Insufficient detail in published study
05-DEC-2005 (128)

In Vitro/in vivo: In vitro
Species: rat

Method: other: Mechanistic Study
Year: 1996
Test substance: other TS: refer to freetext

Method: The involvement of altered pyridine nucleotide concentrations in the cytolethality of 2-propen-1-ol was studied in isolated rat hepatocytes. NAD+, NADH, NADP+, NADPH and viability loss (leakage of lactate dehydrogenase into the medium) were measured in cells incubated with 0.5 mM 2-propen-1-ol with or without the addition of 2 mM dithiothreitol at 30 minutes.
Result: Exposure to 2-propen-1-ol increased NADH levels in the first 15 minutes of incubation. A sharp drop in NADH and NADPH with an accumulation of NADP+ occurred between 30 and 60 minutes of incubation with 2-propen-1-ol, indicating an oxidation and interconversion of pyridine nucleotides. Dithiothreitol prevented the oxidation of pyridine nucleotides but not their reduction or interconversion and protected against cell killing by 2-propen-1-ol. The results suggest that pyridine nucleotide oxidation may be important for 2-propen-1-ol-induced cytotoxicity, although no causal relationship between pyridine nucleotide oxidation and cell killing has yet been demonstrated.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
Published study using non-standard methods
19-DEC-2005 (132)

In Vitro/in vivo: In vivo
Species: other: rat/mouse

Doses, males: 25 - 200 mg/kg

Method: other: Mechanistic Study
Year: 1986
Test substance: other TS: refer to Freetext

Method: Animals: Male Fischer-344 rats (150-200 g); male B6C3F1 mice (20-25 g).

Dose response toxicity studies: Rats and mice were dosed orally with 25-200 mg/kg of 2-propen-1-ol. Corn oil was used as a vehicle. The animals were anesthetized with ether 24 h after dosing and blood was taken from the inferior vena cava and analyzed for SGPT activity. Histopathlogical evaluation of liver was performed.

Result: Although statistically significant increases in SGPT activities were observed in both rats and mice at 50 and 75 mg/kg, the magnitude of these increases at 50 or 75 mg/kg was considerably greater in rats. Mice given 100 to 220 mg/kg of 2-propen-1-ol died within 24 h after administration. Therefore in mice it was not possible to identify a nonlethal dose of 2-propen-1-ol that resulted in extensive liver damage. Histopathological examination confirmed a marked species difference in 2-propen-1-ol-induced hepatic injury. Doses of 25, 50 and 75 mg/kg of 2-propen-1-ol were not hepatotoxic in mice. In rats, periportal necrosis was evident in 9 of 12 animals treated with 2-propen-1-ol. The severity of the injury was greater at the 50 and 75 mg/kg doses that at the 25 mg/kg dose.

Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)

Chemical name: [14]C-2-Propen-1-ol
Purity: >99%
Synthesised by authors from [14]C-diallyl phthalate (specific activity 12.2 mCi/mmol)

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (40)

In Vitro/in vivo: In vitro

Method: other: Mechanistic Study
Year: 1989
Test substance: other TS: refer to freetext

Method: Animals: male Sprague-Dawley rats (180-250 g)

Hepatocytes were isolated by collagenase perfusion of the liver. Cells were incubated at a concentration of 1E06 cells/mL in rotating round bottom flasks at 37°C in Krebs-Henseleit buffer, pH 7.4 supplemented with 12.5 mM Hepes under an atmosphere of 95% 02, 5% CO2. The final incubation volume was 20 mL with a cell concentration of 1E06/mL The hepatocytes were preincubated for 30 minutes before the addition of 2-propen-1-ol or acrolein. 2-Propen-1-ol and acrolein (100 mM stock solution in DMSO) were prepared

immediately prior to use.

Glutathione (total GSH and GSSG) content of hepatocytes was measured.

Aldehyde dehydrogenase (ALDH) activity of the S9 fraction was determined spectrophotometrically by measuring the production of NADH at 340 nm.

Malondialdehyde in hepatocytes was determined spectrophotometrically at 535 nm.

Result: The addition of either 2-propen-1-ol or acrolein to hepatocytes resulted in the appearance of membrane surface blebs followed by cell death in a time- and dose-dependent manner. Acrlein was more toxic than 2-propen-1-ol. Intracellular GSH was also depleted when hepatocytes were incubated with toxic concentrations of either 2-propen-1-ol or acrolein. Subtoxic doses partially removed the GSH. Depletion of GSH by acrolein was very rapid, while 2-propen-1-ol-induced depletion was slower. No GSSG formation occurred following incubation of either of these chemicals with hepatocytes.

Inclusion of cyanamid (an aldehyde dehydrogenase inhibitor) in the cell incubate prior to addition of acrolein increased the rate and extent of GSH depletion as well and cytotoxicity.

Pretreatment of the hepatocytes with 1 mM pyrazole (an alcohol dehydrogenase inhibitor) prior to 2-propen-1-ol treatment resulted in no GSH depletion or cytotoxicity. This result further indicates that acrolein formation is necessary for toxicity to occur. Pyrazole had no effect on acrolein treated cells.

Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Supplier: Aldrich (Milwaukee, WI)

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (146)

In Vitro/in vivo: In vitro

Method: other: Mechanistic Study
Year: 1987
Test substance: other TS: refer to Freetext

Method: The activity of alcohol dehydrogenase (ADH) with 2-propen-1-ol as substrate was measured in liver cystolic fractions of rats representing young adulthood, middle age and old age.

Result: 2-Propen-1-ol-induced hepatotoxicity was more severe in old male rats than in young male rats, as measured by release of hepatic enzymes from injured cells and loss of hepatic microsomal cytochrome P-450. The extent of toxicity in female rats was greater than in males and unaffected by aging.

ADH activities were 1.7+/-0.1, 2.3+/-0.1 and 2.6+/-0.1 µmol/min/g of liver in males rats aged 4, 14 and 25 months, respectively. ADH activities in young adult and old female rats were 3.8+/-0.1 and 3.7+/-0.1 µmol/min/g of liver. There was good correlation between liver ADH activity and 2-propen-1-ol-induced hepatotoxicity, measured as release of

sorbitol dehydrogenase into the bloodstream. Cytosolic free NAD+/NADH ratios in male rats were not significantly different among the three age groups; the ratios were lowest in young adult female rats. Low Km aldehyde dehydrogenase activities in liver mitochondrial and cytosolic fractions were similar among the three age groups of male rat, and the activities in female rats were not substantially different. The results indicated that increased ADH activity is the principal cause of the age associated enhancement of 2-propen-1-ol hepatotoxicity in male rats.

Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)
Purity: highest available
Supplier: Eastman Kodak Co. (Rochester, NY)

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (130)

In Vitro/in vivo: In vivo

Method: other: Mechanistic Study
Year: 1990
Test substance: other TS: refer to Freetext

Method: Male Swiss albino mice (25-30 g) were used. The animals were starved for 16 h before use to decrease the GSH stores of the liver.

In a first group of experiments, animals were dosed with 2-propen-1-ol (1.5 mmol/L, i.p.) dissolved in saline, or an equivalent volume of saline (as control). A number of the treated animals were treated with desferrioxamine (59 µmol/kg bw, dissolved in saline, i.p.) 10 and 40 min after 2-propen-1-ol treatment. All animals were killed 1-2 hours after dosing.GSH and MDA levels were measured on blood taken from the animals.

In a second group of experiments, animals treated as above were killed 15 minutes after dosing. Washed erythrocytes were incubated and treated with desferrioxamine (50 µM) and determination made of desferrioxamine-chelatable iron, MDA and hemolysis.

Result: 2-Propen-1-ol administration in a toxic dose (1.5 mmol/kg) to starved mice caused the development of hemolysis in nearly 50% of the animals. Malonic dialdehyde (MDA) appeared in the plasma of animals showing hemolysis. Treatment with desferrioxamine after dosing with 2-propen-1-ol completely prevented lipid peroxidation and hemolysis, suggesting the involvement of iron in the 2-propen-1-ol-induced erythrocyte damage. Erythrocytes obtained from intoxicated mice before the development of hemolysis show, upon incubation, release of iron, lipid peroxidation and lysis.

Test substance: Name:2-Propen-1-ol (CAS No. 107-18-6)
Purity: analytical grade

Reliability: (2) valid with restrictions
Well reported published study

30-NOV-2005 (42)

In Vitro/in vivo: In vitro

Method: other: Mechanistic Study
Year: 1988
Test substance: other TS: refer to Freetext
Method: Hepatocytes originating from predominantly the periportal or perivenous region of the acinus were isolated from male rats of the Alko mixed strain (7-9 weeks old, 220-280g).

Aliquots (475 uL) of cell suspension (10-13 mg/mL) were preincubate under carbogen in 20 mL chromatograph vials for 15 min at 37°C and 25µL of 8 mM 2-propen-1-ol added. At 1 or 5 min 100 uL of 2.4M PCA was injected through the rubber septum to stop the oxidation of 2-propen-1-ol. Concentration of 2-propen-1-ol was assayed using head-space GC.

16 uM of 2-propen-1-ol (700 uM) was added to preincubated cells and GSH determined using a fluorometric method.
Result: Periportal and perivenous cells isolated from rat liver oxidized 2-propen-1-ol at rates of 3.4 and 3.1 umol/(g.min), respectively. Cellular GSH was rapidly depleted (95%) by oxidation of 700 uM 2-propen-1-ol.
Test substance: Name: 2-propen-1-ol (CAS No. 107-18-6)
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (115)

In Vitro/in vivo: In vivo
Type: Metabolism
Species: rat
Doses, males: 64 mg/kg

Year: 1989
Test substance: other TS: refer to freetext

Method: Animals: Male Sprague-Dawley rats (220-250 g)

Rats (3/chemical/dose) were given allylamine.HCl (5, 25, 50, 100 or 150 mg/kg), acrolein (13 mg/kg), 2-propen-1-ol (64 mg/kg), allyl chloride (76 mg/kg) allyl bromide (120 mg/kg) allyl cyanide (115 mg/kg) or cyclophasphamide (160 mg/kg) by gavage in water. 3-Hydroxypropylmercapturic acid (3-OHPrMCA) was quantitatively measured by HPLC in 24-h urine collection.
Result: Various doses of allylamine resulted in 3-OHPrMCA excretion at a fairly constant percentage of the does, ca. 44-48% at 0-24 h and 3% at 24-48 h, indicating rapid metabolism through glutathione conjugation in the first 24 h. Similarly, 3-OHPrMCA was recovered in the urine of rats given acrolein (78.5%), 2-propen-1-ol (28.3%), allyl chloride (21.5%), allyl bromide (3.0%), allyl cyanide (3.7% and cyclophosphamide (2.6%).
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Purity: 99%
Conclusion: This study supports the concept that allylic compounds are metabolised to acrolein.
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (136)

In Vitro/in vivo: In vivo
Species: rat

Method: other: Mechanistic Study
Year: 1992
Test substance: other TS: refer to Freetext

Method: To assess whether potential toxic interactions occur between ethanol and 2-propen-1-ol or carbon tetrachloride following subacute concurrent chemical exposure, male Fischer 344 rats (approximately 70 d old) were given ethanol at 0, 0.05, 0.1, 0.2 or 0.5 mL/kg in corn oil daily for 14 days, or the same levels of ethanol with 21 mg/kg 2-propen-1-ol or the same levels of ethanol with 20 mg/kg carbon tetrachloride.
Result: No interactive toxicity was observed
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Purity: >99%
Supplier: Aldrich Chemical Co. (Milwaukee, WI)
Reliability: (4) not assignable
Published study using non-standard methods
05-DEC-2005 (22)

In Vitro/in vivo: In vivo
Species: rat

Method: other: Mechanistic Study
Year: 2003
Test substance: other TS: refer to freetext

Method: Animals: male Sprague-Dawley rats (250-300 g, 8-9 weeks old)

Treatments:
After an acclimation period of 1 week, the rats were divided into four groups (3 rats/group) for individual treatments (5, 20, 35 or 50 mg/ kg 2-propen-1-ol; 74, 185, 370 or 740 mg/kg chloroform) and three groups (3 rats/group) for combined treatment [75 + 5 (low dose combination), 185 + 20 (moderate dose combination) or 370 + 35 (high dose combination)]. At 0, 24, 36, 48 and 72 h after dosing, rats were anesthetised and blood samples collected to measure plasma ALT. Liver samples were processed for assay of [3]H-T incorporation into hepatonuclear DNA. An aliquot of blood and liver homogenate were taken for quantitation of chloroform and 2-propen-1-ol by GC.

Mean and standard deviations were calculated for all values and statistical differences determined using one-way ANOVA followed by Duncan's multiple range tests.
Result: Liver injury was more than additive compared to the two chemicals alone when rats were dosed at the highest combination of 370 + 35 mg/kg chloroform and 2-propen-1-ol, respectively and peaked at 24 h. Though the liver damage was higher, the greater stimulation of repair kept injury from progressing, preventing hepatic failure and death. At lower dose combinations, the liver injury was no more than additive.
Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)
Purity: Technical grade
Supplier: Chemservice (Westchester, PA)

Reliability: (4) not assignable
Published study using non-standard methods
05-DEC-2005 (14)

In Vitro/in vivo: In vitro
Species: rat

Method: other: Mechanistic Study
Year: 2002
Test substance: other TS: refer to Freetext

Result: Lipopolysaccharide augments the hepatotoxicity of 2-propen-1-ol through a mechanism involving extrahepatic factors, one of which may be a component of the coagulation cascade.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
Published study using non-standard methods
05-DEC-2005 (72)

In Vitro/in vivo: In vivo
Type: Metabolism
Species: rat

Year: 1973
Test substance: other TS: refer to Freetext

Method: Animals: male albino rats of the CFE strain (200-250 g)

Animals were housed in metabolism cages which permitted the collection of urine separately from faeces. When the urine of rats dosed with an allyl compound was to be examined by paper chromatography the compound was administered as follows: 2-Propen-1-ol, allyl formate, allyl acetate, allyl propionate, allyl benzoate, allyl nitrite and acrolein were each injected subcutaneously into the lumbar region as 1% v/v solutions in arachis oil. Diallyl phthalate was injected as a 2% v/v solution in arachis oil, allyl nitrate as a 5% v/v solution in arachis oil and triallyl phosphate as a 10% v/v solution in arachis oil. Allyl stearate (0.060 g) was liquified by heating before injection and sodium allyl sulfate was administered as a 6% w/v suspension. S-(3-Hydroxypropyl)-L-cysteine was administered subcutaneously as a 10% w/v suspension. Allyl propionate and triallyl phosphate were also injected intraperitoneally or given by stomach tube. Urine was collected for two succesive 24 h periods immediately after dosing.

Experiments with rats fed with [35]S-labelled yeast: [35]S-labelled yeast was prepared from a medium containing [35]S-sulfate and fed to rats as a 5% w/w mixture in their diet. The urine was chromatographed in four solvent mixtures and the chromatograms examined for [35]S-labelled compounds.
Result: For 2-propen-1-ol: 3-hydroxypropylmercapturic acid was detected in the urine of rats (72 animals) dosed with 2-propen-1-ol during the first 24 hour period only. The percentage conversion was 6.3%. Paper chromatograms of urine of rats fed with [35]S-labelled yeast and dosed with

2-propen-1-ol supported this finding. No allylmercapturic acid was detected.

Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)
Supplier: BDH Chemicals Ltd, Poole Dorset UK

Conclusion: The failure to detect allylmercapturic acid as a metabolite of 2-propen-1-ol suggests that 2-propen-1-ol is not metabolised by alkylation of gutathione with the allylic double bond remaining intact. It is possible that acrolein (a derivative of 2-propen-1-ol containing a carbonyl moiety) is the molecule that conjugates with glutathione.

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (70)

In Vitro/in vivo: In vitro
Species: rat
Doses, males: 0, 0.4 or 0.8 mM

Method: other: Mechanistic Study
Year: 1989
Test substance: other TS: refer to Freetext

Method: The time course of 2-propen-1-ol-induced toxicity was studied in hepatocytes freshly isolated from male Fischer 344 rats by a two step collagenase perfusion technique.

Result: Incubation of hepatocytes with 0.4 or 0.8 mM 2-propen-1-ol rapidly depleted GSH. GSH concentrations decreased 65-75% within 10 minutes of incubation and remained depressed throughout the 2 h incubation period. Control hepatocytes demonstrated slow loss of GSH during the 2-h period. Significant accumulations of MDA were detected 20 min in hepatocytes incubated with either dose of 2-propen-1-ol. PSH concentrations were significantly diminished at 20 min with 0.8 mM and at 30 min with 0.4 mM 2-propen-1-ol and continued to decline throughout the incubation. Hepatocyte viability, measured as LDH leakage into the medium, was the last of the parameters to be affected by exposure to 2-propen-1-ol (30 min for 0.8 mM and 45 min for 0.4 mM).

Sulfhydryl compounds makedly delayed the depletion of GSH, prevented significant loss of PSH and protected cells against viability loss.

Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)

Conclusion: The sequence of events was as follows: an initial rapid depletion of glutathione (GSH), a subsequent increase in malondialdehyde (MDA) and decrease in protein sulfhydryl groups (PSH) and the eventual loss of membrane integrity.

Overall, the results suggest that the inactivation of protein thiol groups is critical for 2-propen-1-ol toxicity whereas lipid peroxidation is not essential to the toxic process.

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (56)

In Vitro/in vivo: In vivo
Species: rat

Method: other: Mechanistic Study
Year: 1997
Test substance: other TS: refer to Freetext

Method: Rats were pretreated with lipopolysaccharide (LPS) (100 ug/kg) 2 hours before treatment with a minimally toxic dose of 2-propen-1-ol (30 mg/kg). Liver toxicity was assessed 18 hours later from activities of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) in plasma and from histologic changes in liver sections.
Result: Plasma ALT and AST activities were not elevated significantly in rats treated with vehicle, LPS or 2-propen-1-ol alone, but pronounced increases were seen in rats treated with LPS and 2-propen-1-ol. Significant liver damage occurred as early as 2 hours after 2-propen-1-ol treatment in LPS-pretreated rats and peaked at 6 hours. LPS treatment did not affect the rate of production of NADH in isolated livers perfused with 2-propen-1-ol; thus LPS does not appear to increase the bioactivation of 2-propen-1-ol into acrolein. However, pretreatment with 4-methylpyrazole, an inhibitor of alcohol dehydrogenase, abolished the hepatoxicity of 2-propen-1-ol in LPS treated rats, indicating the production of acrolein was needed for LPS enhancement of the toxicity of 2-propen-1-ol.
Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (152)

In Vitro/in vivo: In vitro

Method: other: Mechanistic Study
Year: 2003
Test substance: other TS: refer to Freetext

Method: Male Sprague-Dawley rats weighing 125-175 g were used for hepatic parenchymal cell isolation. Hepatocytes were isolated from the livers of the rats by collagenase perfusion. Cells were exposed to various compounds for 0-2 hours at 37°C before the addition of 2-propen-1-ol (0-100 µM) or acrolein (0-125 uM). After incubation they were assessed for hepatocellular injury by measuring the release of ALT into the medium. Other assays performed were PKCdelta immunoblotting and single-stranded DNA ELISA. Immunochemistry was also studied.
Result: The data provide evidence that PKCdelta is critically involved in the signal transduction pathway of 2-propen-1-ol/acrolein-mediated celll death in hepatocytes.
Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)
Supplier: Sigma-Aldrich Chemicals (St Louis, MO)
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (92)

In Vitro/in vivo: In vitro

Method: other: Mechanistic Study
Year: 1987

Test substance: other TS: refer to Freetext

Method: 2-Propen-1-ol and bromobenzene were studied using an in vitro system of cultured liver slices from control and phenobarbital-treated rats, respectively.
Result: Dose- and time-dependent increases in media lactate dehydrogenase (LDH) and decreases in slice K+ content and in protein synthesis were observed in rat liver slices incubated with either compound at concentrations between 0.1 and 1 mM over a period of 6 hours. The histopathological changes which occurred in the intoxicated slices appeared to parallel these biochemical changes. 2-Propen-1-ol toxicity, evaluated at 4 hours, was inhibited when slices were preincubated with pyrazole (1.0 mM).
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
Published study using non-standard methods
05-DEC-2005 (148)

In Vitro/in vivo: In vitro

Method: other: Mechanistic Study
Year: 1995
Test substance: other TS: refer to Freetext

Method: Male Fischer 344 rats weighing 200-250 g were used for hepatic cell isolation. Hepatocytes were isolated from the livers of the rats by collagenase perfusion.
After exposure to 2-propen-1-ol, cytotoxicity was assessed on the basis of leakage of lactate dehydrogenase (LDH) from hepatocytes.Changes in mitochondrial membrane potential were measured on the basis of the cellular retention of rhodamine 123, a fluorescent cationic dye.
Result: 2-Propen-1-ol induced LDH leakage from isolated rat hepatocytes was preceded by a decrease in rhodamine 123 retention, signifying a loss of mitochondrial membrane potential. Addition of dithiothreitol (DTT) prevented the drop in membrane potential and completely prevented cell killing by 2-propen-1-ol. In contrast, cyclosporin A and trifluorperazine delayed the loss of membrane potential without affecting cytolethality.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Purity: 99%
Supplier: Aldrich Chemical Co. (Milwaukee, WI)
Conclusion: The mitochondrial disfunction produced by 2-propen-1-ol appears to be the consequence of an earlier event in the toxicity that is reversible by DTT.
Reliability: (4) not assignable
Published study using non-standard methods
05-DEC-2005 (131)

Species: rat
Doses, males: 30 or 40 mg/kg
Vehicle: physiol. saline

Method: other: Mechanistic Study
Year: 1992
Test substance: other TS: refer to Freetext

Method: Animals: Male Sprague-Dawley rats (200-350 g)

Comparison of the action of 2-propen-1-ol on the liver in vivo was made using control animals and gadolinium chloride (GdCl3)-treated animals (injected with 10 mg/kg 24 hr prior to 2-propen-1-ol administration; reduces the number and activity of Kupffer cells). 2-Propen-1-ol administered intraperitoneally to control or GdCl3-treated rats at a dose of 30 or 40 mg/kg.
Hepatocyte isolation and liver perfusion: Rats were anesthetised with sodium pentobarbital and livers perfused with Krebs-Henseleit bicarbonate buffer in a non-recirculating system. For perfusion studies 2-propen-1-ol (350 uM) was infused at a flow rate of 25 mL/min into the livers of untreated or GdCl3-treated rats for 2 hours for a total dose of approximately 6 mg 2-propen-1-ol per gram of tissue.

Viability and energy status: Viability of isolated hepatocytes was assessed by following the time course of trypan blue uptake and release of lactate dehydrogenase (LDH) at 15 to 30 minute intervals during incubations with or without 2-propen-1-ol (300 uM).

In other experiments, the dose response of injury to isolated hepatocytes by 2-propen-1-ol and acrolein (100, 250 and 500 uM) was studied.

Histopathology: The effects of 2-propen-1-ol and GdCl3 on hepatic morphology in vivo were assessed by light microscopy.

Result: Administration of 2-propen-1-ol (30 or 40 mg/kg, ip) to rats caused extensive hepatic necrosis localised primarily to periportal regions. The extent of hepatic damage assessed by light microscopy and serum enzymes aspartate aminotransferase and alanine aminotransferase was markedly attenuated by pretreatment with GdCl3. Thus O2-dependent hepatic necrosis caused by 2-propen-1-ol involves the presence of Kupffer cells. GdCl3 did not prevent toxicity in the perfused liver indicating that circulating blood elements may also contribute to injury of the liver by 2-propen-1-ol in vivo.

Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Supplier: Kodak Chemical Co.

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (122)

In Vitro/in vivo: In vitro
Species: other: rat, guinea pig, monkey, human

Method: other: Mechanistic Study
Year: 1996
Test substance: other TS: refer to Freetext

Method: Investigation of 2-propen-1-ol toxicity (as well as coumarin and menadione) in precision cut liver slice cultures.

Doses 1.0, 2.5 mM
Result: Rat liver less sensitive to 2-propen-1-ol than other species.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (3) invalid
05-DEC-2005 (120)

In Vitro/in vivo: In vivo
Species: rat
No. of animals, males: 39
Doses, males: 20 mg/kg
Vehicle: other: sunflower oil

Method: other: Mechanistic Study
Year: 1977
Test substance: other TS: refer to Freetext

Result: In male Sprague-Dawley rats the relative organ weight and mitotic activity of the adrenal cortex were investigated after intoxication by a single dose of 200 mg/kg b.w. of 2-acetylaminofluorene (AAF) or 20 mg/kg b.w. of 2-propen-1-ol. At 36, 42, 48 and 72 hours after application a significant enhancement of the mitotic rate of the adrenocortical cells was produced by AAF in comparison with that following 2-propen-1-ol administration. Interrelations of organ weight and proliferative activity could not be found.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
06-JAN-2006 (36)

In Vitro/in vivo: In vivo
Species: rat
No. of animals, males: 12
Doses, males: 0 or 30 mg/kg
Vehicle: other: sunflower oil

Method: other: Mechanistic Study
Year: 1977
Test substance: other TS: refer to freetext

Result: Cyclic(C)AMP was studied in male Sprague-Dawley rats administered a single dose of 2-propen-1-ol. Adrenal (C)AMP concentration increased 10 hr after intoxication compared to controls. In final stage of experiment (30-39 hr after application) all intoxication was associated with significant increases in adrenal (C)AMP concentration.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
05-DEC-2005 (35)

In Vitro/in vivo: In vivo
Species: rat

Method: other: Mechanistic Study
Year: 1978
Test substance: other TS: refer to Freetext

Result: Three hours after oral administration to rats of 1 ml/100 g body weight of 2-propen-1-ol, cytochrome p450 was increased by 33% in liver microsomes. Aminopyrine demethylase and dimethylaniline demethylase were stimulated. Activity of microsomal hydroxylation system gradually decreased.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (3) invalid
05-DEC-2005 (47)

In Vitro/in vivo: In vivo
Species: rat
No. of animals, males: 4
Doses, males: 40 mg/kw
Vehicle: other:corn oil

Method: other: Mechanistic Study
Year: 1984
Test substance: other TS: refer to Freetext

Method: Strain: Sprague Dawley
Result: 2-Propen-1-ol administration increased serum alanine aminotransferase activity but had no effect on serum gamma-glutamyl transferase activity.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
05-DEC-2005 (81)

In Vitro/in vivo: In vivo
Species: rat
No. of animals, females: 56
Doses, females: 100, 120, 140, 160, 180, 200 uL in 2 mL of saline (100 ug/kg)

Method: other: Mechanistic Study
Year: 1984
Test substance: other TS: refer to Freetext

Result: The prothrombin index was reduced to a minimum after 12 hr and reestablished after 24 hr. The galactose elimination capacity was not changed. Hepatic glutathione content was unchanged for the first 24 hr but was then elevated two-fold. Microsomal D-nitroanisole demethylase showed a slight initial increase and a subsequent reduction. This indicates that in chemical liver damage ribosomal function is more vulnerable than cytosolic phosphorylation of carbohydrate.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Supplier: Merck
Reliability: (4) not assignable
05-DEC-2005 (118)

In Vitro/in vivo: In vivo
Species: mouse
No. of animals, males: 3
Doses, males: 0.05 mL/kg
Vehicle: physiol. saline

Method: other: Mechanistic Study
Year: 1977
Test substance: other TS: refer to Freetext

Method: Strain: NMRI
Result: G-SH (micromoles/g liver fresh weight)
Control; 9.87 (9.46-10.27)
2-Propen-1-ol; 5.48 (5.17-5.79)

The 0.05 ml/kg of 2-propen-1-ol administered orally to mice caused depletion of hepatic glutathione activity. The extent of depletion was 44.4% of control values.
Test substance: Name: 2-Propen-1-ol (CAS No. 107-18-6)
Reliability: (4) not assignable
05-DEC-2005 (145)

In Vitro/in vivo: In vivo
Species: rat
No. of animals, males: 130

Method: other: Mechanistic Study
Year: 2000
Test substance: other TS: refer to Freetext

Method: 130 wistar rats (male) divided into 2 groups: Control group (n=10) and Group II (n=120) given 2-propen-1-ol (intraperitoneally; 0.62 mmol/kg) and rhinogastric administration of carbon tetrachloride (0.66 mL/kg, 1:1 dilution in corn oil).

Animals sacrificed 2, 4, 6, 12, 18, 24, 33, 48, 57, 81 and 133 hrs after dosing.

Liver analysed.
Result: Liver sections demonstrated periportal and pericentral necrosis, peaking 57 hours after administration.
Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)
Chemical name: carbon tetrachloride (CAS No. 56-23-5)
Reliability: (3) invalid
Animals treated with two chemicals simultaneously
05-DEC-2005 (111)

In Vitro/in vivo: In vitro
Species: rat

Method: other: Mechanistic Study
Year: 1985
Test substance: other TS: refer to Freetext

Method: Animals: male and female Sprague-Dawley rats (200-300 g)

Renal epithelial cells were taken and incubated. GSH was assayed. To obtain cells with a low activity of aldehyde dehydrogenase, rats pretreated with disulfiram for 2 days before cell isolation. Partial depletion of renal cellular GSH content by treatment with diethyl maleate was observed.
Result: Cells from female rats demonstrated a greater susceptibility to 2-propen-1-ol toxicity as assessed by glutathione depletion and loss of cell viability. Sensitivity of female rat renal cells appears to relate to the higher activity of alcohol dehydrogenase found in the female rat kidney, which metabolizes 2-propen-1-ol to the highly reactive aldehyde, acrolein. Pyrazole, which inhibits alcohol dehydrogenase, abolished the cytotoxic effects of 2-propen-1-ol, whereas inhibition of aldehyde dehydrogenase by disulfiram treatment was found to increase the sensitivity of renal cells to 2-propen-1-ol. The toxicity of 2-propen-1-ol was decreased by a number of treatments which resulted in increased levels of glutathione or other low molecular weight thiols.

Test substance: Chemical name: 2-Propen-1-ol (CAS No. 107-18-6)
Purity: reagent grade

Conclusion: These results indicate that acrolein is the toxic metabolite responsible for the renal cell injury following exposure to 2-propen-1-ol, and unless immediately inactivated acrolein interacts with critical nucleophilic sites of the cell and initiates cell injury.

Reliability: (4) not assignable

05-DEC-2005 (110)

In Vitro/in vivo: In vivo

Species: rat

Method: other: Mechanistic study

Year: 1978

GLP: no

Test substance: other TS: 2-Propen-1-ol (CAS No.

Method: Male Wistar albino rats (120-150 g) were used in these studies. They were allowed free access to laboratory diet and water. 2-Propen-1-ol was administered by daily gastric intubation at a dose of 30 mg/kg bw/day for periods of 1, 10 or 28 days. Control animals received appropriate quantities of of the corn-oil vehicle.

Rats were killed by cervical dislocation and liver homogenates (0.25 g of tissue/mL) were prepared in 0.154M-KCl containing 50mM-tris/HCl buffer, pH 7.4. Succinate dehydrogenase (EC 1.3.99.1) activity was determined on the whole-liver homogenates by the method of Pennington (1961). A portion of the whole homogenate was centrifuged at 10,000 gav. for 20 minutes and the postmitochondrial-supernatant fraction was used for determination of benzo[a]-pyrene hydroxylase activity. The 10,000 gav supernatant fraction was further centrifuged at 105,000gav for 60 minutes to separate the microsomal fraction from the ytosol. Cytochrome P-450 content was determined in the microsomal fraction and alcohol dehydrogenase activity was measured in the cytosol. Histological examination of liver tissue and the histochemical demonstration of alcohol dehydrogenase and succinate dehydrogenase activities were performed.

Result: The administration of a single oral dose of 2-propen-1-ol produced marked periportal necrosis that was associated with losses of alcohol dehydrogenase and succinate dehydrogenase activities from the portal areas of the liver lobule. Parallel biochemical investigations confirmed the decrease of activities of both dehydrogenase enzymes. In addition, the activity of benzo[a]pyrene hydroxylase and hepatic content of cytochrome p-450 were decreased to 59-66% of control values showing an inhibition of hepatic xenobiotic metabolism.

However, further daily administration of 2-propen-1-ol for periods of either 10 or 28 days did not produce any potentiation of the hepatotoxic effects observed after a single dose. All the parameters measured had reverted to control values and histological examination of liver sections from treated animals did not reveal any obvious abnormalities.

Conclusion: The results of the present study demonstrate the unusual finding that the prolonged administration of a hepatotoxic agent resulted in the complete regression of the initial liver

damage. In the absence of any results on alterations in the absorption or distribution of 2-propen-1-ol from the gastrointestinal tract, it must be assumed that the hepatic metabolism of the hepatotoxin becomes modified as a result of repeated treatment. Such an alteration in the metabolism of 2-propen-1-ol may involve the suppression of the alcolhol dehydrogenase-catalysed activation pathway and the diversion of the alcohol via alternative pathways leading to non-toxic metabolites.

Reliability: (2) valid with restrictions
Well reported published study

07-DEC-2005 (79)

5.1 Acute Toxicity

5.1.1 Acute Oral Toxicity

Type: LD50
Species: rat
Strain: Long-Evans
Sex: male
No. of Animals: 5
Vehicle: water
Doses: 75-130 mg/kg bw; 79-140 mg/kg bw
Value: 99 - 105 mg/kg bw

Year: 1958
GLP: no
Test substance: other TS: name: 2-Propen-1-ol (CAS No. 107-18-6); supplied by Shell Chemical Company; purity 98.5%; impurities diallyl ether, water.

Method: Graded amounts of a 1% solution of 2-propen-1-ol were administered by gavage (dosing needle) to groups of 5 male rats (body weights: 111-143 g or 170-252 g; two studies). Surviving animals were observed for up to 10 days.

No further experimental details provided.

The LD50 was calculated according to the method of Weil (1952) Biometrics, 8, 343.

Result: The main clinical sign was described as apathy, along with anxiety. Coma and diarrhea preceded death in moribund animals.

Gross post mortem findings in decedent animals included:
- edema and congestion of the lungs
- visceral congestion
- presence of mucus in the intestinal tract
- discolored liver (some necrosis)
- swollen, discolored kidneys

Histopathological examination of tissue from decedent animals revealed:
- lung congestion
- liver damage (congestion and necrosis of periportal sinusoids, central pallor and necrosis)
- presence of heme casts and cloudy swelling in the kidney.

Similar (but less frequent) lesions were present in animals that survived the 10 d observation period.

Calculated oral LD50 values of 99 mg/kg bw (for animals weighing 170-252 g) and 105 mg/kg bw (for animals weighing 111-143 g) were obtained from the study.

Conclusion: Under the conditions of the test, an oral LD50 of 99-105 mg/kg bw was obtained in male rats given 2-propen-1-ol by oral gavage.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

19-DEC-2005 (39)

Type: LD50
Species: mouse
Strain: Swiss Webster
Sex: male
No. of Animals: 6
Vehicle: water
Doses: 84-110 mg/kg bw
Value: 96 mg/kg bw

Year: 1958
GLP: no
Test substance: other TS: name: 2-Propen-1-ol (CAS No. 107-18-6); supplied by Shell Chemical Company; purity 98.5%; impurities diallyl ether, water.

Method: Graded amounts of a 1% solution of 2-propen-1-ol were administered by gavage (dosing needle) to groups of 6 male mice (17.5-22.5 g). Surviving animals were observed for up to 10 days.

No further experimental details provided.

The LD50 was calculated according to the method of Weil (1952) Biometrics, 8, 343.

Result: The main clinical sign was described as apathy preceded by excitability. Ataxia was occasionally present.

Gross post mortem findings in decedent animals included:
- occasional edema and congestion of the lungs
- no other abnormalities present

No microscopic changes were detected

The calculated oral LD50 was 96 mg/kg bw.

Conclusion: Under the conditions of the test, an oral LD50 of 96 mg/kg bw was obtained in male mice given 2-propen-1-ol by oral gavage.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

05-DEC-2005 (39)

Type: LD50
Species: rabbit
Strain: other: albino (no further details)

Sex: male
No. of Animals: 3
Vehicle: water
Doses: 42-125 mg/kg bw
Value: 71 mg/kg bw

Year: 1958
GLP: no
Test substance: other TS: name: 2-Propen-1-ol (CAS No. 107-18-6); supplied by Shell Chemical Company; purity 98.5%; impurities diallyl ether, water.

Method: Graded amounts of a 2% solution of 2-propen-1-ol were administered by means of a soft rubber catheter to groups of 3 male rabbits (body weights: 2.9-3.2 kg). Surviving animals were observed for up to 10 days.

No further experimental details provided.

The LD50 was calculated according to the method of Weil (1952) Biometrics, 8, 343.

Result: The main clinical sign was described as an attitude of bellicosity. Flushing of skin were noted. Ataxia was noted occasionally; convulsions proceeded the death. Diarrhea occurred before death.

Gross post mortem findings in decedent animals included:
- edema and congestion of the lungs
- visceral congestion
- presence of mucus in the intestinal tract
- discolored liver (some necrosis)
- swollen kidneys

Histopathological examination of tissue from decedent animals revealed:
- lung congestion
- liver damage (congestion and necrosis of periportal sinusoids, central pallor and necrosis)
- presence of heme casts and cloudy swelling in the kidney.

Similar (but less frequent) lesions were present in animals that survived the 10 d observation period.

No microscopic changes were detected

The calculated oral LD50 was 71 mg/kg bw.

Conclusion: Under the conditions of the test, an oral LD50 of 71 mg/kg bw was obtained in male rabbits given 2-propen-1-ol by oral gavage.
Reliability: (2) valid with restrictions
Well reported published study
Flag: Critical study for SIDS endpoint
05-DEC-2005 (39)

Type: LD50
Species: rat
Strain: Osborne-Mendel
Sex: male/female
No. of Animals: 10
Vehicle: water
Doses: 2%

Value: 70 mg/kg bw

Year: 1964
GLP: no
Test substance: other TS: name: 2-Propen-1-ol (CAS No. 107-18-6); no other information available

Method: Groups of young Osborne-Mendel rats (5 per sex per dose level) were fasted (18 hr) prior to administration of 2% aqueous 2-propen-1-ol by gavage (dose range or number of treatment groups not stated). Animals were observed for up to 2 weeks. The LD50 was calculated according to the method of Litchfield and Wilcoxon (1949) J Pharmacol 96, 99.
Result: LD50 = 70 mg/kg bw (95% CI=63-79).

Clinical signs: depression, colourless secretion from eyes, diarrhoea, scrawny appearance for several days.

Death time: between 4 hr and 4 days.
Conclusion: Under the conditions of the test, an oral LD50 of 70 mg/kg bw was obtained in male rats given 2-propen-1-ol by oral gavage.
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (64)

Type: LD50
Species: rat
Strain: Wistar
Sex: no data
No. of Animals: 10
Value: 64 mg/kg bw

Year: 1948
GLP: no
Test substance: other TS: name: 2-Propen-1-ol (CAS No. 107-18-6); no other information available

Remark: Ten rats per group were used.

No more information on methods or findings available
Result: Maximum dose having no effect: 4.0 mg/kg

LD50 = 64 (56-74) mg/kg
Reliability: (4) not assignable
05-DEC-2005 (150)

Type: LD50
Species: mouse
Value: 85 mg/kg bw

Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (4) not assignable
No details of study or source of data available
05-DEC-2005 (165)

5.1.2 Acute Inhalation Toxicity

Type: LC50
Species: rat
Strain: Long-Evans
Sex: male
No. of Animals: 6
Doses: 95-545- mg/m3 (nominal)
Value: 140 - 150 mg/m³

Year: 1958
GLP: no
Test substance: other TS: Name: 2-Propen-1-ol (CAS No. 107-18-6); Supplier: Shell Chemical Company, Purity: 98.5%, Impurities diallyl ether, water.

Method: Exposure Time: 1, 4, 8 hour(s)

Groups of 6 male rats (100-200 g) were exposed for 1, 4 or 8 hr to 40-2300 ppm+ 2-propen-1-ol vapor in a glass chamber (nominal volume 19.5 L). Animals were observed for 10 days post-treatment.
[+ equivalent to 95-5450 mg/m3; based upon 1 ppm = 2.37 mg/m3; Bevan (2001), Patty's Toxicology, 5th edition, p4631

The test atmosphere was generated by passing liquid 2-propen-1-ol via a syringe pump into an evaporation chamber through which air flowed at 8.6 to 12.9 L/min, depending on the desired exposure concentration. The atmosphere within the chamber was allowed to equilibrate to 95-99% of the desired concentration before introduction of the animals. The nominal concentration in the chamber was calculated according to Jacobs (1949) The Analytical Chemistry of Industrial Poisons, Hazards and Solvents, 2nd edition, Interscience Publishers Inc., NY.

Glass bottles of 1 L capacity containing distilled water were connected to the sampling port o f the chamber and vapor drawn through the water by suction. 0.01N bromine in acetic acid and a mercuric acetate catalyst were added to the sample, the excess bromine reduced by iodide and the iodide titrated with 0.01N thiosulfate (Reid and Beddard (1954) Analyst, 79, 456)

The LC50 was calculated according to the method of Weil (1952) Biometrics, 8, 343.

Result: Coma and diarrhea preceded death in moribund animals.

Gross post mortem findings in decedent animals included: -
edema and congestion o f the lungs
- visceral congestion
- presence of mucus in the intestinal tract
- discolored liver (some necrosis)
- swollen, discolored kidneys

Histopathological examination of tissue from decedent animals revealed:
- lung congestion
- liver damage (congestion and necrosis of periportal sinusoids, central pallor and necrosis)

- presence of heme casts and cloudy swelling in the kidney. Similar (but less frequent) lesions were present in animals that survived the 10 d observation period.

A calculated LC50 values was obtained from the study.
1h LC50 = 2510 (2060-3060) mg/m3
4h LC50 = 380 (300-530) mg/m3
8h LC50 = 180 (160-200) mg/m3
Chemical analysis of vapor drawn from the exposure chamber revealed a 15-25% loss of 2-propen-1-ol.

After correction therefore, the LC50s were in a range of
1h LC50 = 1900-2130 mg/m3
4h LC50 = 300- 330 mg/m3
8h LC50 = 140- 150 mg/m3

Conclusion: Under the conditions of the test, each acute LC50 of 1900-2130, 300-330, 140-150 mg/m3 was obtained for male rats exposed to 2-propen-1-ol vapor by inhalation for 1, 4, 8 hr, respectively.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

05-DEC-2005 (39)

Type: LC50
Species: rat
Strain: Sherman
Sex: male/female
No. of Animals: 6
Exposure time: 4 hour(s)
Value: ca. 590 mg/m³

Year: 1949
GLP: no
Test substance: other TS: Name: 2-Propen-1-ol (CAS No. 107-18-6), no further information available

Method: Six male or female Sherman rats (approx. 100 - 150 g) were exposed to 2-propen-1-ol vapor (nominal concentrations up to 590 mg/m3 (250 ppm)) for 4 hr, and the animals observed for a 14 d.

The test atmosphere was generated by passing liquid 2-propen-1-ol into an heated evaporation chamber through which metered air was forced. Rats were exposed in a 9 L desiccator fitted with inlet and outlet ports.

The reported values are nominal (based on weight of material evaporated) and not verified analytically.

Result: Tabulated summary information included in the report notes that exposure to 250 ppm 2-propen-1-ol resulted in mortality in 2/6, 3/6 or 4/6 rats.

Conclusion: Under the conditions of the test, an acute LC50 of 590 mg/m3 (250 ppm) was obtained for male rats exposed to 2-propen-1-ol vapor by inhalation for 4 hr.

Reliability: (4) not assignable

05-DEC-2005 (30)

Type: other:

Species: rat
No. of Animals: 6
Doses: 1000 ppm
Exposure time: 1 hour(s)

Year: 1948
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Result: Mortality: 4/6
Reliability: (4) not assignable
Range-finding study
05-DEC-2005 (150)

5.1.3 Acute Dermal Toxicity

Type: LD50
Species: rabbit
Strain: other: albino (no further details)
Sex: male
No. of Animals: 3
Doses: 25-200 mg/kg bw (4 treatment levels)
Value: 89 mg/kg bw

Year: 1958
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: 98.5%, Supplier: Shell Chemical Company

Method: 4 groups of 3 male rabbits (1.3-3.9 kg) were exposed to 25 to 200 mg/kg bw 2-propen-1-ol.

Patches of rubber dam (3x3 cm) were placed over gauze (1 cm diameter) and sealed to clipped skin using rubber cement. 2-Propen-1-ol (25-200 mg/kg bw) was injected through the dam, onto the skin surface, and the puncture site sealed (rubber cement). The body was then further wrapped with toweling and adhesive tape to protect the dressing.

Animals were observed for 10 days post-treatment.

The LD50 was calculated according to the method of Weil (1952) Biometrics, 8, 343.

Result: The main clinical sign was described as apathy, along with flushing of the skin. Ataxia and diarrhea preceded death in moribund animals.

Gross post mortem findings in decedent animals included:
- edema and congestion of the lungs
- visceral congestion - presence of mucus in the intestinal tract
- discolored liver (some necrosis)
- swollen kidneys.

Histopathological examination of tissues from decedent animals revealed:
- lung congestion
- liver damage (congestion and necrosis of periportal sinusoids, central pallor and necrosis)
- heme casts and cloudy swelling in the kidney

Similar (but less frequent) histopathological lesions were present in animals that survived the 10 d observation period.

The calculated dermal LD50 was 89 mg/kg bw.

Conclusion: Under the conditions of the test, a dermal LD50 of 89 mg/kg bw was obtained in the rabbit following 24 hr occluded exposure to 2-propen-1-ol.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

05-DEC-2005 (39)

Type: LD50
Species: rabbit
Value: .053 ml/kg bw

Year: 1948
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: No information on methods or findings available.

Based on a density of 0.85 g/mL, this is equivalent to approx. 45 mg/kg bw.

Reliability: (4) not assignable

05-DEC-2005 (150)

Type: LD50
Value: 45 mg/kg bw

Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (4) not assignable

05-DEC-2005 (82)

5.1.4 Acute Toxicity, other Routes

Type: LD50
Species: rat
Strain: Long-Evans
Sex: male
No. of Animals: 10
Vehicle: water
Doses: 32-55 mg/kg bw
Route of admin.: i.p.
Value: 42 mg/kg bw

Year: 1958
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: 98.5%, Supplier: Shell Chemical Company; Impurities diallyl ether, water

Method: Graded amounts of a 1% solution of 2-propen-1-ol were administered by intraperitoneal injection to groups of 10 male rats (178-200 g). Surviving animals were observed for up to 10 days.

No further experimental details provided.

The LD50 was calculated according to the method of Weil (1952) Biometrics, 8, 343.

Result: The main clinical sign was described as apathy, along with anxiety. Coma and diarrhea preceded death in moribund animals.

Gross post mortem findings in decedent animals included:
- edema and congestion of the lungs
- visceral congestion
- presence of mucus in the intestinal tract
- discolored liver (some necrosis)
- swollen, discolored kidneys

Histopathological examination of tissue from decedent animals revealed:
- lung congestion
- liver damage (congestion and necrosis of periportal sinusoids, central pallor and necrosis)
- presence of heme casts and cloudy swelling in the kidney.

Similar (but less frequent) lesions were present in animals that survived the 10 d observation period.

Calculated i.p. LD50 value of 42 mg/kg bw was obtained from the study.

Conclusion: Under the conditions of the test, an i.p. LD50 of 42 mg/kg bw was obtained in male rats given 2-propen-1-ol intraperitoneally.

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (39)

Type: LD50
Species: mouse
Strain: Swiss Webster
Sex: male
No. of Animals: 6
Vehicle: water
Route of admin.: i.p.
Value: 60 mg/kg bw

Year: 1958
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: 98.5%, Supplier: Shell Chemical Company; Impurities diallyl ether, water

Method: Graded amounts of a 1% solution of 2-propen-1-ol were administered by intraperitoneal injection to groups of 6 male mice (18.5-23 g). Surviving animals were observed for up to 10 days.

No further experimental details provided.

The LD50 was calculated according to the method of Weil (1952) Biometrics, 8, 343.

Result: The main clinical sign was described as apathy preceded by excitability. Ataxia was occasionally present.

Gross post mortem findings in decedent animals included:

- occasional edema and congestion of the lungs
- no other abnormalities present

No microscopic changes were detected

The calculated i.p. LD50 was 60 mg/kg bw.

Conclusion: Under the conditions of the test, a i.p. LD50 of 60 mg/kg bw was obtained in male mice given 2-propen-1-ol by intraperioneal.

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (39)

Type: LD50
Species: rat
Strain: Fischer 344
Sex: male
No. of Animals: 29
Vehicle: other: corn oil
Route of admin.: i.p.
Value: 37 mg/kg bw

Year: 1986
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Method: ANIMALS: Fischer 344 rats were obtained from Charles River Labs (Cambridge, Mass.). Immature animals were 11 days old at the beginning of the experiments (both sexes from litters of 10 pups), and adults were 75 to 90 days old (male, grouped five per cage). Both sexes of immature rats but males only of adult rats were used. The animals were fed NIH-07 diet and given water ad libitum. There were three groups.

Immature rats were injected i.p. with 0.1 ml/10 g bw of a 1:500 solution in 0.85% saline, and adults were injected i.p. 1.8 ml/kg of a 1:50 solution. Consequently, immature animals received 0.294 mmol/kg while adults rats received 0.53 mmol/kg. Immature animals were treated 14 hr and adult rats 24 hr prior to being killed. These times and doses differed because immature animals are more sensitive to respiratory toxicity than are adults.

Control: Immature rats received 0.2ml corn oil/10g bw i.p., and adults received 1.5ml corn oil/kg 24 or 36 hr prior to being killed.

Result: LD50, 7 days observation (with 95% confidence limits)

Immature rats (11 days, n=21): 0.500 mmol/kg (29.0 mg/kg), 0.382-0.617 mmol/kg

Adult rats (75 days, n=29): 0.641 mmol/kg (37.2 mg/kg), 0.600-0.682 mmol/kg

Immature rat:
-no histological alteration

Adult rat:
-moderate to marked periportal necrosis with attendant

inflammation and hemorrhage
-considerable variability in extent of hepatotoxicity in different lobes
-1/5; passive congestion

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (74)

Type: EC50
Species: mouse
Strain: other: Ssc:CF-1
Sex: male
No. of Animals: 20
Doses: 0.42, 2.00, 4.55, 15.10 ppm (normal) 3.62 (cannula)
Route of admin.: other: inhalation (normal or via tracheal cannula)
Exposure time: 30 minute(s)
Value: 3.9 ppm

Year: 1984
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Supplier: Merck, Purity: 98%

Result: RD50 = 3.9 ppm (equivalent to 9.26 mg/m3)
RD50(20-30 min) = 4.8 ppm (equivalent to 11.4 mg/m3)

2-Propen-1-ol induced a decrease in the respiratory rate due to sensory irritation in normal (non-cannulated) mice. The effects were very rapid, normally reaching a plateau within the first 10 minutes. After exposure, the sensory irritating responses died away very rapidly. No pulmonary irritation was observed at concentrations up to the RD50 in cannulated mice, therefore the respiratory depression observed was due entirely to the chemical's sensory irritating effect.

Test condition: Groups of 4 mice used for each exposure level.
Evaluation of sensory irritation, pulmonary irritation and anaesthesia:
Sensory irritation of the upper respiratory tract causes a characteristic pause before exhalation and thereby a decrease in respiratory rate. This bradypnoea occurs reflexively from stimulation of the trigeminal nerve endings in the nasal mucosa. The level of response for each exposure was taken as the percentage change in the average respiratory rate of each group of mice from their preexposure level.
Concentration-response relationships were obtained by plotting the percentage decrease in the average respiratory rate of each group versus the logarithm of the exposure concentration. From these relationships, the concentration expected to cause a 50% decrease in respiratory rate due to sensory irritation was calculated. Two values were obtained, one for the maximum decrease within the first 10 minutes (RD50) and another for the mean value of the period from 21-30 min (RD50(20-30 min)).

If exposures were carried out in animals via tracheal cannula, the bypass of the nasal trigeminal nerve endings excludes the development of sensory irritation of the upper respiratory tract. Stimulation of pulmonary receptors by airborne irritants in mice results in a different reflex decrease in respiratory rate due to a pause between the end of expiration and the beginning of the following inspiration resulting in a

net decrease in respiratory rate proportional to the log of the concentration of the irritants.

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (104)

Type: LD50
Species: mouse
Route of admin.: i.v.
Value: 78 mg/kg bw

Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (4) not assignable
Data reported in the secondary literature

05-DEC-2005 (82)

Type: other: hepatic effect
Species: rat
Strain: Sprague-Dawley
Sex: male
Vehicle: other: corn oil
Doses: 0.05 mL/kg
Route of admin.: i.p.

Year: 1982
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Result: 2-Propen-1-ol reduced hepatic cytochrome p450 in liver, and the activities of ethylmorphine demethylase, benzphetamine demethylase, benzo[a]pyrene hydroxylase, and ethoxyresorufin deethylase. No significant decrease in epoxide hydrolase or glucuronyltransferase activities were observed. The activities of cytosolic conjugating enzymes (glutathione-, sulfo- and acetyltransferases) also were minimally affected by toxic liver injury.

Test condition: The effect on the activity of enzymes of hepatic phase I (cytochrome p450-linked microsomal monooxygenases, epoxide hydrolase) and phase II (glucuronyl-, glutathione-, acetyl- and sulfotransferases) biotransformation were studied in rats.

Reliability: (4) not assignable

05-DEC-2005 (48)

Year: 1987
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Male CD1 mice and male CD rats were administered 2-propen-1-ol (0.05 ml/kg in corn oil, ip), and were sacrificed after 24 hr.

Pentobarbital sleeping time, serum glutamic pyruvic transaminase (SGPT), histologic evidence of liver necrosis, and respiratory activity of liver mitochondria were used as indices of hepatotoxicity. 2-Propen-1-ol treatment resulted in a significant decrease in pentobarbital sleeping time in mice ($p<0.05$), but significantly prolonged the sleeping time in rats ($p<0.005$). SGPT was significantly elevated in treated mice (3-fold, $p<0.05$) and rats (20-fold).

There was no visible liver necrosis in mice; livers from treated rats showed variable necrosis. Micrographs of mitochondria from treated rats showed flocculent densities in the matrix compartment. Mitochondria from control mice and rats and treated mice had normal state 3 respiratory activity and normal respiratory control. In treated rats state 3 respiratory activity was depressed relative to the control value and respiratory control was absent, indicating inability to carry out oxidative phosphorylation.

Reliability: (4) not assignable

05-DEC-2005 (61)

Type: other: hepatotoxicity
Species: rat
Strain: Fischer 344
Sex: male
Vehicle: physiol. saline
Doses: 0.036 mL/kg bw (equivalent to 30.7 mg/kg bw
Route of admin.: i.p.

Year: 1984
GLP: no data
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6) Supplied by Eastman Kodak Company

Result:

Treatment	Age	ALT (units/mL)	AST	Hepatocellular necrosis
Control	Y	57	309	0
	M	65	308	0
	O	62	240	0
Allyl Alcohol	Y	200	620	0
	M	2200	3600	+1
	O	5000	5000	+2

The toxicity was more severe in middle-aged and old rats than in young-adult rat

Test condition: -Animal
Purchased: Charles River Breeding Laboratory
Age: 4-5(Y:young-adult), 14-15(M:middle-aged), 24-25(O:old) months of age.

Number: >= 6 /group

-Test compound
0.036 mL/kg, 1.8 mL/kg of a 2% solution of 2-propen-1-ol in saline
-Control
corn oil, 2.0 mL/kg

-Parameter
(1) severity of hepatocellular necrosis as judged by light microscopy of liver section
(2) activity of alanine aminotransferase and asparate aminotransferas in serum
(3) hepatic microsomal cytochrome P-450 content and NADPH-cytochrome P-450 reductase activity

Reliability: (4) not assignable
03-JAN-2006 (127)

Species: dog

Year: 1925
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: After first depressing dogs with paraldehyde in order to delay vomiting, it was found that 0.05 mL of 2-propen-1-ol in one per cent solution causes death in about seven hours. The toxicity, based upon the lethal dose, is therefore about 150 times that of methyl alcohol. 2-Propen-1-ol was found to be present to the extent of 0.5 per cent in one sample of wood alcohol obtained from the drug store and labeled chemically pure methyl alcohol.
The symptoms of 2-propen-1-ol poisoning are similar to those commonly ascribed to wood alcohol especially the marked hyperemia of the gastric mucosa, intense vomiting convulsive movements and coma. One animal receiving a sublethal dose, developed in two days an opacity, apparently of the cornea, with blindness in one eye. This cleared up in two days more.
Reliability: (4) not assignable
05-DEC-2005 (17)

5.2 Corrosiveness and Irritation

5.2.1 Skin Irritation

Species: rabbit
Concentration: undiluted
Exposure: Occlusive
Exposure Time: 24 hour(s)
No. of Animals: 3
Result: slightly irritating

Year: 1958
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6) Supplied by Shell Chemical Company, purity 98.5%; impurities allyl ether, water

Method: 2-Propen-1-ol (0.5ml) was applied to intact and abraded skin (ventral surface) of 3 male albino rabbits. (It is not stated if fur at the treatment site was clipped first.) The application site was covered with gauze under a rubber dam, fastened with adhesive tape.
The test site was examined 24 hr post-application.
Result: Slight erythema was present at the application site (intact skin) of one animal when the patch was removed (24 hr timepoint) but this had fully resolved by 48 hr. No other reactions were noted.
Conclusion: Under the conditions of this test, 2-propen-1-ol was slightly irritating to rabbit skin.
Reliability: (2) valid with restrictions
Well reported published study
Flag: Critical study for SIDS endpoint
05-DEC-2005 (39)

Species: rabbit

Result: not irritating
EC classificat.: not irritating

Year: 1948
Test substance: other TS: 2-Propen-1-ol (CAS No 107-18-6)

Remark: 10 mg/24 h open
Reliability: (4) not assignable
05-DEC-2005 (150)

Year: 1965
Test substance: other TS: 2-Propen-1-ol (CAS No 107-18-6)

Remark: Application to skin causes slight erythema lasting about 48 hours.
Reliability: (4) not assignable
05-DEC-2005 (27)

5.2.2 Eye Irritation

Species: rabbit
Concentration: undiluted
Dose: .1 ml
Exposure Time: 4 hour(s)
No. of Animals: 6
Vehicle: none
Result: irritating

Method: Directive 84/449/EEC, B.5 "Acute toxicity (eye irritation)"
Year: 1989
GLP: no data
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), >99% pure, UCB, Brussels, Belgium.

Method: 2-Propen-1-ol (0.1 ml) was instilled in the eye (between lower eyelid and eyeball) of 3 adult Rsk:NZW rabbits, and the lids held together for approx. 1 s. The other eye served as a control. Eyes were examined and responses noted at 4, 24, 48, 72, 96 and 168 hr post-instillation. Erythema, chemosis, iritis and corneal opacity were recorded according to the method of Draize et al. (1944; J Pharmac exp Ther 82, 337) under a Philips TLE 22W/29 lamp.

In a second study conducted 6 mo later (3 additional rabbits), corneal swelling (corneal thickness) was also assessed using an ultrasonic pachometer (Ophthasonic pachometer, TEKNAR Inc, St Louis, MO) and the results expressed as the mean percentage increase for all 3 animals at 24, 48 and 72 hr.

The mean scores for erythema, chemosis and corneal opacity were calculated for all 6 rabbits at 24, 48 and 72 hr.
Result: Mean results at 24, 48 and 72 hr (n):

Erythema :	2.89 (6)
Chemosis :	1.23 (6)
Corneal opacity:	2.09 (6)

Corneal swelling (thickness) : 76% (3)

(Individual, animal- or time specific results not reported).

Conclusion: Under the conditions of the test, 2-propen-1-ol was irritating to rabbit eye.

Reliability: (2) valid with restrictions
Well reported published study using Guideline method

Flag: Critical study for SIDS endpoint

05-DEC-2005 (60)

Species: rabbit
Concentration: undiluted
Dose: 100 other: uL
No. of Animals: 6
Result: irritating
EC classificat.: irritating

Method: OECD Guide-line 405 "Acute Eye Irritation/Corrosion"
Year: 1992
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Method: -Animal: New Zealand White albino rabbits
Application 100 uL into the lower conjunctival sac. Draize scoring criteria. Application of one drop of 2% sodium fluorescein before scoring the percentage corneal damage.

-Compound preparation: neat

No further experimental details provided.

Result:

Time after application :	4h	24h	48h	72h	96h
Mean score of					
conjunctivitis (3 max.):	1.9	2.7	3.0	2.9	2.8
chemosis (4 max.) :	1.7	1.5	1.0	0.3	0.3
iritis (2 max.) :	1.0	1.0	1.0	1.0	0.7
corneal opacity (4 max.):	1.2	1.7	2.2	2.3	2.5
-Mean surface of					
corneal damage (100% max.):	90	84	56	45	28

Classification according to the EC criteria: Irritating to eyes (mean of conjunctivitis over 24/48/72 hrs > 2.5, mean of iritis over 24/48/72 hrs = 1, mean of corneal opacity over 24/48/72 hrs = 2)

Reliability: (2) valid with restrictions
Well reported published study using Guideline method

Flag: Critical study for SIDS endpoint

05-DEC-2005 (59)

Species: rabbit
Concentration: undiluted
Dose: .05 ml
Exposure Time: 48 hour(s)
No. of Animals: 3
Result: irritating

Method: Draize Test

Year: 1958
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Supplied by Shell Chemical Company, purity 98.5%; impurities diallyl ether, water
Method: 2-Propen-1-ol (0.05 ml) was instilled into the left eye of 3 male albino rabbits.

The eyes were examined after 1 hr for signs of irritation (first unstained, then after application of 5% fluorescein sodium). Further examinations were carried our at 24 hr and 48 hr and during the subsequent week.
Result: Conjunctival erythema (affecting 3/3 rabbits) and edema (affecting 1/3 rabbits) was present 1 hr post-instillation (no numerical scores reported).

At 24 hr, conjunctival erythema (score 4-6; affecting 3/3 rabbits), corneal opacity (score 5-10; affecting 2/3) and injection of the iris (score 1; affecting 1/3) was noted.

48 hr post-instillation, conjunctival redness (score 2-6; affecting 3/3 rabbits) and corneal opacity (score 5; affecting 1/3) but no iridial effects were present.

All eyes appeared normal by the end of 1 week.
Conclusion: Under the conditions of this test, 2-propen-1-ol was irritating to rabbit eye producing reversible conjunctival redness, iridial injection and corneal opacity that persisted at least 48 hr post instillation.
Reliability: (2) valid with restrictions
Well reported published study
Flag: Critical study for SIDS endpoint
19-DEC-2005 (39)

Species: rabbit
Result: irritating

Year: 1946
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), no other information available

Method: 0.005 ml or 0.02 ml 2-propen-1-ol was instilled into the eye of an undefined number of rabbits.

18-24 hr later, the eye was examined in strong daylight, then re-examined after staining with fluorescein.

The following grading system was used to record any injuries present:
- corneal opacity (max. score = 6)
- keratoconus (max. score = 6)
- iris effects (max. score = 2)
- necrosis (visible after fluorescein staining; max. score = 6)
- total maximum score = 20

Result: Descriptive information presented in the report indicates that 0.02 ml 2-propen-1-ol resulted in a total score of 5/20, while instillation of 0.005 ml resulted in a total score of 5 or less out of 20.

INTERPRETATION
The volume applied to the eye in these studies (0.005-0.02 ml) is less than that recommended in Guideline 405 (0.1 ml). A more pronounced response would be anticipated after instillation of 0.1 ml, suggesting that 2-propen-1-ol would be irritating to the eye.
Conclusion: Based on the available information, 2-propen-1-ol appears irritating to the eye of the rabbit.
Reliability: (4) not assignable
Insufficient detail in published study
05-DEC-2005 (29)

Species: other: bovine corneal
Result: irritating

Method: other: in vitro Bovine Corneal Opacity Assay
Year: 1992
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Method: -Drug: concentration: 20, 40, 60, 80, 100 (%, v/v)

-Number of corneas: 6
Result: -Concentration (%, v/v)

20;	288.7	+/-	9.2
40;	34.5	+/-	13.4
60;	63.2	+/-	13.7
80;	79.5	+/-	9.9
100;	72.0	+/-	16.1

-In vitro activity
Severe
Reliability: (4) not assignable
Insufficient detail in published study
05-DEC-2005 (44)

Year: 1965
Test substance: other TS: 2-Propen-1-ol (CAS no. 1-7-18-6)

Remark: Application to eyes causes erythema of conjunctiva and swelling of cornea, sometimes with opacity, but no permanent injury.
Reliability: (4) not assignable
Secondary data reported in the literature
05-DEC-2005 (27)

5.3 Sensitization

Type: Guinea pig maximization test
Species: guinea pig
Concentration 1st: Induction 1 other: w/v% intracutaneous
2nd: Induction 2.5 other: w/v% occlusive epicutaneous
3rd: Challenge undiluted occlusive epicutaneous
No. of Animals: 40
Vehicle: water
Result: not sensitizing

Classification: not sensitizing

Method: OECD Guide-line 406 "Skin Sensitization"
Year: 2004
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity 99.9%, Lot No. 040420

Result: (1) Skin reaction after challenge
-At 2-propen-1-ol treated groups
No change

(2) Mortality and clinical signs
-At all treated groups
No deaths and abnormality

(3) Body weight
-At all treated groups
Similar with that of control group

Table 1. Skin reactions after challenge

Group	Test substance		Control for test substance	
First induction substance				
	2-propen-1-ol (1 w/v%)		Water for injection	
Second induction substance				
	2-propen-1-ol (2.5 w/v%)		Water for injection	
Challenge substance				
	2-propen-1-ol (undiluted)		2-propen-1-ol (undiluted)	
No. of animals				
	20		10	
Hours a)				
	24	48	24	48
	Skin reaction b)			
0	20 c)	20	10	10
1	0	0	0	0
2	0	0	0	0
3	0	0	0	0
Mean response				
	0.0	0.0	0.0	0.0
Sensitization rate (%)				
	0	0	0	0

a): Time after challenge
b): 0; no visible change, 1; discrete or patchy erythema, 2; moderate and confluent erythema, 4; intense erythema and swelling
c): Number of animals

Table 2. Skin reactions after challenge (Positive control)

Group	Test substance	Control for test substance
First induction substance		

	DNCB	Olive oil	
	(0.05 w/v%)		
Second induction substance			
	DNCB	Olive oil	
	(0.05 w/v%)		
Challenge substance			
	DNCB	DNCB	
	(0.05 w/v%)	(0.05 w/v%)	
No. of animals			
	5	5	
Hours a)			
24	48	24	48
Skin reaction b)			
0	0 c)	0	5	5
1	0	0	0	0
2	0	0	0	0
3	5	5	0	0
Mean response			
3.0	3.0	0.0	0.0
Sensitization rate (%)			
100	100	0	0

DNCB: 2,4-dinitrochlorobenzene
a): Time after challenge
b): 0; no visible change, 1; discrete or patchy erythema, 2; moderate and confluent erythema, 4; intense erythema and swelling
c): Number of animals

Test condition: (1) Animals:
-At the first induction
Guinea pig (Std: Hartley), male, 5 weeks old, obtained from Japan SLC company
They were put in quarantine for 5 days beforehand.
Body weight; 331-384 g

(2) Number of animals/group:

Group	Test substance	Control for test substance
First induction substance (intracutaneous)		
	2-propen-1-ol (1 w/v%)	Water for injection
Second induction substance (occlusive epicutaneous)		
	2-propen-1-ol (2.5 w/v%)	Water for injection
Challenge substance (occlusive epicutaneous)		
(	2-propen-1-ol undiluted)	2-propen-1-ol (undiluted)
No. of animals		
	20	10

Group	Test substance	Control for test substance
First induction substance (intracutaneous)		
	DNCB (0.05 w/v%)	Olive oil

Second induction substance (occlusive epicutaneous)		
	DNCB (0.05 w/v%)	Olive oil
Challenge substance (occlusive epicutaneous)		
	DNCB (0.05 w/v%)	DNCB (0.05 w/v%)
No. of animals		
	5	5

(3) Evaluation:
According to Magnusson and Kligman method
0; no visible change
1; discrete or patchy erythema
2; moderate and confluent erythema
4; intense erythema and swelling

(4) Other
Mortality and clinical signs, body weight were checked.

Reliability: (1) valid without restriction
OECD TG study

Flag: Critical study for SIDS endpoint

05-DEC-2005 (7)

5.4 Repeated Dose Toxicity

Type: Sub-chronic
Species: rat **Sex:** male
Strain: Long-Evans
Route of administration: inhalation
Exposure period: 12 weeks
Frequency of treatment: 7 hr/day, 5 days/week
Doses: 0 (air), 1, 5, 20 ppm; 0 (air), 40, 60 ppm ; 0 (air), 100, 150 ppm
Control Group: yes, concurrent vehicle
NOAEL: 12 mg/m³
LOAEL: 47 mg/m³

Year: 1958
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS no. 107-18-6), supplied by Shell Chemical Company, purity 98.5%, impurities diallyl ether, water

Method: ANIMALS AND TREATMENTS
Groups of male Long-Evans rats (10/treatment level) were exposed to 2-propen-1-ol in three separate studies using the following exposure concentrations: 0, 1, 5 or 20 ppm; 0, 40 or 60 ppm; and 0, 100 or 150 ppm. Exposures lasted 7 hr/d, 5 d/wk for a total of 60 exposures (12 wk).

The animals were exposed in stainless steel chambers (200 L capacity). Airflow within the chamber was 10.9-21.1 L/min (3-6 air changes per hour), and the temperature in the exposure room was 20-25 degrees C.

The test atmosphere was generated by passing liquid 2-propen-1-ol via a syringe pump into an evaporation chamber through which air flowed at 8.6 to 12.9 l/min, depending on

the desired exposure concentration. The atmosphere within the chamber was allowed to equilibrate to 95-99% of the desired concentration before introduction of the animals. The nominal concentration in the chamber was calculated according to Jacobs (1949) The Analytical Chemistry of Industrial Poisons, Hazards and Solvents, 2nd edition, Interscience Publishers Inc., NY.

Clinical observations: daily
Body weights: weekly
Diet: no details
Water: not specified

NECROPSY AND HISTOPATHOLOGY
At the end of the experimental period, survivors were weighed, decapitated under ether anesthesia and subject to a post-mortem examination. Livers, kidneys and lungs from all animals were weighed and preserved (10% formalin) along with samples of thyroid, heart, thymus, pancreas, spleen, adrenal gland, testis, bladder and brain collected from alternate animals (i.e. 5/10 per treatment level). All preserved tissues were subject to microscopic evaluation.

STATISTICAL METHODS
Relative organ weights and percentage body weight gains were analyzed using Student's T-test.

Remark: Based on a conversion factor of 1 ppm = 2.37 mg/m3, (Patty's Toxicology (2001) 5th edition, p463), the following exposure concentrations can be derived:

ppm	mg/m3
1	2.4
2	4.7
5	12
20	47
40	95
60	142
100	237
150	355

Result: The achieved concentration of 2-propen-1-ol within the exposure chambers for the higher exposure conditions was:

40.7	+/- 3.2 ppm (24)
61.1	+/- 2.4 ppm (24)
103.2	+/- 8.7 ppm (22)
166.7	+/- 17.7 ppm (14)

Values given as mean +/-SD, number of determinations in parenthesis.

MORTALITY AND CLINCIAL SIGNS
Four rats from the 150 ppm group died during the first exposure, 2 were dead by the following morning and 2 died during the second exposure. The remaining 2 rats from the 150 ppm group died by the 10th exposure (end of week 2). There were 6 deaths in animals exposed to 100 ppm (time period inadequately characterized), and 1 death following 4 exposures to 60 ppm 2-propen-1-ol.

Clinical signs in the 150 ppm group included gasping, severe depression, nasal discharge, eye irritation and corneal opacity. Similar but less intense clinical signs were present in animals exposed to 40-100 ppm. No clinical signs were present in animals exposed to 20 ppm and below.

BODY WEIGHT
Mean percentage body weight gain was statistically significantly lower in animals exposed to 20 ppm or above:

0 ppm	134%
1 ppm	133%
5 ppm	126%
20 ppm	110% (P<0.05)

0 ppm	128%
40 ppm	90% (P<0.05)
60 ppm	75% (P<0.05)

0 ppm	135%
100 ppm	75% (P<0.05)
150 ppm	(no survivors at 15 wk)

RELATIVE ORGAN WEIGHTS
Relative kidney weight (g/100 g bw) was increased 8-10% in animals exposed to 40 ppm or 60 ppm 2-propen-1-ol vapor for 12 wk:

0 ppm	0.724
1 ppm	0.706
5 ppm	0.765
20 ppm	0.715

0 ppm	0.582
40 ppm	0.629
60 ppm	0.643 (P<0.05)

Relative lung weight (g/100 g bw) was increased after exposure to 40 ppm 2-propen-1-ol vapor for 12 wk:

0 ppm	0.410
40 ppm	0.435 (p<0.05)
60 ppm	0.531 (P<0.05)

(No data given for lower exposures)

Relative liver weights for treated animals were indistinguishable from those of the controls.

NECROPSY, HISTOPATHOLOGY
Livers from rats exposed to 150 ppm 2-propen-1-ol appeared hemorrhagic and the lungs pale and spotted. The kidneys appeared normal. The only microscopic observation was slight congestion of the lungs and liver (no further details). Lesions and microscopic findings at 100, 60 arid 40 ppm were described as similar but less intense to those reported at 150 ppm.

There were no unusual gross or microscopic findings at 20 ppm or below.

DERIVATION OF NOAEL
Although only limited data are available from this study, increases in relative kidney and lung weight are consistent with a LOAEL of 40 ppm. No lung data are available for animals exposed to lower concentrations, however relative kidney weights were unaffected indicating a NOAEL of 20 ppm. The decrease in bw gain was statistically significantly observed at 20 ppm. At 20 ppm and below, the rats behaved normally, and showed no gross or microscopic lesions.

Conclusion: From a sub-chronic inhalation study in rats exposed to 2-propen-1-ol over 12 weeks, the NOAEL and LOAEL were found to be 12 mg/m3 (5 ppm) and 47 mg/m3 (20 ppm) respectively, based on retardation of body weight gain and 47 mg/m3 (20 ppm) and 95 mg/m3 (40 ppm) in terms of liver and lung histopathology, respectively.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

05-DEC-2005 (39)

Type: Sub-chronic
Species: rat **Sex:** male/female
Strain: Long-Evans
Route of administration: drinking water
Exposure period: 13 weeks
Frequency of treatment: continuous
Doses: 0 (water), 1, 5, 50, 100 or 250 ppm; 0, 500 or 1000 ppm
Control Group: yes, concurrent vehicle
NOAEL: 11.6 - 13.2 mg/kg bw
LOAEL: 25.5 - 34 mg/kg bw
NOAEL (male: more sensitive sex) : 11.6 mg/kg bw

Year: 1958
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS no. 107-18-6), supplied by Shell Chemical Company, purity 98.5%, impurities diallyl ether, water

Method: ANIMALS AND TREATMENTS
Groups of male and female Long-Evans rats (10/sex/treatment level) were exposed to 2-propen-1-ol in drinking water in two separate studies using the following exposure concentrations: 0, 1, 5, 50, 100 or 250 ppm; 0, 500 or 1000 ppm. Treatment was continuous and lasted for 13 wk.

Stock solutions were prepared weekly in brown glass bottles with plastic stoppers.

Clinical observations: daily
Body weight: weekly
Water consumption: weekly
Diet: no details

NECROPSY AND HISTOPATHOLOGY
At the end of the experimental period, survivors were

weighed, decapitated under ether anesthesia and subject to a post-mortem examination. Livers and kidneys from all animals were weighed and preserved (10% formalin). Samples of duodenum, thyroid, heart, thymus, pancreas, spleen, adrenal gland, testis, ovary, bladder and brain collected from alternate animals (i.e. 5/10 per treatment level). All preserved tissues were subject to microscopic evaluation.

STATISTICAL METHODS
Relative organ weights and percentage body weight gains were analyzed using Student's T-test.

Result: WATER CONSUMPTION AND INTAKE OF TEST SUBSTANCE
Water consumption decreased in a treatment-related manner in both sexes:

	--ml/rat/day--	
	males	females
0 ppm	145	177
1 ppm	131	170
5 ppm	124	188
50 ppm	118	117
100 ppm	116	133
250 ppm	102	135
0 ppm	151	172
500 ppm	82	87
1000 ppm	72	67

Calculated intake of 2-propen-1-ol was as follows:

	--mg/kg bw/d--	
	males	females
0 ppm		
1 ppm	0.13	0.17
5 ppm	0.62	0.94
50 ppm	5.9	7.3
100 ppm	11.6	13.2
250 ppm	25.5	34.0
0 ppm		
500 ppm	41.0	43.7
1000 ppm	72.0	67.4

CLINICAL SIGNS
Occasional crusting or swelling of the eyelids was the only clinical sign observed (no detail of any dose/severity relationship).

Two males from the 250 ppm treatment group lost weight; one was sacrificed after 5 wk, the other died during week 10. Pulmonary edema was observed in one animal at post-mortem with necrosis of the intestinal mucosa in the other; liver and kidney were normal.

BODY WEIGHT
Mean percentage body weight gain was statistically

significantly lower in animals exposed to 500 ppm 2-propen-1-ol in drinking water and above:

	males	females
0 ppm	76	51
1 ppm	79	56
5 ppm	98	51
50 ppm	108	60
100 ppm	92	41
250 ppm	106	42
0 ppm	229	139
500 ppm	99*	70*
1000 ppm	51*	43*

* = P<0.05

RELATIVE ORGAN WEIGHTS
Relative kidney weight (g/100 g bw) was increased in a dose related manner following 13 wk treatment with 2-propen-1-ol in drinking water:

	males	females
0 ppm	0.817	0.776
1 ppm	0.792	0.759
5 ppm	0.816	0.766
50 ppm	0.842	0.767
100 ppm	0.829	0.794
250 ppm	0.826*	0.882*
0 ppm	0.610	0.612
500 ppm	0.760*	0.778*
1000 ppm	0.815*	0.834*

*= P<0.05

Relative liver weight (g/100 g bw) was increased 11-22% in males given 2-propen-1-ol in drinking water at 250 ppm or above for 13 wk; less consistent increases present in females:

	males	females
0 ppm	3.02	3.59
1 ppm	2.91	3.60
5 ppm	3.01	3.62
50 ppm	3.03	3.12
100 ppm	3.32+	3.30
250 ppm	3.35*	3.46
0 ppm	3.03	3.26
500 ppm	3.50	3.66
1000 ppm	3.69*	3.41

* = P<0.05

(+ Note: value reported as 0.332; presumed type-setting error)

NECROPSY OBSERVATIONS
Few abnormalities were noted at necropsy at week 13:
- perirenal fat was decreased in the 500 ppm group and absent at 1000 ppm
- the livers from two high dose females were pale with a soft, spongy yellowish appearance with well organized areas of necrosis with regeneration observed upon microscopic examination (appearance and distribution considered consistent with infarction by the study authors)
- perivascular cuffing present in brain from one high dose female
- the authors state there were no other findings of interest

DERIVATION OF NOAEL
Although only limited data are available from this study, statistically significant increases in relative kidney weight in rats of both sexes given 250 ppm or above 2-propen-1-ol in drinking water, with a concurrent decrease in body weight gain (significant at 500 ppm and 1000 ppm), appears indicative of toxicity. Relative liver weights were also increased in male rats given 250 ppm and above, although this change was no always statistically significant. These observations point to a NOAEL of 100 ppm (equivalent to 11.6 and 13.2 mg/kg bw/d in males and females, respectively).

Conclusion: Under the conditions of this study, a sub-chronic oral NOAEL of 100 ppm 2-propen-1-ol in drinking water (equivalent to 11.6 and 13.2 mg/kg bw/d) was obtained for the rat, based upon treatment related increases in relative kidney and liver weights at higher exposures.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

09-MAY-2006 (39)

Type: Sub-chronic
Species: rat **Sex:** male/female
Strain: Wistar
Route of administration: drinking water
Exposure period: 15 weeks
Frequency of treatment: continuous
Post exposure period: none
Doses: 0, 50, 100, 200 or 800 ppm
Control Group: yes, concurrent vehicle
NOAEL: 4.8 - 6.2 mg/kg bw
NOAEL (male: more sensitive sex) : 4.8 mg/kg bw

Year: 1978
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), supplied by Bush Boake Allen Ltd, London, Purity 99%

Method: ANIMALS AND TREATMENTS
Groups of Wistar rats (15/sex/treatment level) were exposed to 2-propen-1-ol in the drinking water at 0 (control), 50, 100, 200 or 800 ppm for 15 weeks. Additional groups of 5

rats/sex were given 0, 200 or 800 ppm 2-propen-1-ol for 2 wk or 6 wk. (Comment: group sizes inconsistent with OECD TG 408)
Animal supplier: commercial supplier (not specified), SPF colony.
Housing: 5/cage; 20+/-1 degree C; 50-60% relative humidity.
Body weights: recorded pre-treatment and on days 1,6,8,13,15 or 20. Food and water intake: measured over the 24-h period preceding each weighing.
Diet: Spratts Laboratory Diet No 1, ad libitum
Water: not specified

RENAL FUNCTION AND URINE ANALYSIS
Renal function was investigated during wk 2 or wk 5 (n=5/sex) and in wk 15 (n=12/sex). Concentrating ability (specific gravity, volume) was determined by measuring the urine volume produced during 0-6 hr of water deprivation; at wk 5 and 15, the concentration test was extended to include samples collected over 4 hr following a 16 hr period without water. Diluting ability was then assessed over 2 hr following a water load of 25 ml/kg bw; these samples were also assessed for specific gravity, appearance and microscopic constituents (cells) as well as a semi-quantitative evaluation of glucose, ketones, bile salts and blood. At wk 5 and 15, and studies performed after 2 wk treatment, concentrating ability was determined over 2 hr following a 6 hr deprivation period.

HEMATOLOGY
Blood collected at necropsy was assessed for hemoglobin content, packed cell volume and red cell and total leukocyte counts. A differential leukocyte count and a reticulocyte count was performed on samples from control and high dose animals. (Comment: several omissions compared with OECD TG 408.)

CLINICAL CHEMISTRY
Serum was analyzed for urea, glucose, total protein and albumin, together with ASAT, ALAT and lactic dehydrogenase activity. (Comment: several omissions compared with OECD TG 408.)

NECROPSY AND HISTOPATHOLOGY
At the end of the appropriate treatment period, animals were killed by exsanguination under barbiturate anesthesia following an overnight fast. Animals were subject to a post-mortem examination and any external or internal macroscopic abnormalities noted. A representative range of tissues was weighed, sampled and preserved for histopathological evaluation. (Comment: range of tissues comparable to OECD TG 408, but with some exclusions, notably aorta, trachea/lungs, skin, eye, peripheral nerve, bone marrow).

STATISTICAL METHODS
Mean body weights, food and water intake and organ weights were analyzed using Student's t-test. Renal function data were analyzed by the method of White (1952; Biometrics, 8, 33).

Remark: The evaluation of US EPA, IRIS was based on this study.

Result:

INTAKE OF TEST SUBSTANCE
The calculated mean intake of 2-propen-1-ol over the course of the study (based on body weight and water intake data) was:
Males: 0, 4.8, 8.3, 14.0, 48.2 mg/kg bw/d
Females: 0, 6.2, 6.9, 17.1 and 58.4 mg/kg bw/d

BODY WEIGHT, FOOD INTAKE AND WATER CONSUMPTION
Body weight was significantly decreased in males given 100 or 200 ppm 2-propen-1-ol from wk 2 of treatment, and from males and females given 800 ppm following a single day's treatment. Terminal body weights (g) at week 15 were:

- Males	
Control:	472
50 ppm:	453
100 ppm:	449 (ns)
200 ppm:	420 (P<0.01)
800 ppm:	270 (P<0.001)

-Females	
Control:	253
50 ppm:	260
100 ppm:	260
200 ppm:	257
800 ppm:	205 (P<0.001)

Food intake was significantly decreased in male rats from the 200 (-11%; P<0.01) and 800 ppm (-32%; P,0.001) groups, and in high dose females (-18%; P,0.001).

There was a statistically significant decrease in water intake in all treated groups:

-Males	
Control:	27.7
50 ppm:	24.2 (P<0.01)
100 ppm:	19.4 (P<0.01)
200 ppm:	15.5 (P<0.001)
800 ppm:	10.0 (P<0. 001)

-Females	
Control:	26.5
50 ppm:	22.0 (P<0.05)
100 ppm:	17.2 (P<0.001)
200 ppm:	14.4 (P<0.001)
800 ppm:	9.8 (P<0.001)

HEMATOLOGY AND CLINICAL CHEMISTRY
No abnormalities were reported. (Comment: data not available for evaluation.)

RENAL FUNCTION AND URINE ANALYSIS
There was a statistically significant decrease (-50% to -75%) in excretion of cells in male rats following 2, 5 and 15 wk treatment with 800 ppm 2-propen-1-ol.

Urine concentrating ability (ml/6 hr) was significantly impaired in a time- and dose dependent manner in males:

-Wk 2	
Control:	1.9
200 ppm:	2.2
800 ppm:	0.6 (P<0.05)

-Wk 5	
Control:	3.8
200 ppm:	0.9 (p<0.01)
800 ppm:	0.9 (p<0.01)

-Wk 15	
Control:	4.5
50 ppm:	2.7
100 ppm:	2.4 (P<0.01)
200 ppm:	1.8 (P<0.001)
800 ppm:	0.8 (P<0.05)

Essentially similar, but statistically non-significant, changes were present in females.
Urine concentrating ability over 16-20 hr was unaffected by treatment with 2-propen-1-ol.

Urine volume (ml) was statistically significantly decreased during the dilution test at wk 2, 5 and 15 for animals (both sexes) treated with 2-propen-1-ol in drinking water at 200 ppm or above. Representative data for males:

-Wk 2	
Control:	4.3
200 ppm:	0.7 (P<0.01)
800 ppm:	0.3 (P<0.01)

-Wk 5	
Control:	1.3
200 ppm:	2.2 (P<0.05)
800 ppm:	0.9 (P<0.05)

-Wk 15	
Control:	8.4
50 ppm:	7.5
100 ppm:	5.9
200 ppm:	3.6 (ns)
800 ppm:	0.5 (P<0.001)

Specific gravity was increased (approx. 1-4%) during the dilution test at wk 2, 5 and 15 for males and females given 2-propen-1-ol in drinking water at 200 ppm or above. These changes were generally statistically significant.

POST MORTEM EXAMINATION
No gross abnormalities were present in any sex/treatment group.

Absolute organ weights were generally decreased in males, and to a lesser extent in females, in a time- and treatment related manner after ingestion of 100 ppm 2-propen-1-ol or above. Although statistically significant (especially in high dose males), these decrements were consistent with the lower body weights recorded in treated animals. The exception was absolute kidney weight for females, which was statistically significantly increased (11-13%; P<0.001) at week 15 in the 100, 200 and 800 ppm treatment groups:

	--abs. kidney wt-	
	males	females
Control	2.42	1.48
50	2.43	1.48
100	2.48	1.65***
200	3.07	1.67***
800	2.10	1.64***

*;P<0.05, **;P<0.01, ***;P<0.001

Relative organ weights (g/100 g bwt) were generally increased to a statistically significant extent in high dose animals of both sexes at study termination. Relative kidney weights and relative stomach weights, in contrast, were increased in a dose-dependent manner in females at week 2 and in both sexes following 6 or 15 weeks of treatment. Results at 15 wk summarized below:

	-rel. kidney wt-		-rel. stomach wt-	
	males	females	males	females
Control	0.56	0.59	0.37	0.50
50	0.55	0.60	0.39	0.50
100	0.58	0.67***	0.42***	0.54*
200	0.59**	0.70***	0.41**	0.52
800	0.73***	0.83***	0.50***	0.66***

*;P<0.05, **;P<0.01, ***;P<0.001

HISTOPATHOLOGICAL EVALUATION
Minor changes were present in the microscopic appearance of the liver (occasional vacuolated cells, scattered individual cell necrosis with lymphocyte infiltration), kidneys (occasional vacuolated tubular cells) and spleen (mild degree of peribronchial lymphocyte infiltration) however these occurred at a similar incidence in control and treated

animals. (No microscopic changes were reported in stomach.)

DERIVATION OF NOAEL
The majority of findings from this study, in particular lower body weights, alterations in organ weights and changes in renal function, appear secondary to a reduction in water intake that was particularly pronounced in high dose animals. This is presumed to reflect poor palatability of the dosing solutions. Against this background, there was a more generalized increase in absolute kidney weight (females), relative kidney weight (both sexes) and relative stomach weight (both sexes) in the intermediate and high dose groups after 15 wk of treatment. These results revealed that 2-propen-1-ol administration in the drinking water at or above a level of 100 ppm (a mean 2-propen-1-ol intake of 8.3 mg/kg bw/day in males and of 6.9 mg/kg bw/day in females) has adverse effects on kidney tissues in the rats. Therefore, the NOAEL established in this study was 50 ppm of the drinking water (a level equivalent to a mean intake of 4.8 mg/kg bw/day in male rats and 6.2 mg/kg bw/day in female rats).

Conclusion: The NOAEL was 50 ppm of the drinking water (a level equivalent to a mean intake of 4.8 mg/kg bw/day in male rats and 6.2 mg/kg bw/day in female rats).

Reliability: (1) valid without restriction
Well reported published study

Flag: Critical study for SIDS endpoint

09-MAY-2006 (28)

Type: Sub-chronic
Species: rat **Sex:** male/female
Strain: Fischer 344
Route of administration: gavage
Exposure period: 13 weeks
Doses: 0, 1.5, 3, 6, 12 or 25 mg/kg bw/d

Year: 1995
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Information available from the NTP website indicated that the sub-chronic toxicity of 2-propen-1-ol has been investigated in F-344 rats following gavage administration (NTP study No. C93009).

Reliability: (4) not assignable

06-JAN-2006 (109)

Type: Sub-chronic
Species: mouse **Sex:** male/female
Strain: B6C3F1
Route of administration: gavage
Exposure period: 13 weeks
Frequency of treatment: 0, 3, 6, 12, 25 or 50 mg/kg bw/day

Year: 1995
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Information available from the NTP website indicated that the sub-chronic toxicity of 2-propen-1-ol has been investigated in B6C3F1 mice following gavage administration

(NTP study No. C93009).
Reliability: (4) not assignable
06-JAN-2006 (109)

Type: Sub-acute
Species: other: monkey, rabbit, rat **Sex:**
Route of administration: inhalation
Exposure period: 7 hours daily
Doses: 50, 200, 1000 ppm

Year: 1932
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Method: Six rabbits, one monkey and sixteen white rats were subjected to the action of known concentrations, in such quantities of the vapor as 50, 200 and 1000 ppm of air. The exposure period was seven hours daily , although some animals did not survive that long.

Result: Following exposure to 1000 ppm of 2-propen-1-ol in air, the one rhesus
monkey, long under preliminary observation, died within four hours. Prior to death there occurred profound vomiting and diarrhea, together with every indication of severe pain.
Two rabbits were subjected to the same concentrations. One died after three and a half hours' exposure, the other after four hours and fifteen minutes. During exposure, breathing was labored and noisy. Fluid dripped from the nose and mouth.
Six rats were exposed to this concentration of 1000 ppm and were dead within three hours. The postmortem observation in a lot were the same throughout and closely resembled those found in the rabbits and the monkey.
In another group of three rabbits and four rats, the concentration was reduced to 200 ppm. After one hour's exposure there existed obvious discomfort and labored, noisy breathing, with discharge of fluid from the nose and mouth. One animal survived three days' exposure (seven hours each), dying in convulsions; another survived six day's exposure, and the third survived eighteen days' exposure. During this period the labored, noisy breathing persisted, together with marked nasal and oral discharge of secretion.
In a third group of two rabbits and five rats, the concentration produced was 50 ppm. The first rabbit died on the fourteenth day of exposure; the second survived twenty-eight days' exposure and then killed for examination. The rats in this series, with one exception, died as the result of the action of 2-propen-1-ol, the exposure period averaging about thirty days.
In general, it appears that 2-propen-1-ol is highly irritating chemical, leading in life to pulmonary edema, hemorrhage, gastro-enteritis, profound vomiting, distension of the gastro-intestinal tract, diarrhea, nephritis and hematoma.
A suitable number of controls did not present any of the abnormalities observed before death and at autopsy.

Reliability: (4) not assignable
05-DEC-2005 (94)

Species: other: rat, guinea pig, rabbit, **Sex:**

dog
Route of administration: inhalation
Exposure period: 7 hour exposure
Frequency of treatment: five days/week for five weeks or six months
Doses: 2 or 7 ppm
Control Group: other: air
NOAEL: 2 ppm

Year: 1959
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), purity 99.5 mole%

Method: A water sealed chamber was used for exposure to 7 ppm (equivalent to 16 mg/m3) 2-propen-1-ol in air. Exposures to 2 ppm (equivalent to 5 mg/m3) were conducted in a vault-like chamber.

For the 7 ppm (16 mg/m3) exposure, male and female rats (5 of each sex), male guinea pigs (4 animals) and a female rabbit were exposed 28 times for seven hours each time over a 35 day period.

For the 2 ppm (5 mg/m3) exposure, male and female rats, guinea pigs, rabbits and dogs received 127 to 134 seven hour exposures in 180-194 days.

Result: 7 ppm (16 mg/m3) exposure: All animals showed no evidence of ill-effect as judged by growth, behaviour, mortality, gross appearance and final average body and organ weights. However, there was degeneration observed in the kidneys and livers of almost all animals upon microscopic examination. This was characterised in the liver by dilation of the sinusoids, cloudy swelling and focal necrosis. In the kidney the changes were similar to those commonly seen in the glomerulonephritis, necrosis of the epithelium of the convoluted tubules and proliferation of the interstitial tissue. These changes appeared to be mild in intensity and reversible.

2 ppm (5 mg/m3) exposure: All animals showed no evidence of ill effect as judged by growth, behaviour, mortality and final average body and organ weights. The terminal values reported for BUN and NPN were in several cases below those found for the control animals in this experiment. They were, however, within the normal limits for control animals from the same stock used in previous studies. In the absence of pathology in the liver and kidneys which might be expected if differences in NPN and BUN were due to exposure to 2-propen-1-ol and since the values lie within the normal limits the apparent changes were not considered to be significant or a result of exposure to the chemical. Gross and microscopic examination of the tissues revealed no changes. The average hematological values were within the normal limits for the laboratory. Examination of the rats held 60 days also showed no effect.

Reliability: (4) not assignable
Insufficient detail in published study

09-MAY-2006 (156)

Species: rat **Sex:**
Route of administration: oral feed
Exposure period: 30 days
Doses: 4 or 9.7 mg/kg bw/day

NOAEL: 4 mg/kg bw

Year: 1951
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS NO. 107-18-6)

Reliability: (4) not assignable
Insufficient detail in published study
05-DEC-2005 (151)

Species: rabbit **Sex:**
Route of administration: drinking water
Exposure period: 8 months
Doses: 0.005, 0.05, 2.5 mg/kg bw/day
NOAEL: .05 mg/kg bw

Year: 1939
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS NO. 107-18-6)

Remark: 2.5 mg/kg produced liver hemorrhage and necrosis dystrophy of the epithelium of kidneys tubules, and diffuse reticular-endothelial hyperplasia in the gulp of the spleen suggesting the rabbit is more sensitive to repeated 2-propen-1-ol exposures than the rat.
Reliability: (4) not assignable
Data reported in the secondary literature
05-DEC-2005 (4)

5.5 Genetic Toxicity 'in Vitro'

Type: Bacterial reverse mutation assay
System of testing: Salmonella typhimurium TA1535, TA1537, TA1538, TA98 and TA100
Concentration: 10-500 ug/plate
Cytotoxic Concentration: 500 ug/plate
Metabolic activation: with and without
Result: positive

Year: 1980
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), purity >95%, supplied by Aldrich Chemical Company

Method: Tester strains TA1535, TA1537, TA1538, TA98 and TA100 were used with or without S9 from Arochlor 1254 treated rats or hamsters. [Comment: Hamster S9 was used whenever the test compound was not mutagenic with rat S9.]

Testing was conducted using plate incorporation or liquid pre-incubation (45 min at 37 degrees C) methodology, with independent repeat.

Sodium azide, 9-aminoacridine, 2-nitrofluorene and 2-aminoanthracene were used as positive controls (all tester strains, with or without rat and hamster S9).

A compound was considered mutagenic if the number of revertants was twice the background, and a dose-response

curve was demonstrable.

Result: 2-Propen-1-ol was mutagenic only in TA1535 in the liquid pre-incubation test, with distilled water as solvent in the presence of hamster S9.

Representative results from one repeat (other not reported):

Dose (ug)	Revertants/plate -S9	+S9
0 (water)	16	22
10	14	38
25	21	37
50	15	55+
75	10	62+
100	17	62+
125	15	64+
115	5	81+
200	10	71+
300	10	55+
500	T	21

+ value at least twice control mean
T toxic

Cytotoxicity was apparent at 500 ug in the plate incorporation test but not at this concentration in the liquid preincubation test.

A satisfactory response was obtained with the positive control substances.

Conclusion: Under the conditions of the test, a positive response was obtained with TA1535 in a liquid preincubation assay (negative with plate incorporation) in the presence of hamster S9 (negative in the presence of rat S9, negative in the absence of S9). No mutagenic response was seen with TA1537,
TA1538, TA98 or TA100.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

05-DEC-2005 (84)

Type: Bacterial reverse mutation assay
System of testing: Salmonella typhimurium TA100
Concentration: (-S9) up to 0.55 umol/2mL incubation volume (equivalent to approx. 2-32 ug/2 mL)
(+S9) up to 0.7 umol/2mL incubation volume (equivalent to approx. 2-41 ug/2 mL)
Cytotoxic Concentration: (-S9) <50% survival at 0.15 umol/2 mL incubation volume (approx. 9 ug/2 mL)
(+S9) <50% survival at 0.55 umol/2 mL incubation volume (approx. 32 ug/2 mL)
Metabolic activation: with and without
Result: positive

Year: 1984
GLP: no data
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity >99.9% (acrolein <1ppm), suuplied by Merck, Darmstadt, Germany

Method: Tester strain TA100 was used in a liquid pre-incubation assay (tightly closed screw-capped vials, 37 degrees C, shaking, 90 min) in the absence and presence of S9. An aliquot from each incubation was diluted and plated onto histidine-containing medium. Mutation frequencies were determined as the number of revertants per umol allyl alcohol. The assay was conducted with an independent repeat.

The concentration range used is not stated. Graphical data indicate that 5 concentrations up to 0.7 umol per 2 ml liquid incubation were employed. Based upon a molecular weight of 58.08, this equates to approx. 41 ug.

Sodium azide was used as positive control in the absence of S9, and 2-aminoacridine in the presence of S9.

Remark: The authors note that <1 ppm acrolein was present in sample of 2-propen-1-ol used in these studies. Concurrent data on acrolein, also included in this publication, lead them to conclude that acrolein (at this concentration) would not have contributed to the mutagenic response observed.

The authors suggest that conversion of 2-propen-1-oll to acrolein by bacterial alcohol dehydrogenase(s) may account for the strong positive response obtained.

Result: Graphical data demonstrate a clear inverse correlation between survival and induction of revertants in TA100 in the absence of S9.

In the absence of S9, there was a linear increase in the number of revertants per plate over a concentration range of approx. 0.05-0.3 umol/2 mL (equivalent to approx. 3-17 ug).

Survival was also decreased in the presence of S9 (weak mutagenic response), however this effect was considerably less profound than was seen in the absence of S9.

A mutation frequency of 750 revertants/umol (approx. 13 revertants/ug) was obtained in the absence of S9, and 145 revertants/umol (approx. 2 revertants/ug) in its presence.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

05-DEC-2005 (86)

Type: Bacterial reverse mutation assay
System of testing: Salmonella typhimurium TA100, TA1535, TA97, TA98
Concentration: 0.3-166 ug/plate or 3-333 ug/plate
Cytotoxic Concentration: 333 ug/plate
Metabolic activation: with and without
Result: negative

Method: other: US NTP standard protocol
Year: 1995
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Remark: Only limited information is available for this study which was conducted in the absence or presence of 10% or 30% rat

or hamster S9 using a preincubation protocol.

It was run with an independent repeat.

DMSO was the vehicle control.
Positive controls for each strain were as follows:
-S9
TA1535, TA100: sodium azide (1.0 ug/plate)
TA97: 9-aminoacridine (50 ug/plate)
TA98: 4-nitroo-phenylenediamine (5 ug/plate)
+S9
TA100, TA1535, TA97, TA98: 2-aminoanthracene (1ug/plate)

Result:

STRAIN: TA100

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	
	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	108	2.4	125	2.6	141	2.6	108	3.2	140	2.6	139	2
3	110	4.4	127	3.3	138	2.9	109	2.7	138	3.2	135	3.2
10	119	1.2	126	3.6	138	3.8	111	3.4	136	3.2	130	6.2
33	112	0.7	128	3.5	143	2.6	109	1.3	141	1.5	133	2.6
100	79s	2.7	116	2.3	124	2.9	83	6.5	135	3	121	2.7
333	T	0	107s	3.1	115s	3.5	78s	5.6	94s	4.1	70s	3.8
+Con	891	5.7	890	12.5	961	36.1	783	9.3	1009	7.5	766	17

STRAIN: TA1535

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	
	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	15	1.2	14	1.2	10	1.8	16	2	12	1.5	17	2.4
3	14	1.2	15	0.9	9	1.5	14	2	13	0.3	19	1.2
10	16	1.7	13	1.5	12	2.1	17	1.3	11	2.1	16	2
33	15	2.3	13	0.3	10	2	14	1.7	11	1.5	15	0.7
100	14	2.1	11	2.1	11	1.2	15	2	11	2.9	14	1.5
333	T	0	T	0	6s	2.3	7s	1.2	5s	1.2	5s	1.8
+Con	335	3.8	535	7.2	346	6.8	351	4.8	367	4.3	372	4.6

STRAIN: TA97

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	
	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	98	2	93	2.5	125	2.2	120	4	99	1.8	112	2.3
3	103	3.8	94	2	116	3.6	121	2.6	106	2.6	112	2.5
10	103	3.5	95	1.8	96	3.2	116	3.2	108	3.8	111	2
33	102	2.6	98	1.8	127	3.5	118	4.7	102	3.5	102	5
100	95	2.7	92	2.3	125	2.6	120	2.6	99	3.2	108	2

333	T	0	T	0	95s	3.5	77	2.6	84s	4.4	108	3.2
+Con	508	11.9	625	26.1	537	6.4	604	15.3	938	5.5	572	20.4

STRAIN: TA98

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	
	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	31	2.6	18	0.9	43	2.4	38	1.7	46	1.5	41	2.1
3	31	2.3	18	1.3	40	4.4	41	0.6	46	3.5	40	2.6
10	27	1.9	17	2.3	45	2.3	35	1.2	47	2.4	42	2.6
33	29	0.3	20	3.2	44	1.9	32	3.5	45	3.3	39	2.3
100	17s	1.5	16	1.7	37	2	26	2.6	46	3.8	25	2.6
333	T	0	4s	1.2	16s	3.1	T	0	16s	1.8	12s	2.3
+Con	252	6.8	429	5.5	931	3.5	577	6.1	980	3.8	954	15.2

Abbreviations:

-Act: No activation
Neg: Negative
Mn: Mean
SE: Standard Error
Vcon: Vehicle control
+Con: Positive control
RLI: Induced male Sprague dawley rat liver S9
HLI: Induced male Syrian hamster liver S9
s = slight toxicity; p = precipitate; x = slight toxicity and precipitate; T = toxic; c = contamination

STRAIN: TA100

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	
	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	169	6.7	103	2.4	113	1.5	181	1.2	96	5.9	177	7.7
0.3	175	8.5										
1	183	2.7	100	2.4			188	5.9			183	10
3	166	2	101	4.5	101	7.3	181	8.1	111	11.7	179	3.2
10	163	9.5	105	3.3	107	0.3	184	5.5	102	6.7	198	6.7
33	144	4.4	97	5.9	111	2.1	175	5.9	105	5.1	160	14.4
66			73	3.1								
100					100	5.8	153	2.3	108	8.1	155	4.3
166					49	9.8			48	8.2		
+Con	943	20.4	647	32.4	484	22.4	1096	52.9	619	1.8	954	6.5

STRAIN: TA1535

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	

	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	8	2.3	15	1.5	17	0.6	11	1.7	12	2.5	10	1.2
0.3	9	0.3										
1	8	1.9	14	2.3			14	2.3			10	2.4
3	9	1.9	15	1.5	13	2.3	12	2.2	11	1.7	12	2.6
10	9	1.3	13	2.2	8	0	8	0.6	10	2.3	8	0.9
33	10	2.8	10	1.2	8	0.7	9	1.5	12	2.3	8	0.6
66			10	2.7								
100					8	2.3	7	0.6	11	2.1	11	1
166					5	0.7			8	0.3		
+Con	629	17	812	29.4	123	6.5	230	5.6	125	24.7	468	7.6

STRAIN: TA97

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	
	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	115	9.7	170	4	174	9.8	175	12.2	179	9.2	170	2.6
0.3	129	8.4										
1	124	4.8	171	6.7			171	6.7			166	9.1
3	131	8.3	178	12.5	183	9.5	179	14.5	188	2.1	165	6
10	123	3.8	170	8	192	4.7	187	2.1	187	4.1	163	4.6
33	117	13.7	150	13	185	3.5	169	5.8	189	5.2	142	2.3
66			106	5.5								
100					159	3.2	151	20.2	167	9.3	155	10.5
166					83	9.2			105	12.7		
+Con	426	35.3	515	13.2	478	12.3	518	30.6	545	22.3	657	

219.8

STRAIN: TA98

Dose	- Act		- Act		10% RLI		30% RLI		10% HLI		30% HLI	
ug/pl	(Neg)		(Neg)		(Neg)		(Neg)		(Neg)		(Neg)	
	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE	Mn	SE
Vcon	19	2.3	20	7.5	18	1.7	25	3.2	20	3.2	23	3.3
0.3	15	0.9										
1	18	0.9	16	0.9			19	4.5			22	2.5
3	16	3.5	17	3	23	2.9	18	3.8	22	3.2	16	0.9
10	15	0.7	17	0.7	18	3.5	14	0.9	17	1.7	18	2.4
33	12	1.7	15	1.2	16	1	17	0.7	14	2.4	19	1
66			12	1.2								
100					10	1	10	0.9	10	0.7	17	1.5
166					12	2.3			9	0.6		
+Con	373	6.7	306	5.5	340	17.2	641	25	452	11.4	362	28.2

Abbreviations:

-Act: No activation
Neg: Negative
Mn: Mean
SE: Standard Error

Vcon: Vehicle control
+Con: Positive control
RLI: Induced male Sprague dawley rat liver S9
HLI: Induced male Syrian hamster liver S9
s = slight toxicity; p = precipitate; x = slight toxicity and precipitate; T = toxic; c = contamination

Conclusion: Under the conditions of the test, no mutagenic activity was detected in 4 strains of Salmonella typhimurium (including TA100 and TA1535) in the absence or presence of rat or hamster S9.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

06-JAN-2006 (109)

Type: Bacterial reverse mutation assay
System of testing: Salmonella typhimurium TA1535, TA1537, TA1538, TA98, TA100
Concentration: 0.025, 0.05, 0.10 ul/plate
Cytotoxic Concentration: >0.10 ul/plate
Metabolic activation: with and without
Result: negative

Year: 1981
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: analytical grade, Supplier: Fluka AG

Method: The ability of 2-propen-1-ol (0.05 ul) to induce reversion in Salmonella typhimurium tester strains TA1535, TA1537, TA1538, TA98, TA100 was investigated using a spot test in the absence or presence of S9 (SD rat, Arochlor 1254 induction). The authors state that the system used was suitable for testing volatile substances. Ethyl methansulfonate (5 ul/plate; TA1535), 9-aminoacridine (10 ug/plate; TA1537), 4-nitro-o-phenyldiamine (10 ug/plate; TA1538 and TA98), ethyl methansulfonate (1 ul/plate; TA100) were used as positive control substances in the absence of S9. 2-Aminoanthracene (1 ug/plate) was used as positive control substance for all tester strains in the presence of S9.

PLATE INCORPORATION ASSAY
Mutagenic activity was also investigated in TA1535, TA100 and TA98 using a plate incorporation assay and 0.025, 0.05, 0.1 ul 2-propen-1-ol/plate in the absence or presence of S9.

Comment: Concentrations in excess of 0.05 ul (= 50 nl; spot test) or 0.10 ul (= 100 nl; plate incorporation assay) were cytotoxic.

Result: The number of his+ revertants per plate was highly comparable in control and test cultures for all 5 tester strains both in the absence or presence of S9. A satisfactory response was obtained with the positive control substances.

PLATE INCORPORATION ASSAY
There was no increase in revertants in any of the 3 tester strains in the absence or presence of S9.

Conclusion: Under the conditions of the test, 2-propen-1-ol (highest

non-toxic concentration) was not mutagenic in a spot test (5 stains of Salmonella typhimurium including TA100 and TA1535) or a plate incorporation assay (3 tester strains).
Reliability: (2) valid with restrictions
Well reported published study
Flag: Critical study for SIDS endpoint
05-DEC-2005 (121)

Type: Bacterial forward mutation assay
System of testing: Streptomyces coelicolor, resistance to streptomycin
Concentration: 2-100 ul/plate
Cytotoxic Concentration: >100 ul/plate
Metabolic activation: without
Result: negative

Year: 1981
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: analytical grade, Supplier: Fluka AG

Method: Forward mutation of S. coelicolor to streptomycin resistance was investigated in a spot test (100 ul/test) or a plate incorporation assay (2-100 ul/plate).

The test medium was supplemented with 1.5 ug/ml streptomycin and approx. 2X10E7 spores (method: Carere et al. (1978) Chem Biol Interact 22, 297-308; Carere et al. (1987) Mut Res 57, 271)

Ethyl methansulfonate (2 ul/plate) was used as a positive control substance.
Result: 2-Propen-1-ol was ineffective at inducing mutants in both the spot test and the plate incorporation test.

An acceptable response was obtained with the positive control substance.
Conclusion: Under the conditions of the test, 2-propen-1-ol (100 ul) did not induce forward mutations in Streptomyces coelicolor in a spot test or a plate incorporation assay.
Reliability: (2) valid with restrictions
Well reported published study
05-DEC-2005 (121)

Type: other: fungal point mutation
System of testing: Aspergillus nidulans
Concentration: 10-40 ul/plate
Cytotoxic Concentration: >40 ul/plate
Metabolic activation: without
Result: negative

Year: 1981
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Purity: analytical grade, Supplier: Fluka AG

Method: The induction of point mutations in Aspergillus nidulans (haploid strain 35), as detected by resistance to 8-azaguanine, was investigated using a spot test (20 ul 2-propen-1-ol/test) or a plate incorporation assay (10, 20 or 40 ul 2-propen-1-ol/plate). (Method: Bignami et al.

(1980) Chem Biol Interact 30, 9)

Ethyl methansulfonate (1 ul/plate) was used as a positive control substance.

Result: 2-Propen-1-ol was ineffective at inducing mutations in both the spot test and the plate incorporation test.

An acceptable response was obtained with the positive control substance.

Conclusion: Under the conditions of the test, 2-propen-1-ol (100 ul) did not induce point mutations in Aspergillus nidulans in a spot test (20 ul 2-propen-1-ol/test) or a plate incorporation assay (up to 40 ul/plate).

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (121)

Type: Mammalian cell gene mutation assay
System of testing: V79 cells, 6-thioguanine resistance
Concentration: 1 or 2 uM (equivalent to 58 or 116 ng/ml)
Cytotoxic Concentration: >2 uM
Metabolic activation: without
Result: positive

Year: 1990
GLP: no data
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), Supplier: Aldrich Chemical Co., Milwaukee, WI

Method: Growing cultures of V79 cells in complete Williams medium E (WE; containing 10% fetal bovine serum) were exposed to 2-propen-1-ol (1 uM, 2 uM) for 2 hr, transferred to fresh medium for 24 hr, harvested and then reseeded (10E6 cells) into fresh medium for 10 d, with one subdivision.

These cells were harvested and divided for assessment of absolute plating efficiency (after 7 d growth in complete WE) and for mutation frequency (3.17X10E5 cells plated in the presence of 3 uM 6-thioguanine; 10 d incubation period with one change of medium).

Incubations were performed at 37 degrees C in 95% air:5% carbon dioxide and 80% relative humidity.

No exogenous metabolic activation was included.

Comment: The methods indicate that the concentration of fetal bovine serum present in the WE medium varied between 0-10% during exposure to 2-propen-1-ol. The intention was to investigate the possible protective role of thiol groups on any mutagenic response observed.

Result: A mutation frequency of 14+/-8 mutants/10E6 survivors was reported after exposure to 1 uM 2-propen-1-ol (58 ng/ml), and 37+/-12 after exposure to 2 uM (116 ng/ml; results are mean and SD of 8 plates from a single experiment).

No concurrent control data are reported.

The thiol status of the above incubations is not reported. Other studies described in this paper indicate that the

magnitude of any mutagenic response was greatly diminished by inclusion of 10% fetal bovine serum in the assay.

The authors conclude that 2-propen-1-ol was mutagenic in V79 cells in vitro.

Conclusion: Under the conditions of the assay, 2-propen-1-ol was reported to be mutagenic in V79 cells in the absence of exogenous metabolic activation.

Reliability: (2) valid with restrictions
Well reported published study

05-DEC-2005 (149)

Type: Bacterial reverse mutation assay
System of testing: Salmonella typhimurium TA100
Concentration: 200ug/plate
Metabolic activation: without
Result: negative

Year: 1980
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Method: Solvent: DMSO
Result: 2-Propen-1-ol:
-Revertants per plate 127

Spontaneous:
-Revertants per plate: about 120

Reliability: (3) invalid
05-DEC-2005 (166)

Type: Bacterial reverse mutation assay
System of testing: Salmonella typhimurium TA100
Concentration: 1.16 - 116 ug/plate
Cytotoxic Concentration: 1.16 ug/plate
Metabolic activation: with and without
Result: negative

Year: 1980
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Method: Solvent: DMSO
Result: 2-Propen-1-ol:
-Revertants per plate: 0 both +S9 and -S9

Spontaneous:
-Revertants per plate: 110 - 153

Did not exceed twice the spontaneoud background level.

Reliability: (3) invalid
05-DEC-2005 (133)

Type: Salmonella typhimurium reverse mutation assay
System of testing: microsomal assay
Concentration: 0.1 to 100 ul/plate
Metabolic activation: with and without
Result: negative

Method: other
Year: 1977
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6)

Reliability: (4) not assignable
05-DEC-2005 (2)

5.6 Genetic Toxicity 'in Vivo'

Type: Micronucleus assay
Species: rat **Sex:** male
Strain: Fischer 344
Route of admin.: i.p.
Exposure period: 72 hr
Doses: 0, 5, 10, 20, 40, 60 or 80 mg/kg bw
Result: negative

Method: other: US-NTP standard protocol
Year: 1994
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), ID: A98432

Method: ANIMALS AND TREATMENTS
Male F344 rats were given 2-propen-1-ol at 5, 10 or 20 mg/kg bw (n=5/treatment) by i.p. injection on 3 consecutive days. The control group (n=4) received physiological saline. Bone marrow was collected 24 hr after the final treatment. Two thousand polychromatic erythrocytes (PCEs) were examined microscopically and scored for the presence of micronuclei. No further experimental details available.

POSITIVE CONTROL SUBSTANCE
Cyclophosphamide (7.5 mg/kg bw) was used as positive control (i.p. injection on 3 consecutive days).

STATISTICAL METHODS
Control versus test comparisons are reported together with trend analysis, however no information on methods applied.

Result: There was no statistically significant difference in the number of micronuclei per 1000 PCEs in rats given 2-propen-1-ol at 5, 10 or 20 mg/kg bw/d by i.p. injection on 3 consecutive days:

Dose (mg/kg)	No. of animals	Mean MN-PCE/1000 PCE	Pairwise P
Vehicle Control	4	1.38+/-0.24	
5	5	2.00+/-0.16	0.1575
10	5	1.60+/-0.33	0.3492
20	5	1.40+/-0.19	0.4822
Positive Control	5	24.20+/-0.78	<0.0001

Vehicle control: Phosphate buffered saline
Positive control: Cyclophosphamide (7.5 mg/kg)
The trend for incidence of micronucleated PCEs was

non-significant.

Animals given 40, 60 or 80 mg/kg died prior to scheduled study termination.

A satisfactory response was obtained with the positive control group (24.2MN-PCEs per 1000 PCEs; P=0.0001).

Conclusion: Under the conditions of the study, no increase in micronucleated polychromatic erythrocytes was detected in male F344 rats given 2-propen-1-ol at doses of up to 20 mg/kg bw/d for three consecutive days.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

06-JAN-2006 (108)

Type: Micronucleus assay
Species: mouse **Sex:** male/female
Strain: B6C3F1
Route of admin.: gavage
Exposure period: 13 weeks
Doses: 0, 3, 6, 12, 25 or 50 mg/kg bw/d
Result: negative

Method: other: US-NTP standard protocol
Year: 1995
GLP: yes
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), ID A65209

Method: ANIMALS AND TREATMENTS
Male and female B6C3F1 mice (n=10/sex/dose level) were given 2-propen-1-ol at doses of 0, 3, 6, 12, 25 or 50 mg/kg bw/d by oral gavage for 13 wk. (Comment: the control vehicle is not identified; it is not stated if treatment continued 5 d/wk or 7 d/wk). Peripheral blood was collected 24 hr after the final treatment. At least one thousand normochromatic erythrocytes (NCEs)
were examined microscopically and scored for the presence of micronuclei. No further experimental details available.

POSITIVE CONTROL SUBSTANCE
No positive control substance was used.

STATISTICAL METHODS
Control versus test comparisons are reported together with trend analysis, however no information on methods applied.

Result: There was no statistically significant difference in the number of micronucleated cells at any treatment level in either sex:

Trial 1 (PCE, Female)

Dose (mg/kg)	No. of animals	Mean MN-PCE/1000 PCE	Pairwise P
0	10	0.80+/-0.25	
50	9	1.56+/-0.34	0.0631

Trial 1 (NCE, Female)

Dose (mg/kg)	No. of animals	Mean MN-NCE/1000 NCE	Pairwise P
0	10	0.70+/-0.21	
3	10	0.90+/-0.28	0.3085
6	10	1.00+/-0.21	0.2333
12	10	0.70+/-0.26	0.5000
25	10	1.50+/-0.31	0.0440
50	9	1.11+/-0.26	0.1720

Trial 2 (PCE, Male)

Dose (mg/kg)	No. of animals	Mean MN-PCE/1000 PCE	Pairwise P
0	10	1.20+/-0.29	
50	10	2.20+/-0.49	0.0430

Trial 2 (NCE, Male)

Dose (mg/kg)	No. of animals	Mean MN-NCE/1000 NCE	Pairwise P
0	10	1.10+/-0.38	
3	10	1.20+/-0.25	0.4174
6	10	1.70+/-0.40	0.1283
12	10	1.40+/-0.34	0.2741
25	10	1.20+/-0.33	0.4174
50	10	1.60+/-0.45	0.1678

Conclusion: Under the conditions of the study, no increase in micronucleated normochromatic erythrocytes was detected in male or female B6C3F1 mice given 2-propen-1-ol by gavage at doses of up to 50 mg/kg bw/d for 13 weeks.
Reliability: (2) valid with restrictions
Well reported published study
Flag: Critical study for SIDS endpoint
06-JAN-2006 (109)

Type: other: enhanced dominant lethal test (with karyotypic evaluation)
Species: rat **Sex:** male
Strain: Sprague-Dawley
Route of admin.: gavage
Exposure period: 33 wk
Doses: 25 mg/kg bw
Result: negative

Method: other: research investigation
Year: 1990
GLP: no
Test substance: other TS: 2-Propen-1-ol (CAS No. 107-18-6), supplier: Aldrich Chemical Co., Gillingham, Dorset, UK

Method: ANIMALS AND TREATMENTS
Male SD rats (9-11 wk old) were given 0.85% saline (control group; n=6) or 2-propen-1-ol (25 mg/kg bw/d) by oral gavage

(10 ml/kg bw; 7 d/wk for 12 wk, 5 d/wk to wk 15).

Each male was caged with 2 virgin females (until a sperm-positive smear was obtained; up to 6 nights) on wk 1-11.

After mating was complete the males were subject to a gross postmortem examination and hematological screen. Sperm parameters were also assessed (no further methodological details): Males treated with 2-propen-1-ol were sacrificed in wk 15, while controls were maintained until wk 33 (dosed 5 d/wk, in support of a parallel experiment).

REPRODUCTION PARAMETERS
On GD20, females from mating weeks 1-11 were killed and the uteri examined for:
- total number of corpora lutea
- total implants
- live / dead fetuses
- late / early deaths (calculated as a percentage of the total implants from the pregnant females in each group)

FETAL EXAMINATION
Each fetus was weighed and examined. Abnormal fetuses were photographed (Polaroid) prior to removal of a sample of liver (chromosomal preparation) and preservation for skeletal staining and evaluation. The abnormal fetal index was calculated as a percentage of the total number of term fetuses observed at post-mortem. When karyotype abnormalities were observed the chromosome(s) involved were identified according to the standard karyotype of the Norway rat (Committee for a Standardised Karyotype of Rattus norvegicus; not included in study bibliography). (No further details are given on methods used for chromosomal and karyotypic analysis.)
STATISTICAL METHODS
Litter data were analyzed using Fisher's exact test. Other data were evaluated for significant differences relative to the controls, however the methods used are not reported.

Remark: The authors note that paternal exposure to a mutagenic agent resulted in changes in chromosomal structure and/or number, which may be manifest as fetal abnormalities or changes in karyotype.

Cyclophosphamide (3.5-5.1 mg/kg for up to 33 wk) was also evaluated in this study. It was associated with a highly significant and consistent increase in the numbers of malformed fetuses with karyotypic abnormalities. These effects were paralleled by a large increase in the number of post-implantation losses (dominant lethal events), but no significant effect on sperm parameters. These findings validate the methods used in this study.

Result: PATERNAL EFFECTS
Mean body weight was lower in male rats given 2-propen-1-ol (569+/-49 g) compared to controls (635+/-74 g); this may, in part, have reflected the 18 wk age difference at sacrifice. Relative liver weight was increased 26% ($P<0.05$), and relative spleen weight 22% ($P<0.05$), with non-significant increases in relative kidney and testis weights (data not reported).

Red cell count, mean cell volume, percentage cell volume and hemoglobin concentration were unaffected by treatment with 2-propen-1-ol (data not reported). White cell counts were similar in treated and control animals, however a differential count revealed a significant increase in percentage of lymphocytes with a corresponding significant decrease in eosinophils and neutraphil counts (data not reported). The authors comment that these changes in differential count were within the normal range for the SD rat.

Total sperm count and epididymal sperm concentration were unaffected by treatment (data not reported).

REPRODUCTION PARAMETERS
There was a total of 1669 live implants from 125 pregnancies in the controls (13.4 implants/litter) versus 1371 live implants from 108 litters in the 2-propen-1-ol-treated group (12.7 implants/litter) (non significant).

Mean preimplantation loss was comparable in control (12.8+/-5.4%) and treated (11.7+/-6.2%) groups.

The rate of post-implantation loss (dominant lethality) varied between 2.2-13.2% in the controls, with a mean for the whole study (13 matings) of 6.2%. The comparable range for the male rats given 2-propen-1-ol was 1.7-8.7%, with an overall mean of 4.1%.

FETAL ABNORMALITIES
The incidence of runted, abnormal and grossly abnormal fetuses was comparable in the control and treated groups.

	Control	Treated
Total number runts:	13 (0.78%)	13 (0.95%)
Total number gross abnormalities (%):	0 (0%)	3 (0.22%)
Total number abnormal fetuses (%):	13 (0.78%)	16 (1.17%)

The abnormalities seen in the litters of the three groups of treated animals were diagnosed as:
- anasarca (massive edema)
- exencephaly
- craniofacial and skeletal abnormality.

The combined incidence of gross abnormalities was not statistically significantly different between the control (n=0) and treated (n=3) groups.
Comment: The authors note that the incidence of grossly abnormal fetuses (0.22%) was within the historic range for this strain of rat (not reported) and were therefore considered to be spontaneous in origin.

KARYOTYPIC ANALYSIS
Karyotypic abnormalities were present in 3 of 12 slides prepared from abnormal fetuses from the treated group whereas no abnormal karyotype was present in 5 slides from the controls:

	Control	Treated
No. of abnormal fetuses evaluated:	8	14
Slides with scorable metaphases:	5	12
Slides with no karyotypic abnormality	5	9
Slides with karyotypic abnormality	0	3

The abnormalities from the 2-propen-1-ol treated group were diagnosed as:
- trisomy with 3 fragments of possible centromeric origin in every metaphase (runt)
- trisomy (anasarca/runt)
- trisomy (craniofacial/skeletal)

Comment: Data for other endpoints described in the methods section are not reported in the paper; it is assumed that these were unaltered by treatment with 2-propen-1-ol.

Conclusion: Under the conditions of the study, no increase in post-implantation loss (dominant lethality) or chromosomal/karyotypic abnormalities were present in fetuses sired by male SD rats given 2-propen-1-ol at 25 mg/kg bw/d for up to 12 wk.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

03-JAN-2006 (63)

5.7 Carcinogenicity

Species: rat **Sex:** male/female
Strain: Fischer 344
Route of administration: drinking water
Exposure period: 106 wk
Frequency of treatment: until natural death
Doses: 0 or 300 mg/L (total dose 3.2 g)
Result: negative
Control Group: yes, concurrent vehicle

Year: 1987
GLP: no data
Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), suuplied by Aldrich Chemical Company (no further details)

Method: Male and female F344 rats (n=20/sex; age 7-8 wk; housed 4/cage) were given 2-propen-1-ol (300 mg/l) in drinking water 5 d/wk for up to 106 wk (tap water given on remaining days). A similar number of animals received tap water ad libitum. The animals were allowed to survive until natural death or until wk 123-132 of the study, which ever occurred later.

Fresh solutions were prepared weekly and stored in a refrigerator until use. Stability studies (GC analysis) demonstrated 100% recovery of 2-propen-1-ol after 7 days (no further information available).

Result: Median survival was unaffected by treatment:

- males: controls = 115 wk, treated = 113 wk
- females: controls = 118 wk, treated = 112 wk

The tumors observed in this study were stated to be of the types commonly seen in untreated F344 rats. Findings were as follows:

LIVER:
Hyperplastic nodules and a few well differentiated hepatocellular carcinoma.
Controls: 2/20 M; 2/20 F
Treated: 3/20 M; 6/20 F

ADRENAL CORTEX:
Hyperplastic nodules and adenoma
Controls: 1/20 M; 1/20 F
Treated: 0/20 M; 0/20 F

PITUITARY: No description.
Controls: 14/20 M; 14/20 F
Treated: 10/20 M; 10/20 F

LEUKEMIA: No description.
Controls: 12/20 M; 6/20 F
Treated: 8/20 M; 6/20 F

Comment: While the authors comment that the occurrence of specific tumor types was increased after treatment with other substances included in this study, the increased occurrence of hepatic nodules/carcinoma in females given 2-propen-1-ol relative to that present in the controls is not the subject of discussion. When evaluated in combination with the liver effects found in other studies, the increased occurrence of hepatic nodules/carcinoma in females is considered to be biologically significant and may indicate equivocal evidence for carcinogenicity.

Conclusion: Under the conditions of this study, no clear evidence of carcinogenicity was seen in male F344 rats given 2-propen-1-ol in drinking water (300 mg/L) for 106 weeks, but equivocal evidence of carcinogenicity was seen in the liver of female rats.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

02-DEC-2005 (83) (85)

Species: Syrian hamster **Sex:** male
Route of administration: gavage
Exposure period: 60 wk
Frequency of treatment: until natural death
Post exposure period: 30-32 wk
Doses: 0 or 2 mg/day (total dose 120 mg)
Result: negative
Control Group: yes, concurrent vehicle

Year: 1987
GLP: no data
Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), suuplied by Aldrich Chemical Company (no further details)

Remark: In a poorly reported study, no increase in tumors of the adrenal cortex, forestomach or pancreas duct was noted in male Syrian hamsters give 2 mg 2-propen-1-ol in corn oil for up to 60 wk. The animals were allowed to survive until natural death or for up to a further 30-32 wk post-treatment. Although not stated, it is assumed that gavage administration was employed.
Reliability: (4) not assignable
Poorly reported published study
02-DEC-2005 (83) (85)

5.8.1 Toxicity to Fertility

Species: rat
Sex: male/female
Strain: other:Crj:CD(SD)IGS
Route of administration: gavage
Exposure Period: Males, 42 days. Females, from 14 days before mating to day 3 of lactation
Frequency of treatment: once daily
Premating Exposure Period
male: 14 days before mating
female: 14 days before mating
Duration of test: Refer to Test Conditions
Doses: 0 (vehicle), 2, 8 or 40 mg/kg bw/day
Control Group: yes, concurrent vehicle
NOAEL F1 Offspring: 8 mg/kg bw
other: NOAEL Parental (M) : 8 mg/kg bw
other: NOAEL Parental (F) : 8 mg/kg bw

Method: OECD Guide-line 421
Year: 2004
GLP: yes
Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), Supplied by SHOWA DENKO K.K.

Result: [I]. Toxicity to parental toxicity

(1) Mortality and clinical signs
-Mortality
--Male and female
---At all treated groups
No deaths were observed.

-Clinical signs
--Male
---At 40 mg/kg/day group
Salivation, decrease in locomotor activity, irregular respiration, lacrimation, loose stool
---At 2 and 8 mg/kg/day group
No abnormality

--Female
---At 40 mg/kg/day group
Salivation, decrease in locomotor activity, irregular respiration
(Total litter loss)
---At 2 and 8 mg/kg/day group
No abnormality

(2) Body weight and body weight gain
-Male and female
--At all treated groups
The body weight was similar with that of control group.

(3) Food consumption
-Male and female
--At all treated groups
The food consumption was similar with that of control group.

(4) Organ weight (male, only testis and epididymis)
-Male
--At all treated groups
No toxicological significances at testis and epididymis.

(5) Necropsy findings
See the tables

Table 1. Necropsy findings
-Male

Dose (mg/kg/day)	0	2	8	40
No. of animals	12	12	12	12
Stomach				
Thickening, forestomach, wall	0	0	0	1
Thickening, limiting ridge	0	0	0	1
Liver				
Brownish patch	0	0	0	2
Rough surface	0	0	0	3
Testis				
Asymmetry	0	0	1	1
Small	1	0	0	0
Epididymis				
Nodule, tail	0	1	0	0
Small	1	0	0	0

Table 2. Necropsy findings
-Female

Dose (mg/kg/day)	0	2	8	40
No. of animals	11	12	12	11
Thymus				
Atrophy				

	0	0	0	7
Liver				
Enlargement				
	0	0	0	8
Rough surface				
	0	0	0	1
Yellowish patch				
	0	0	0	7
Adrenal				
Whitish change				
	0	0	0	7

(6) Histological findings
See the tables

Table 3. Histological findings
-Male

Dose (mg/kg/day)	0	2	8	40
Stomach				
Edema, forestomach				
	-	-	-	1/2
Hyperplasia, squamous, forestomach				
	-	-	-	2/2
Liver				
Fibrosis, perilobular				
	-	-	-	3/3
Hypertrophy, hepatocyte, perilobular				
	-	-	-	3/3
Necrosis, hepatocyte, perilobular				
	-	-	-	2/3
Proliferation, bile duct				
	-	-	-	3/3
Testis				
	1/12	-	1/1	1/12
Epididymis				
	1/12	0/1	-	1/12
Adrenal				
	-	1/1	-	-

*: Significant difference from control, p<0.05
**: Significant difference from control, p<0.01
-: Not tested
(No. animals with histological change)/(No. animals examined)

Table 4. Histological findings
-Female

Dose (mg/kg/day)	0	2	8	40
Thymus				
Atrophy				
	-	-	-	5/7
Liver				
Brown pigment deposition, perilobular				

```
                          -    -    -    2/10
 Clear cell change, hepatocyte, diffuse
                          -    -    -    8/10
 Fibrosis, perilobular
                          -    -    -    8/10
 Hypertrophy, hepatocyte, perilobular
                          -    -    -    5/10
 Necrosis, hepatocyte, perilobular
                          -    -    -    8/10
 Proliferation, bile duct
                          -    -    -    8/10
Ovary
 Hypertrophy, luteal cell
                         0/11 0/12 0/12 5/11*
Adrenal
                          -    -    -    0/7
-------------------------------------------------------------
*: Significant difference from control, p<0.05
**: Significant difference from control, p<0.01
-: Not tested

(No. animals with histological change)/(No. animals
examined)

[II]. Reproductive and developmental toxicity
(1) Reproductive performance
-At 40 mg/kg/day group
 Extension of mean estrous cycle and increase of females
with irregular estrous cycle
-At 2 and 8 mg/kg/day group
 No abnormality

Table 5. Reproductive performance
-------------------------------------------------------------
Dose (mg/kg/day)
                          0         2         8         40
-------------------------------------------------------------
No. of pairs
                          12        12        12        12
Mean estrous cycle
                          4.03      4.00      4.00      4.30**
No. of females with irregular estrous cycle
                          0/12      0/12      0/12      4/12*
Mating period
 No. of estrous
                          0.0       0.0       0.0       0.0
 Day of conceiving
                          2.2       2.6       2.7       2.3
Copulation index (%) a)
                          100.0     100.0     100.0     100.0
Fertility index (%) b)
                          91.7      100.0     100.0     100.0
-------------------------------------------------------------
a): (No. of copulated females)/(No. of pairs)
b): (No. of pregnant females)/(No. of copulated females)
*: Significant difference from control, p<0.05
** Significant difference from control, p<0.01

(2) Delivery and lactation
-At all treated groups
```

No abnormality

Table 6. Delivery data

Dose (mg/kg/day)	0	2	8	40
n	11	12	12	12
Gestation length (days)	22.3	22.2	22.3	22.5
No. of corpora lutea	15.8	15.1	15.8	17.1
No. of implantation sites	15.1	14.4	15.6	16.3
Total No. of offspring	14.5	13.8	14.8	15.3
Implantation index (%)	95.73	95.47	98.48	95.85
Delivery index (%)	95.64	96.28	94.74	93.56
Gestation index (%) a)	100.0	100.0	100.0	100.0

a): (No. of pregnant animals delivered live offspring)/(No. of pregnant animals)
*: Significant difference from control, p<0.05
**: Significant difference from control, p<0.01

(3) Morphology, body weight and necropsy findings of offspring

Table 7: Litter Size and Viability Index (F1)

Dose (mg/kg)		Total Number of Offspring at Birth			Number of Live Offspring at Birth		
		M	F	Total	M	F	Total
0	Mean	6.6	7.8	14.5	6.6	7.8	14.5
	S.D	2.2	1.9	1.9	2.2	1.9	1.9
	N	11	11	11	11	11	11
(M/F)		(73/86)			(73/86)		
2	Mean	7.0	6.8	13.8	7.0	6.8	13.8
	S.D	1.9	2.6	1.9	1.9	2.6	1.9
	N	12	12	12	12	12	12
(M/F)		(84/82)			(84/82)		
8	Mean	6.8	8.0	14.8	6.8	7.9	14.7
	S.D	2.0	2.5	0.9	2.0	2.4	0.7
	N	12	12	12	12	12	12
(M/F)		(81/96)			(81/95)		
40	Mean	8.3	7.0	15.3	7.8	6.9	14.8
	S.D	1.8	1.0	1.7	1.3	0.9	1.5
	N	12	12	12	12	12	12
(M/F)		(99/84)			(94/83)		

Dose (mg/kg)		Number of Live Offspring at Birth Before Culling			Viability Index (%)	
		M	F	Total	Day 0	Day 4
0 (M/F)	Mean	6.6	7.7	14.4	100.00	99.39
	S.D	2.2	2.1	1.9	0.00	2.02
	N	11	11	11	11	11
	(73/85)					
2 (M/F)	Mean	7.0	3.8	13.8	100.00	100.00
	S.D	1.9	2.6	1.9	0.00	0.00
	N	12	12	12	12	12
	(84/82)					
8 (M/F)	Mean	6.7	7.9	14.6	99.51	99.41
	S.D	1.8	2.4	0.8	1.7	2.05
	N	12	12	12	12	12
	(80/95)					
40 (M/F)	Mean	6.8	6.4	13.2	96.97	89.48
	S.D	2.4	2.1	4.4	4.85	28.50
	N	12	12	12	12	12
	(81/77)					

Table 8: Clinical Signs (F1 before Weaning)

Dose (mg/kg)	Findings (M/F)	Day 0	1	2	3	4
0	Number of dams	11	11	11	11	11
	Number of offspring	73/86/0	73/86	73/86	73/86	73/85
	Number of dams with abnormal offspring	1	1	0	0	0
	No abnormality	72/86	73/85	73/86	73/85	73/85
	Death (M/F/U)	0/0/0	0/0	0/0	0/1	0/0
	Loss of Suckling	1/0	0/1			
2	Number of dams	12	12	12	12	12
	Number of offspring	84/82/0	84/82	84/82	84/82	84/82
	Number of dams with abnormal offspring	0	0	0	0	0
	No abnormality	84/82	84/82	84/82	84/82	84/82
	Death (M/F/U)	0/0/0	0/0	0/0	0/0	0/0
8	Number of dams	12	12	12	12	12
	Number of offspring	81/96/0	81/95	81/95	80/95	80/95
	Number of dams with abnormal offspring	0	0	0	0	0
	No abnormality	81/95	81/95	80/95	80/95	80/95
	Death (M/F/U)	0/1/0	0/0	1/0	0/0	0/0
	Number of dams	12	12	11	11	11

40	Number of offspring	99/84/0	94/83	82/77	81/77	81/77
	Number of dams with abnormal offspring	3	0	0	0	0
	No abnormality	88/78	82/77	81/77	81/77	81/77
	Death (M/F/U)	5/1/0	12/6	1/0	0/0	0/0
	Loss of Suckling	6/5				

M: Male, F: Female, U: Unable to be sexed on day 0

Table 9: External Examination of Offspring (F1) on Day 0 (Birth day)

Dose (mg/kg)	0	2	8	40
No. of Dams	11	12	12	12
No. of Offspring	159	166	176	177
No. Dams with anomalous offspring	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
No. Offspring with any anomalies	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

Significantly different from control: * P<0.05; ** P<0.01

-Morphology
--At 40 mg/kg/day group
  Decrease in viability index on day 4 (not statistically)
  Total litter loss (from one dam)
-Body weight
--At all treated groups
  No toxicological significances

Table 10: Body weight of Male and Female Offspring (F1 before Weaning)

Dose (mg/kg)		Male day 0-4	Female day 0-4
0	Mean	3.3	3.2
	SD	0.3	0.4
	N	11	11
2	Mean	3.4	3.2
	SD	0.6	0.4
	N	12	12
8	Mean	3.8	3.6
	SD	0.4	0.4
	N	12	12
40	Mean	2.9	2.7
	SD	1.1	1.0
	N	11	11

--

-Necropsy findings
  No toxicological significances

Table 11: Necropsy findings (F1 Offspring)

Dose (mg/kg)	Findings	Scheduled Sacrifice Day 4 M	F
0	Number of offspring examined	73	85
	No abnormality	73	85
2	Number of offspring examined	84	82
	No abnormality	83	82
	Liver	1	
	Yellow patch		
8	Number of offspring examined	80	95
	No abnormality	80	95
40	Number of offspring examined	81	77
	No abnormality	81	77

Dose (mg/kg)	Findings	Dead (M/F Day: 0	1	2	3	4
0	Number of offspring examined					0/1
	No abnormality			0/1		
2	Number of offspring examined					
	No abnormality					
	Liver					
	Yellow patch					
8	Number of offspring examined			0/1		
	No abnormality		0/1			
40	Number of offspring examined			5/1	8/5	1/0
	No abnormality		5/1	8/5	1/0	

M: Male, F: Female

The NOAEL for general toxicity and reproductive and developmental toxicity is considered to be 8 mg/kg/day

Test condition: (1) Animals:
-At the start of administration
9 weeks old, Sprague-Dawley (Crj:CD(SD)IGS, SPF) rats (obtained from Charles River Japan, Inc.)
They were put in quarantine for 5 days beforehand.
Body weight; male 337-388 g, female 202-233 g
(2) Number of animals/group:
Males, 12;
Females, 12
(3) Rout:
Oral (gavage)

(4) Dosage:
0 (vehicle), 2, 8, 40 mg/kg/day
(5) Vehicle:
Water for injection, JP
(6) Confirmation of test solution concentration:
Test solutions were analyzed and warranted to be stable for 8 days.
(8) Administration period:
Males, 42 days from 14 days before mating and the mating period to the day before necropsy.
Females, from 14 days before mating to day 3 of lactation
(9) Terminal killing:
Males, day 43.
Females, day 4 of lactation.
(10) Statistics Analysis:
Bartlett's tests were initially performed. As the population was in equal variance on Bartlett's tests, one-way analysis of variance was performed. And as the population was not in equal variance on Bartlett's tests, Kruskal-Wallis's test was performed. In the case of the significant difference between dose groups, Dunnett's multiple comparison test was performed. Kruskal-Wallis's test were performed directly for some parameters. Wilcoxon rank-sum test was performed for histopathological findings. Fisher's exact test was performed for the other parameters. Five
percent as significant level was adopted for all tests.

Conclusion: The NOAEL for general toxicity and reproductive and developmental toxicity is considered to be 8 mg/kg/day.

Reliability: (1) valid without restriction
OECD TG Study

Flag: Critical study for SIDS endpoint

09-MAY-2006 (6)

Species: rat
Sex: male
Strain: Wistar
Route of administration: drinking water
Exposure Period: 15 wk
other: NOAEL Parental (M) : 48.2 mg/kg bw
other: NOAEL Parental (F) : 58.4 mg/kg bw

Year: 1978
GLP: no

Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), 99% pure, SG (20 degree C) 0.849-0.852; bpt
95-98 degrees C, supplied by Bush Boake Allen Ltd, London.

Method: ANIMALS AND TREATMENTS
Groups of Wistar rats (15/sex/treatment level) were exposed to 2-propen-1-ol in the drinking water at 0 (control), 50, 100, 200 or 800 ppm for 15 weeks.

NECROPSY AND HISTOPATHOLOGY
At the end of the appropriate treatment period, animals were killed by exsanguination following an overnight fast and subject to a post-mortem examination. This included gonadal weights and histopathological examination of testis, ovary and uterus.

See Section 5.4 for further experimental details.

Result: INTAKE OF TEST SUBSTANCE
The calculated mean intake of 2-propen-1-ol over the course of the study (based on body weight and water intake data) was:
Males: 0, 4.8, 8.3, 14.0, 48.2 mg/kg bw/d
Females: 0, 6.2, 6.9, 17.1 and 58.4 mg/kg bw/d

POST MORTEM EXAMINATION
Absolute organ weights (including gonadal weights) were generally decreased in males, and to a lesser extent in females, in a time- and treatment related manner. Relative organ weights (including gonadal weights) were generally increased to a statistically significant extent in high dose animals of both sexes at study termination. These changes appeared secondary to a reduction in water intake (presumably due to unpalatability of the treatment solution) and body weight, that was particularly pronounced in high dose animals.

HISTOPATHOLOGICAL EVALUATION
No histopathological abnormalities were reported for testis, ovary or uterus.

Conclusion: No treatment-related changes were present in gonadal weights or histopathology in male and female rats given allyl alcohol at received doses of up to 48.2, 58.4 mg/kg bw/d.

Reliability: (2) valid with restrictions
Well reported published study

Flag: Critical study for SIDS endpoint

09-MAY-2006 (28)

Species: rat
Sex: male
Strain: Sprague-Dawley
Route of administration: gavage
Exposure Period: up to 15 wk
Frequency of treatment: 7d/wk
Premating Exposure Period
male: up to 11 wk
female: untreated
Doses: 25 mg/kg bw
Control Group: yes
NOAEL Parental: 25 mg/kg bw

Method: other: research investigation
Year: 1990
GLP: no
Test substance: as prescribed by 1.1 - 1.4

Method: ANIMALS AND TREATMENTS
Male SD rats (9-11 wk old) were given 0.85% saline (control group; n=6) or 2-propen-1-ol (25 mg/kg bw/d) by oral gavage (10 ml/kg bw; 7 d/wk for 12 wk, then 5 d/wk to wk 15).

Each male was caged with 2 virgin females (until a sperm-positive smear was obtained; up to 6 nights) on wk 1-11.

After mating was complete the males were subject to a gross postmortem examination. Gonadal weights and sperm parameters

(no further methodological details) were assessed. Males treated with 2-propen-1-ol were sacrificed in wk 15, while controls were maintained until wk 33 (dosed 5 d/wk, in support of a parallel experiment).

REPRODUCTION PARAMETERS
On GD20, females from mating weeks 1-11 were killed and the uteri examined for:
- total number of corpora lutea
- total implants
- live / dead fetuses
- late / early deaths (calculated as a percentage of the total implants from the pregnant females in each group)

STATISTICAL METHODS
Litter data were analyzed using Fisher's exact test. Other data were evaluated for significant differences relative to the controls, however the methods used are not reported.

Result: PATERNAL EFFECTS
Mean body weight was lower in male rats given 2-propen-1-ol (569+/-49 g) compared to controls (635+/-74g); this may, in part, have reflected the 18 wk age difference at sacrifice.

Relative testis weights were increased (data not reported; non-significant).

Total sperm count and epididymal sperm concentration were unaffected by treatment (data not reported).

REPRODUCTION PARAMETERS
There was a total of 1669 live implants from 125 pregnancies in the controls (13.4 implants/litter) versus 1371 live implants from 108 litters in the 2-propen-1-ol-treated group (12.7 implants/litter) (non significant).

Mean preimplantation loss was comparable in control (12.8+/-5.4%) and treated (11.7+/-6.2%) groups.

Comment: Data for other endpoints described in the methods section are not reported in the paper; it is assumed that these were unaltered by treatment with 2-propen-1-ol.

Test substance: 2-Propen-1-ol, Aldrich Chemical Co., Gillingham, Dorset, UK (no further details).

Conclusion: Under the conditions of the study, no statistically significant changes were present in relative testis weight, total sperm count and epididymal sperm concentration or reproductive performance for male SD rats given 2-propen-1-ol at 25 mg/kg bw/d for up to 12 wk.

Reliability: (2) valid with restrictions
Well reported published study

02-DEC-2005 (63)

5.8.2 Developmental Toxicity/Teratogenicity

Species: rat **Sex:** female
Strain: other: Crl:CD (SD) IGS BR
Route of administration: gavage
Exposure period: Gestation days 6-19
Frequency of treatment: daily

Duration of test: 14 days
Doses: 0, 10, 35, 50 mg/kg bw/day
Control Group: yes, concurrent vehicle
NOAEL Teratogenicity: 10 mg/kg bw
LOAEL Maternal Toxicity : 10 mg/kg bw

Method: OECD Guide-line 414 "Teratogenicity"
Year: 2005
GLP: yes
Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), Purity 99.38%, Lot No. 20906MB, supplier Aldrich Chemical Co, Allentown, Pennsylvania.

Method: Statistical Methods:

All statistical tests were performed using appropriate computing devices or programs. Analyses were conducted using two-tailed tests (except as noted otherwise) for minimum significance levels of 1% and 5%, comparing each test article-treated group to the control group.

Mean maternal body weights (absolute and net), body weight changes (absolute and net) and food consumption, gravid uterine weights, numbers of corpora lutea, implantation sites and viable fetuses, and fetal body weights (separately by sex and combined) were subjected to a parametric one-way analysis of variance (ANOVA) to determine intergroup differences. If the ANOVA revealed statistically significant ($p<0.05$) intergroup variance, Dunnett's test was used to compare the test article-treated groups to the control group. Mean litter proportions (percent per litter) of prenatal data (viable and nonviable fetuses, early and late resorptions, total resorptions, pre- and postimplantation loss and fetal sex distribution), total fetal malformations and developmental variations (external, visceral, skeletal malformation or variation were subjected to the Kruskal-Wallis nonparametric ANOVA test to determine intergroup differences. If the ANOVA revealed statistically significant ($p<0.05$) intergroup variance, the Dunn's test was used to compare the test article-treated groups to the control group.

Result: Maternal data:

Mortality: One female in the 35 mg/kg bw/day group died on gestation day 20. Six females in the 50 mg/kg bw/day group died on gestation days 9, 10 (two), 11 (two) and 16. The following clinical observations were noted within 4 days for all females found dead. All females (except one in the 50 mg/kg bw/day group) had findings of salivation and/or clear material on various body surfaces and wiping the mouth on the cage floors and/or walls at the daily examinations and/or one hour folowing dose administration as a result of the irritant properties of the test article. All females that died displayed signs of poor grooming as a result of declining health, including unkempt appearance and/or yellow, brown and/or red colored amterial on various body surfaces. All females (except one in the 50 mg/kg bw/day group) had behavioural findings indicative of moribundity, including extremities cool to the touch, rocking, lurching or swaying while walking and/or hypoactivity. The female that died in the 35 mg/kg bw/day group had shallow respiration on gestation day

19 and two females from the 50 mg/kg bw/day group had decreased defecation on gestation days 8 and 10.

At necropsy of the animals that died, the female in the 35 mg/kg bw/day and two from the 50 mg/kg bw/day had a distended stomach, dark red stomach contents and/or dark red areas on the stomach lining. The 35 mg/kg bw/day female also had test article-related white and yellow areas on the liver at necropsy and an entirely resorbed litter. All animals found dead had large body weight losses and reduced food consumption within 2-4 days prior to death.

All other animals survived to the scheduled necropsy. At the time of dose administration, salivation was noted in six animals in the 35 mg/kg bw/day group and 13 animals in the 50 mg/kg bw/day group between gestation days 11-19. Three of the animals in the 50 mg/kg bw/day group had salivation prior to dosing. Approximately one hour following dose administration, incidences of salivation and evidence of salivation occurred in 17 females in the 35 mg/kg bw/day group and 20 females in the 50 mg/kg bw/day group. Excessive pawing of and/or mouth wiping on the cage floor and/or walls occurred in 19 animals dosed at 10 mg/kg bw/day, 23 animals dosed at 35 mg/kg bw/day and 19 animals dosed at 50 mg/kg bw/day. Lacrimation was observed in six females dosed at 50 mg/kg bw/day.

Other clinical findings noted in the test article-treated groups at the time of dosing and/or approximately one hour after dosing including hair loss on various body surfaces were noted infrequently and did not occur in a dose related manner.

Maternal body weights and gravid uterine weights:

50 mg/kg bw/day: A test article-related mean maternal body weight loss of 12g was noted in animals during gestation days 6-9 compared to a mean body weight gain of 11g in the control group (statistically significant, $p<0.01$). Mean body weight gain was slightly reduced (not statistically significant) when the entire treatment period (gestation days 6-20) was evaluated. Mean net body weight, net body weight gain and gravid uterine weight were slightly reduced (not statistically significant) compared to the control group.

35 mg/kg bw/day: A test article-related mean maternal weight loss of 4 g was noted during gestation days 6-9 compared to a mean body weight gain of 11 g in the control group. The difference was statistically significant ($p<0.01$). Mean maternal body weight gains were similar to the control group during gestation days 9-12, but statistically significantly ($p<0.05$ or $p<0.01$) reduced on gestation days 12-20 and when the entire treatment period (gestation days 6-20) was evaluated. The sustained reductions in mean body weight gain were attributed to the continued survival of the animals most affected by test article administration. As a result of the effect on mean body weight gain, mean body weights were reduced 4.0% to 7.6% on gestation days 16 to 20; the difference on gestation day 20 was significantly significant ($p<0.05$). Mean net body weight, net body weight gain and gravid uterine weight were reduced (not statistically significant) compared to the control group. The decrease in

gravid uterine weight was attributed to two females that had entirely resorbed litters.

10 mg/kg bw/day: Mean maternal body weights, body weight gains, net body weight, net body weight gain and gravid uterine weight were similar to the control group. Differences from the control group were slight and not statistically significant.

Maternal food consumption:

50 mg/kg bw/day: Statistically significant decrease ($p<0.01$) in mean meternal food consumption during gestation days 6-9 and 9-12. Mean food consumption was similar to the control group when the entire treatment period was evaluated, as a result of the deaths of the most severely affected animals by gestation day 16.

35 mg/kg bw/day: Statistically significant ($p<0.01$) reduction in mean food consumption during gestation days 6-9, which corresponded to the reduced mean body weight gain for this group for the same interval. Mean food consumption was statistically significantly ($p<0.05$) lower on gestation days 9-12. Due to mortality and the number of animals not consuming an appreciable amount of food, supplemental feed (an approximately 50/50 mixture of Hills Prescription Diet canine feed and water) was administered to all animals consuming less than 10 g/day (see Table below).

Table: Animals given supplemental feed

Animal No.	Dose Level (mg/kg bw/day)	Gestation Day
63865	35	14, 15, 16
63882	35	14, 15
63860	50	14
63932	35	17, 18, 19
63920	35	16, 17, 19
63826	35	15
63876	35	15, 16, 17, 18, 19
63818	0	17

Following supplementation of the diet for six animals in the 35 mg/kg bw/day group beginning on gestation day 14, mean food consumption during gestation days 12-20 remained slightly reduced to the control group, but was increased from the gestation days 6-9 and 9-12 values. There was a statistically significant ($p<0.01$) reduction in mean food consumption in the 35 mg/kg bw/day group when the entire treatment period (gestation days 6-20) was evaluated.

10 mg/kg bw/day: Food consumption was similar to that in the control group throughout gestation. Differences from the control group were slight and not statistically significant.

Maternal necropsy data:

50 mg/kg bw/day: Females that died early in this group had distended stomachs, red stomach contents and/or dark red areas on the stomach lining. These findings were attributed to the irritant properties of the test article. One of the females had no significant internal findings at necropsy and was nongravid. The other two females had 18 and 19 normally developing implantation sites, respectively. At the scheduled necropsy on gestation day 20, 12 of the surviving 19 females in this group had test article-related liver findings (yellow and/or white areas on the liver, liver adhesions and/or misshapen or mottled livers). Of the animals with liver findings, one female had an enlarged spleen and one female had dark red discoloration of the lungs.

35 mg/kg bw/day: The female that died early in this group was found to have white and yellow areas on all lobes of the liver and an entirely resorbed litter (all early resorptions). At the scheduled necropsy on gestation day 20, 11 of the surviving 24 females in this group had test article-related liver findings (yellow and/or white areas on the liver, liver adhesions and/or misshapen or mottled livers). Of the animals with liver findings, one female had an enlarged spleen and two females had a thickened pericardium and/or pericardium adhesions, one of which also had white discoloration of the heart. One female had white fluid in the vagina.

10 mg/kg bw/day: One female had yellow areas on the liver. No clinical observations or effects on body weight gain, food consumption or intrauterine growth and survival were noted for this animal. However, the yellow areas on the liver were considered to be test article-related because this finding was observed at a higher incidence in the 35 and 50 mg/kg bw/day groups, but was not observed in any control females. Other incidental findings observed in this group included one to two females with dark red uterine contents, fused placentae or a thyroid mass.

Control: In the control group, one female had a mammary gland mass. No other internal findings were observed in the control group females.

Organ weights:

50 mg/kg bw/day: Test article-related increases in mean liver weights (11.6%) were observed when compared to controls. The difference was statistically significant ($p<0.01$). The increased liver weights correlated with the macroscopic findings observed in this group.

35 mg/kg bw/day: Test article-related increases in mean liver weights (5.4%) were observed when compared to controls. The difference was not statistically significant. The increased liver weights correlated with the macroscopic findings observed in this group.

10 mg/kg bw/day: No test article-related effect on mean liver weight was noted.

Gestation day 20 laparohysterectomy data:

50 mg/kg bw/day: Test article-related increases (not statistically significant) in the mean litter proportions of postimplantation loss (early resorptions) were observed (14.3% per litter) compared to the control group value (6.9% per litter). These values also exceeded the maximum value in the laboratory's developmental historical control data (8.6% per litter). Corresponding reductions in the mean litter proportion of viable fetuses were also observed. The increased postimplantation loss in this group was primarliy atirbuted to two females that had entirely resorbed litters. These animals also had test article-related effects in mean body weight gains during the treatment period. Mean fetal weight was unaffected by test article administration. Other parameters evaluated, including mean live litter size, fetal sex ratios and numbers of corpora lutea and implantation sites, were similar to the control group values.

35 mg/kg bw/day: Test article-related increases (not statistically significant) in the mean litter proportions of postimplantation loss (early resorptions) were observed (16.2% per litter) compared to the control group value (6.9% per litter). These values also exceeded the maximum value in the laboratory's developmental historical control data (8.6% per litter). Corresponding reductions in the mean litter proportion of viable fetuses were also observed. The increased postimplantation loss in this group was primarliy atirbuted to two females that had entirely resorbed litters. These animals also had test article-related effects in mean body weight gains during the treatment period. Mean fetal weight was slightly reduced (3.4 g) compared to the control group (3.6 g). This was primarily due to one female that had a drastically reduced mean fetal weight (1.7 g) and also resorbed 38.9% of its litter. This dam also had a large body weight loss over the entire treatment period. Due to these factors, as well as a lack of dose response across groups, the resuction in mean fetal weight was not considered to be treatment related. Other parameters evaluated, including mean live litter size, fetal sex ratios and numbers of corpora lutea and implantation sites, were similar to the control group values.

10 mg/kg bw/day: The mean litter proportion of postimplantation loss was unaffected by test artilce administration in this group. Other parameters evaluated, including mean live litter size, fetal sex ratios and numbers of corpora lutea and implantation sites, were similar to the control group values.

Fetal morphological data:

The numbers of fetuses (litters) available for morphological evaluation were 387(25), 406(24), 296(19) and 241(16) in the control, 10, 35 and 50 mg/kg bw/day groups, respectively. Malformations were observed in 0(0), 2(2), 1(1) and 0(0) fetuses (litters) in these same respective dose groups and were considered spontaneous in origin. When the total malformations (0.0%, 0.5%, 0.5% and 0.0% per litter) and developmental variations (37.6%, 37.5%, 41.2% and 40.9%) were evaluated on a proportional basis in the control, 10, 35 and

50 mg/kg bw/day groups, respectively, no statistically significant differences from the controls were noted. Fetal malformations and developmental variations, when observed in the test article-treated groups, occurred infrequently or at a frequency similar to that in the control group, did not occur in a dose-relatedmanner and/or were within the laboratory's historical control data ranges. Based on these data, no fetal malformations or developmental variations were attributed to the test article.

Test condition: Age at study initiation: Approximately 12 weeks old when paired for breeding.

Number of animals/sex/dose: 25 females/dose

Vehicle: Deionized water

Clinical observations performed and frequency: All rats were observed twice daily (morning and afternoon) for moribundity and mortality. Individual detailed clinical observations were recorded from gestation days 0 through 20 (prior to dose administration during the treatment period). Animals were also observed for signs of toxicity at the time of dose administration and approximately one hour following dose administration.

Mating procedures: At the conclusion of the 13-day acclimation period, wach female was placed in a suspended wite mesh cage with a resident male from the same strain and source for breeding. resident males were untreated, sexually mature rats utilized exclusively for breeding. Positive evidence of mating was confirmed by the presence of a vaginal copulatory plug or the presence of sperm following a vaginal lavage. Each mating pair was examined daily. The day on which evidence of mating was identified was termed gestation day 0 and the animals were separated.

Parameters assessed during study:
Maternal:
Body weights and gravid uterine weights: Individual body weights were recorded on gestation days 0 and 6-20 (daily). Gravid uterine weight was collected and net body weight (the day 20 body weight exclusive of the weight of the uterus and contents) calculated for each gravid female at the scheduled laparohysterectomy.

Food consumption: Individual food consumption was recorded on gestation days 0 and 6-20 (daily).

Necropsy: Gross necropsy was performed on females that died during the course of the study. Sections of the liver and all gross lesions were retained in 10% neutral-buffered formalin for possible future histopathologic examination. The number and location of implantation sites and corpora lutea were recorded.

All surviving females were euthanized on gestation day 20. The thoracic, abdominal and pelvic cavities were opened by a ventral mid-line incision and the contents examined. The uterus and ovaries were exposed and excised. The number of corpora lutea on each ovary was recorded. The trimmed uterus

was weighed and opened and the number and location of all fetuses, early and late resorptions and the total number of implantation sites were recorded. Liver weights were recorded and representative section sof the liver and all gross lesions were preserved in 10% neutral-buffered formalin for possible future histopathologic examination.

Fetal:
Each viable fetus was examined externally, individually sexed, weighed and then euthanized. The detailed external examination of each fetus included, but was not limited to, an examination of the eyes, palate, and external orifices. Nonviable fetuses (if the degree of autolysis was minimal or absent) were examined, the crown-rump length measured, weighed and sexed. Crown-rump measurements and degrees of autolysis were recorded for late resorptions, if present.

Each viable fetus was subjected to visceral examination to include the heart and major blood vessels. The sex of each fetus was confirmed by internal examination. Fetal kidneys were examined and graded for renal papillae development. Headsfrom approximately half the fetuses in each litter were fixed for subsequent soft-tissue examination. The heads from the remaining half of the fetuses were examined by a mid-coronal slice. All carcasses were eviscerated, fixed in 100% ethanol, macerated in KOH and stained with Alizarin Red S and Alcian Blue.

Conclusion: Maternal toxicity in the 35 and 50 mg/kg bw/day groups consisted of mortalities, clinical findings, reductions in body weight gain and food consumption, macroscopic liver findings and increased liver weights. One female in the 10 mg/kg bw/day group also had macroscopic findings. Therefore, a dose level of 10 mg/kg bw/day was considered to be the LOAEL for maternal toxicity.

Developmental toxicity in the 35 and 50 mg/kg bw/day groups was expressed by an increase in postimplantation loss. Therefore, a dose level of 10 mg/kg bw/day was considered to be the NOAEL for developmental toxicity wneh 2-propen-1-ol was administered orally by gavage to pregnant rats.

Reliability: (1) valid without restriction
OECD TG Study

23-NOV-2005 (89)

Species: rat **Sex:** male/female
Strain: other:Crj:CD(SD)IGS
Route of administration: gavage
Exposure period: Males, 42 days. Females, from 14 days before mating to day 3 of lactation
Frequency of treatment: daily
Doses: 0 (vehicle), 2, 8 or 40 mg/kg bw/day
Control Group: yes, concurrent vehicle
NOAEL Maternal Toxity: 8 mg/kg bw
NOAEL Teratogenicity: 8 mg/kg bw

Method: other: OECD Guideline 421
Year: 2004
GLP: yes
Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), Supplied by SHOWA DENKO K.K.

Result:

[I]. Parental (Females) toxicity

(1) Mortality and clinical signs
-Mortality
---At all treated groups
No deaths were observed.

-Clinical signs

---At 40 mg/kg/day group
Salivation, decrease in locomotor activity, irregular respiration
(Total litter loss)
---At 2 and 8 mg/kg/day group
No abnormality

(2) Body weight and body weight gain

--At all treated groups
The body weight was similar with that of control group.

(3) Food consumption

The food consumption was similar with that of control group.

(4) Necropsy findings
See the tables

Table 1. Necropsy findings

-Female

Dose (mg/kg/day)	0	2	8	40
No. of animals	11	12	12	11
Thymus				
Atrophy	0	0	0	7
Liver				
Enlargement	0	0	0	8
Rough surface	0	0	0	1
Yellowish patch	0	0	0	7
Adrenal				
Whitish change	0	0	0	7

(5) Histological findings
See the tables

Table 2. Histological findings

-Female

Dose (mg/kg/day)	0	2	8	40
Thymus				
Atrophy	-	-	-	5/7
Liver				
Brown pigment deposition, perilobular	-	-	-	2/10
Clear cell change, hepatocyte, diffuse	-	-	-	8/10
Fibrosis, perilobular	-	-	-	8/10
Hypertrophy, hepatocyte, perilobular	-	-	-	5/10
Necrosis, hepatocyte, perilobular	-	-	-	8/10
Proliferation, bile duct	-	-	-	8/10
Ovary				
Hypertrophy, luteal cell	0/11	0/12	0/12	5/11*
Adrenal	-	-	-	0/7

*: Significant difference from control, p<0.05
**: Significant difference from control, p<0.01
-: Not tested

(No. animals with histological change)/(No. animals examined)

[II]. Developmental toxicity

(1) Morphology, body weight and necropsy findings of offspring

Table 3: Litter Size and Viability Index (F1)

Dose (mg/kg)		Total Number of Offspring at Birth			Number of Live Offspring at Birth		
		M	F	Total	M	F	Total
0	Mean	6.6	7.8	14.5	6.6	7.8	14.5
	S.D	2.2	1.9	1.9	2.2	1.9	1.9
	N	11	11	11	11	11	11
(M/F)		(73/86)			(73/86)		
2	Mean	7.0	6.8	13.8	7.0	6.8	13.8
	S.D	1.9	2.6	1.9	1.9	2.6	1.9
	N	12	12	12	12	12	12
(M/F)		(84/82)			(84/82)		
8	Mean	6.8	8.0	14.8	6.8	7.9	14.7
	S.D	2.0	2.5	0.9	2.0	2.4	0.7

	N	12	12	12	12	12	12
(M/F)		(81/96)			(81/95)		
	Mean	8.3	7.0	15.3	7.8	6.9	14.8
40	S.D	1.8	1.0	1.7	1.3	0.9	1.5
	N	12	12	12	12	12	12
(M/F)		(99/84)			(94/83)		

Dose (mg/kg)	Number of Live Offspring at Birth Before Culling			Viability Index (%)	
	M	F	Total	Day 0	Day 4
Mean	6.6	7.7	14.4	100.00	99.39
0 S.D	2.2	2.1	1.9	0.00	2.02
N	11	11	11	11	11
(M/F)	(73/85)				
Mean	7.0	3.8	13.8	100.00	100.00
2 S.D	1.9	2.6	1.9	0.00	0.00
N	12	12	12	12	12
(M/F)	(84/82)				
Mean	6.7	7.9	14.6	99.51	99.41
8 S.D	1.8	2.4	0.8	1.7	2.05
N	12	12	12	12	12
(M/F)	(80/95)				
Mean	6.8	6.4	13.2	96.97	89.48
40 S.D	2.4	2.1	4.4	4.85	28.50
N	12	12	12	12	12
(M/F)	(81/77)				

Table 4: Clinical Signs (F1 before Weaning)

Dose (mg/kg)	Day 0	1	2	3	4
Findings (M/F)					
Number of dams	11	11	11	11	11
0 Number of offspring	73/86/0	73/86	73/86	73/86	73/85
Number of dams with abnormal offspring	1	1	0	0	0
No abnormality	72/86	73/85	73/86	73/85	73/85
Death (M/F/U)	0/0/0	0/0	0/0	0/1	0/0
Loss of Suckling	1/0	0/1			
Number of dams	12	12	12	12	12
2 Number of offspring	84/82/0	84/82	84/82	84/82	84/82
Number of dams with abnormal offspring	0	0	0	0	0
No abnormality	84/82	84/82	84/82	84/82	84/82
Death (M/F/U)	0/0/0	0/0	0/0	0/0	0/0

	Number of dams	12	12	12	12	12
8	Number of offspring	81/96/0	81/95	81/95	80/95	80/95
	Number of dams with abnormal offspring	0	0	0	0	0
	No abnormality	81/95	81/95	80/95	80/95	80/95
	Death (M/F/U)	0/1/0	0/0	1/0	0/0	0/0
	Number of dams	12	12	11	11	11
40	Number of offspring	99/84/0	94/83	82/77	81/77	81/77
	Number of dams with abnormal offspring	3	0	0	0	0
	No abnormality	88/78	82/77	81/77	81/77	81/77
	Death (M/F/U)	5/1/0	12/6	1/0	0/0	0/0
	Loss of Suckling	6/5				

M: Male, F: Female, U: Unable to be sexed on day 0

Table 5: External Examination of Offspring (F1) on Day 0 (Birth day)

Dose (mg/kg)	0	2	8	40
No. of Dams	11	12	12	12
No. of Offspring	159	166	176	177
No. Dams with anomalous offspring	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
No. Offspring with any anomalies	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

Significantly different from control: * P<0.05; ** P<0.01

-Morphology
--At 40 mg/kg/day group
  Decrease in viability index on day 4 (not statistically)
  Total litter loss (from one dam)

-Body weight
--At all treated groups
  No toxicological significances

Table 6: Body Weight Gain of Male and Female Offspring (F1 before Weaning)

Dose (mg/kg)		Male day 0-4	Female day 0-4
0	Mean	3.3	3.2
	SD	0.3	0.4
	N	11	11
2	Mean	3.4	3.2
	SD	0.6	0.4
	N	12	12
8	Mean	3.8	3.6
	SD	0.4	0.4
	N	12	12
40	Mean	2.9	2.7

	SD	1.1	1.0
	N	11	11

-Necropsy findings
No toxicological significances

Table 7: Necropsy findings (F1 Offspring)

Dose (mg/kg)	Findings	Scheduled Sacrifice Day 4 M	F
0	Number of offspring examined	73	85
	No abnormality	73	85
2	Number of offspring examined	84	82
	No abnormality	83	82
	Liver	1	
	Yellow patch		
8	Number of offspring examined	80	95
	No abnormality	80	95
40	Number of offspring examined	81	77
	No abnormality	81	77

Dose (mg/kg)	Findings	Dead (M/F Day: 0	1	2	3	4
0	Number of offspring examined					0/1
	No abnormality					0/1
2	Number of offspring examined					
	No abnormality					
	Liver					
	Yellow patch					
8	Number of offspring examined	0/1				
	No abnormality	0/1				
40	Number of offspring examined	5/1	8/5	1/0		
	No abnormality	5/1	8/5	1/0		

M: Male, F: Female

The NOAEL for general toxicity and reproductive and developmental toxicity is
considered to be 8 mg/kg/day

Test condition: (1) Animals:
-At the start of administration
9 weeks old, Sprague-Dawley (Crj:CD(SD)IGS, SPF) rats (obtained from Charles River Japan, Inc.)
They were put in quarantine for 5 days beforehand.
Body weight; male 337-388 g, female 202-233 g
(2) Number of animals/group:
Males, 12;
Females, 12
(3) Rout:
Oral (gavage)
(4) Dosage:
0 (vehicle), 2, 8, 40 mg/kg/day
(5) Vehicle:
Water for injection, JP
(6) Confirmation of test solution concentration:
Test solutions were analyzed and warranted to be stable for 8 days.
(8) Administration period:
Males, 42 days from 14 days before mating and the mating period to the day before necropsy.
Females, from 14 days before mating to day 3 of lactation
(9) Terminal killing:
Males, day 43.
Females, day 4 of lactation.
(10) Statistics Analysis:
Bartlett's tests were initially performed. As the population was in equal variance on Bartlett's tests, one-way analysis of variance was performed. And as the population was not in equal variance on Bartlett's tests, Kruskal-Wallis's test was performed. In the case of the significant difference between dose groups, Dunnett's multiple comparison test was performed. Kruskal-Wallis's test were performed directly for some parameters. Wilcoxon rank-sum test was performed for histopathological findings. Fisher's exact test was performed for the other parameters. Five
percent as significant level was adopted for all tests.

Conclusion: The NOAEL for general toxicity and reproductive and developmental toxicity is considered to be 8 mg/kg/day.

Reliability: (1) valid without restriction
OECD TG Study

Flag: Critical study for SIDS endpoint

06-JAN-2006 (6)

Species: rat **Sex:** female
Strain: Sprague-Dawley
Route of administration: other: intraamniotic injection

Year: 1985
GLP: no

Test substance: other TS: 2-propen-1-ol (CAS No. 107-18-6), supplier: Aldrich Ltd, Montreal, Canada

Method: Timed-gestation pregnant Sprague-Dawley rats (225-250) were obtained from Charles River Canada Inc. The day on which spermatozoa were found in the vaginal smear was considered day zero of pregnancy. On day 13 the rats were laparotomized under anesthesia (ether) and the uteri
exposed. Embryos in one uterine horn received an intraamniotic

injection (10 ul; 30-gauge needle) of 2-propen-1-ol (10, 100 or 1000 ug/fetus in 0.9% NaCl in 5, 8 and 7 litters, respectively) while those in the other horn were untreated. Saline injected controls were also included in the study and treated in a similar manner (intraamniotically injected or sham treated). The uterus was repositioned and the laporotomy closed (nylon sutures).

Rats were sacrificed on day 20 of gestation (ether overdose) and the number of dead or resorbed fetuses recorded. Live fetuses were
examined for external malformations, blotted dry and weighed.

The results were analyzed using the Mann-Whitney U-Test.

Result: Approx. 24% and 12% of the saline-injected control fetuses and 12% of the sham control fetuses were resorbed; 6% and 5%, respectively, were malformed. Comment: The total number of control litters is reported as 18.

2-Propen-1-ol treatment caused a dose dependent increase in the incidence of resorbed fetuses, with significance found at 100 µg and 1000 µg/fetus (treated uterine horn versus untreated contralateral horn). While no tabulated results are available, interpolation from graphical data included in the publication indicates that the mean number of dead or resorbed fetuses was 0.3, 0.5 (P<O.05) and 0.6 (P<O.05) in the 10 (5 litters), 100 (8 litters) and 1000 (7 litters) ug/fetus groups.
Comment: The occurrence of dead or resorbed fetuses also increased in a treatment-related manner in the fetuses from the contra-lateral (untreated) uterine horn : <0.1, 0.2, 0.4 for the low, intermediate and high dose groups.

Two fetuses from 7 high dose litters were malformed (limb defects; non-significant). Two contralateral controls (untreated) from 8 intermediate dose litters were also malformed (omphalocele, edema, micromelia of the limbs, clubfoot, short neck and micrognathia; the other had a minor forelimb defect).

There was one maternal death at the high dose of 2-propen-1-ol the day after surgery and treatment of the embryos; this was the only evidence of maternal toxicity.

Conclusion: A treatment related increase in dead and resorbed fetuses was reported following intraamniotic injection of 10, 100 or 1000 ug 2-propen-1-ol/fetus on GD13. However the non-physiological route of exposure, together with an increased occurrence of dead/resorbed fetuses in the untreated (contra-lateral) uterine horn, suggests these observations to be of doubtful reliability for the purposes of hazard identification.

Reliability: (4) not assignable
19-DEC-2005 (147)

5.8.3 Toxicity to Reproduction, Other Studies

5.9 Specific Investigations

5.10 Exposure Experience

Type of experience: other: CASE REPORTS

Remark: Oral ingestion of 2-propen-1-ol by a 55-yr-old man resulted in death within 100 min. At autopsy, bloody, reddish fluid was found in mouth, larynx, esophagus, and trachea. The mucous membranes of the trachea, stomach, and duodenum were congested and inflamed. The stomach contained a pungent green-black fluid, and all internal organs exhibited a strong pungent odor. Toxicological analysis of blood identified 2-propen-1-ol. Total amounts of 2-propen-1-ol in gastric contents, bile, and urine were 3.6 g, 15 mg, and 0.5
mg, respectively. The concentration in blood was 309 mg/L. Acrolein was not detected in gastric contents and only in small amounts in bile and urine. The concentration of acrolein in blood was 7.2 mg/L. Death was attributed to acrolein-induced acute cardiotoxicity, similar to that previously documented in animal experiments.

Reliability: (2) valid with restrictions

Flag: Critical study for SIDS endpoint

25-NOV-2005 (155)

Type of experience: other: HUMAN EXPOSURE

Method: SENSORY RESPONSE STUDIES

These studies were carried out on groups of volunteers (numbers of the staff or medical students). The age range was 19 to 39, with a mean of 22. All participants was in apparent good health before and during the experimental period, which extended over 50 days. All of the exposed subjects were under the supervision of physician (C.H.H.). The eyes were visually inspected after each exposure. Physical examination of the chest was made at the conclusion
of the day's run, or when subjective complaints were elicited.

Group of five to seven subjects were exposed from one to three times a week for exactly five minutes, during which time they noted the degree of subjective response at one-minute intervals. Evaluation was made by checking: absent, slight, moderate, severe, or extreme.

The exposure room was specially designed for the purpose. It
was equipped with an entrance port, a one-way-view window, an exhaust fan capable of changing the air in one minute, and a revolving fan for mixing the vapors. The room was approximately cubical, with a volume of approximately 18,000
liters. Exposures were static, vapors being generated by flash vaporization from a heat source. Exactly five minutes was allowed for vaporization and equilibration before the subjects entered, and at least 10 minutes was allowed for evacuating the vapors after the subjects were dismissed.

Result: In no case was there any pulmonary discomfort or noticeable effect on the central nervous system. Eye irritation, occurring immediately , was not more than slight until the level of 25 ppm was reached, although nose irritation was regarded as at least moderate by four of seven subjects at 12.5 ppm. Olfactory cognition was checked off as more than moderate only at 6.25 ppm, and by only two of five subjects.

ppm	No. of subjects	Eye irritation		Nose irritation	
		A	B	A	B
0.78	6	0	0	2	0
6.25	6	1	0	3	1
12.5	7	1	0	7	4
25.0	5	5	5	5	5

ppm	No. of subjects	Pulmonary Discomfort		Olfactory Cognition	
		A	B	A	B
0.78	6	0	0	5	1
6.25	6	0	0	5	2
12.5	7	0	0	6	1
25.0	5	0	0	3	1

ppm	No. of subjects	CNS Effects	
		A	B
0.78	6	0	0
6.25	6	0	0
12.5	7	0	0
25.0	5	0	0

Note: Under A are listed the number showing any response at all; Under B, those listing higher responses than slight.

Reliability: (4) not assignable
06-JAN-2006 (39)

Type of experience: Human - Medical Data

Remark: The following medical procedures should be made available to each employee who is exposed 2-propen-1-ol at potentially hazardous levels: Initial Medical Screening: Employees should be screened for history of certain medical conditions which might place the employee at increased risk from 2-propen-1-ol exposure. Chronic respiratory disease: In persons with impaired pulmonary function, especially those with obstructive airway diseases, the breathing of 2-propen-1-ol might cause exacerbation of symptoms due to its irritant properties. Skin disease: 2-propen-1-ol can cause skin burns. Persons with existing skin disorders may be more susceptible to the effects of this agent. Liver disease: the

importance of the liver in the biotransformation and detoxification of foreign substances should be considered before exposing persons with impaired liver function. Kidney
disease: the importance of the kidney in the elimination of toxic substances justifies special consideration in those with impaired renal function. Eye disease: Because 2-propen-1-ol is reported to cause eye injury, those with existing
eye diseases may be at increased risk from exposure. Any employee developing the above-listed conditions should be referred for further screening.
SRP: 2-Propen-1-ol is the most commonly used experimental model for a hepatic periportal toxicant. The possibility of hepatic disease should be considered in humans exposed to 2-propen-1-ol because of its extreme periportal toxicity in experimental animals.

Reliability: (4) not assignable
09-MAY-2006 (91)

Type of experience: Human - Epidemiology

Remark: The breath of eight male volunteer subjects was analyzed to determine the levels of trace organic compounds in the respired air. 2-Propen-1-ol was found at 0.52 and 9.5 ug/hr in expired air from one subject that was a smoker and from one non-smoking subject, respectively. 2-Propen-1-ol was not found in the breath of the other 6 subjects. 2-Propen-1-ol was categorized as a chemical that is thought to result from or be related to normal human metabolism.

Reliability: (4) not assignable
19-DEC-2005 (33)

Type of experience: Human

Remark: HUMAN EXPOSURE:
Absorption through the skin leads to deep muscle pain, presumably due to spasm. Lacrimation, retro-bulbar pain, photophobia, and blurring of vision may be associated with exposure to vapors, and corneal injury has been described.
25-NOV-2005 (51)

Type of experience: Human

Remark: HUMAN EXPOSURE:
Absorbed through intact skin in toxic and even lethal concentration. Dermatitis of variable types and degrees results, in addition to first and second-degree burns with vesiculation. The vapors are especially irritating to the eyes and nose.
Ingestion of this material is most toxic, and severe symptoms can occur from contamination of food, cigarette, etc. The estimation fatal dose is 10 gm. The TLV is 2 ppm.

Reliability: (4) not assignable
05-DEC-2005 (16)

Type of experience: Human

Remark: HUMAN CLINICAL CASE:

In air moderately contaminated with 2-propen-1-ol (concentration unspecified), men complained of excessive secretion of tears, pain behind the eyes, sensitivity to light, and some blurring of vision.

Reliability: (4) not assignable
25-NOV-2005 (23)

Type of experience: Human

Remark: HUMAN EXPOSURE:
Vapors are quite irritating to eyes, nose, and throat. Eye irritation may be accompanied by complaints of photophobia and pain in the eyeball; pain may not begin until 6 hr after
exposure.

Reliability: (4) not assignable
25-NOV-2005 (119)

Type of experience: Human

Remark: SIGNS AND SYMPTOMS: Potential symptoms of overexposure are eye irritation, tissue damage; irritation of upper respiratory system and skin; pulmonary edema.

Reliability: (2) valid with restrictions
30-NOV-2005 (95)

Type of experience: Human

Remark: HUMAN CLINICAL CASE:
Volunteers have complained that 12.5 ppm was moderately irritating to the nose; only slight nasal irritation was reported at 0.8 ppm, the lowest concentration tested.

Reliability: (4) not assignable
25-NOV-2005 (23)

5.11 Additional Remarks

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