Workplace Exposure Standard (WES) review

1,1-DICHLOROETHANE
(CAS NO: 75-34-3)

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Te Kāwanatanga o Aotearoa
New Zealand Government

WORKSAFE
Mahi Haumaru Aotearoa

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1.0
Introduction

This WorkSafe New Zealand (WorkSafe) review considers whether the **WES** for 1,1-dichloroethane should be changed.

It considers the potential for exposures to 1,1-dichloroethane in New Zealand, the health effects and risks, exposure standards from other jurisdictions around the world, and the practicability of measuring exposures.

The review includes a recommendation to change the WorkSafe WES for 1,1-dichloroethane, which is currently set at a **WES-TWA** of 200 **ppm** (810 **mg/m³**) and **WES-STEL** of 250 ppm (1,010 mg/m³) as published in the special guide *Workplace Exposure Standards and Biological Exposure Indices*, 12th Ed., November 2020 (WorkSafe, 2020).

The WES recommended in this document are guidance values, not prescribed exposure standards. The intention is for them to be used as **risk criteria** for health risk assessment and risk management purposes and to be applied or interpreted only by people with appropriate training and experience. The values proposed in this document are considered by WorkSafe to be health-based WES. This means they are based on minimising health risk and do not take the practicability of achieving or measuring the values into consideration. This also means that in some instances the current analytical or sampling methods will not be sensitive enough to allow measurement at a level sufficiently below the WES to assess risk with a high degree of confidence. In this case there will be some uncertainty as to whether risk is suitably managed. As with any risk assessment, the uncertainties inherent in the assessment need to be considered and minimised so far as is reasonably practicable through good risk assessment and control.

We consider it critical to set health-based values as risk criteria, so that risk assessment is based on an actual understanding of health risk, rather than merely measuring a level of exposure.

Terms that are **bold** (first occurrence only) are further defined in the Glossary. Synonyms: Ethylidene chloride; Ethane, 1,1-dichloro-; 1,1-Ethylidene dichloride; 1,1-DCE.

2.0 Chemical and physical properties

1,1-Dichloroethane is a colourless, oily liquid with a chloroform-like odour (**ATSDR**, 2015; **ACGIH®**, 2001).

Chemical and physical properties of 1,1-dichloroethane include:

Formula	$C_2H_4Cl_2$
Molecular weight	98.97g/mol
Physical form	Colourless, oily liquid
Specific gravity	1.175 g/cm^3 at 20°C
Melting point	-96.9°C
Boiling point	57.3°C
Relative vapour density	3.44 (air = 1)
Vapour pressure	230 mmHg at 25°C
Flash point	Closed cup: -12°C; Open cup: 14°C
Autoignition temperature	457.8°C
Flammability limits	Lower: 5.4%; Upper: 11.4% by volume in air
Solubility	Water: 0.55 g/100 g at 20°C; miscible with oxygenated and chlorinated solvents
Partition coefficients	log KOW = 1.79 log KOC = 1.48
Conversion factors	1 ppm = 4.05 mg/m^3 at 25°C, 760 torr 1 mg/m^3 = 0.25 ppm at 25°C, 760 torr

ATSDR, 2015; ACGIH®, 2001

TABLE 1: Physicochemical properties of 1,1-dichloroethane

Health-related hazard classifications for 1,1-dichloroethane:

SUBSTANCE	CAS NUMBER	CLASSIFICATION
Ethane, 1,1-dichloro-	75-34-3	6.3B; 6.4A; 6.7B

TABLE 2: **HSNO** health-related hazard classifications of ethane, 1,1-dichloro- (**EPA**, 2020)

For a full listing of all HSNO health-related hazardous substances classification codes and their descriptions, see Appendix 2.

[All] Overall classification for that endpoint.

[O] Oral exposure route.

[D] Derman exposure route.

[I] Inhalation exposure route.

3.0
Uses

1,1-Dichloroethane is primarily used as an intermediate in the production of 1,1,1-trichloroethane, with lesser uses as a solvent; and, as a component of varnish and finish removers, fumigant and insect sprays (ATSDR, 2015; **AICIS**, 2015; ACGIH®, 2001).

Solvent uses of 1,1-dichloroethane include: for plastics, oils, and fats. Other uses of 1,1-dichloroethane include: organic synthesis, ore floatation, and in the manufacture of plastic wrap, adhesives, and synthetic fibres (ATSDR, 2015; AICIS, 2015).

Occupational exposure to 1,1-dichloroethane can occur during production, storage, transportation and end-use.

Workers can be exposed to 1,1-dichloroethane vapour and liquid via inhalation, and eye and skin contact.

The number of workers exposed or potentially exposed to 1,1-dichloroethane in New Zealand workplaces is unknown.

4.0
Health effects

IN THIS SECTION:

4.1 Non-cancer

Humans

The ATSDR review of 1,1-dichloroethane summarised toxicity in exposed humans:

> "Relatively little information is available on the health effects of 1,1-dichloroethane in humans or animals. Chlorinated aliphatics as a class are known to cause central nervous system depression and respiratory tract and dermal irritation when humans are exposed by inhalation to sufficiently high levels. In the past, 1,1-dichloroethane was used as an anesthetic; however, this use was discontinued due to the risk of cardiac arrhythmia induction in humans at anesthetic doses (approximately 26,000 ppm)." (References cited in ATSDR, 2015).

The PubChem Compound Summary for 1,1-dichloroethane notes:

> "Symptoms of exposure to this compound may include liver and kidney damage, skin and eye irritation, dermatitis, skin burns, unconsciousness, **CNS** depression, drowsiness, nausea, vomiting, faintness, irritation of the respiratory tract, salivation, sneezing, coughing, dizziness, lacrimation, reddening of the conjunctiva, cyanosis, and circulatory failure." (**NLM** PubChem, 2020).

The AICIS review of 1,1-dichloroethane summarised the irritation/corrosion potential:

> "Exposure to the chemical vapour has been reported to irritate the eyes, skin and respiratory tract. Effects reported following exposure to the chemical vapour were salivation, sneezing and coughing (**HSDB**)." (Reference cited in AICIS, 2015).

The New Zealand EPA classifies 1,1-dichloroethane as a 6.3B and 6.4A substance – a substance that is mildly irritating to the skin and irritating to the eye, respectively (EPA, 2020).

The AICIS review of 1,1-dichloroethane summarised the sensitisation potential in humans:

> "There is an alert for protein binding using the **OECD** Quantitative Structure Activity Relationship (**QSAR**) Application toolbox for nucleophilic substitution to alkyl halides. The electron deficient carbon attracts nucleophiles and reacts with proteins via substitution by a protein amino group on the carbon, with subsequent displacement of the halide ion (Roberts et. al., 2007). However, the known metabolic pathway of the compound does not yield reactive metabolites that could trigger this reaction (see Toxicokinetics).
>
> "QSAR modelling using **OASIS-TIMES** resulted in negative predictions for both the parent and metabolite, however the prediction was out of the total domain of the model." (Reference cited in AICIS, 2015).

The AICIS review of 1,1-dichloroethane summarised the repeat dose toxicity in exposed humans:

> "It has been reported that repeated or prolonged dermal exposure can cause burns, scaliness and rash in humans (HSDB). Details are not available." (Reference cited in AICIS, 2015).

The ATSDR review of 1,1-dichloroethane noted that no studies were located regarding reproductive or developmental toxicity in humans following exposure to 1,1-dichloroethane (ATSDR, 2015).

The ATSDR review of 1,1-dichloroethane noted that no studies were located regarding genotoxicity in humans following exposure to 1,1-dichloroethane (ATSDR, 2015).

Animals

The AICIS review of 1,1-dichloroethane summarised the acute toxicity in experimental animals:

> "The chemical has low acute inhalation toxicity.
>
> "The calculated median lethal concentration (**LC50**) was 53–65 **mg/L** (13,000–16,000 ppm) in rats and 70 mg/L (17,300 ppm) in mice (HSDB).
>
> "Prominent anaesthetic effects were observed in mice exposed to the chemical at 32–40 mg/L (8,000–10,000 ppm) for two hours (HSDB)." (Reference cited in AICIS, 2015).

The AICIS review of 1,1-dichloroethane noted that no studies were located regarding irritation/corrosion potential in experimental animals. (AICIS, 2015).

No studies were located regarding the sensitisation potential in experimental animals.

1,1-Dichloroethane is not classified as a sensitiser under **UN GHS** Classification (NLM PubChem, 2020).

The ATSDR review of 1,1-dichloroethane summarised the repeat dose toxicity in experimental animals:

> "A small number of animal studies have examined the toxicity and carcinogenicity of 1,1-dichloroethane; these studies have failed to conclusively identify the critical targets of toxicity. Nonneoplastic effects are limited to renal toxicity in cats, maternal and fetal toxicity in rats, and alterations in body weight gain. Crystal precipitations and obstruction in the renal tubule lumina and increases in serum urea and creatinine were observed in cats exposed to 500 ppm for 13 weeks followed by a 13-week exposure to 1,000 ppm for 13 weeks. However, these effects were not observed in rats, guinea pigs, or rabbits similarly exposed to 1,1-dichloroethane, and renal effects have not been observed following gavage administration of 764 or 950 **mg/kg/day** in rats or 2,885 or 3,331 mg/kg/day in mice 5 days/week for 78 weeks or in mice exposed to 465 mg/kg/day 1,1-dichloroethane in drinking water for 52 weeks. Kidney effects have also been observed in mice administered a lethal intraperitoneal injection of 1,1-dichloroethane; the effects included increased glucose and protein in the urine and tubular swelling. The toxicological significance of the nephrotoxicity observed in cats and the mice with regard to human health is not known given the small number of animals tested (cats), the lack of a nephrotoxic effect in other species and in other studies where 1,1-dichloroethane was administered orally.
>
> "The liver is the only other organ that has been examined in multiple studies; no hepatic effects have been reported following intermediate-duration inhalation exposure of rats, guinea pigs, rabbits, or cats, intermediate-duration oral exposure of mice, or chronic-duration exposure of rats and mice." (ATSDR, 2015).

The **US EPA** review of 1,1-dichloroethane noted:

> "Groups of 10 Sprague-Dawley rats, 10 Pirbright-White guinea pigs, 4 colored rabbits and 4 cats were exposed to 0 or 500 ppm of 1,1-dichloroethane (2,024 mg/m^3, assuming 25°C and 760 mmHg) for 6 hours/day, 5 days/week for 13 weeks (Hofmann *et al*., 1971). Each group was composed of an equal number of males and females (2 each for cats and rabbits, 5 each for guinea pigs and rats). Behavior and body weight were monitored in all species. Hematologic and urinalysis values, serum **ALT**, **AST**, and creatinine, and **BUN** were monitored in rats, rabbits and cats. Sulfobromophthalein excretion was tested in rabbits and cats. It was not clearly specified what endpoints were tested in guinea pigs. After 13 weeks of treatment, none of the species tested showed any clinical or biochemical changes attributable to treatment with 1,1-dichloroethane. The animals of the treated groups were then exposed to 1,000 ppm (4,047 mg/m^3) for an additional 10–13 weeks, while the control animals were maintained without exposure for the same period. Upon study termination, all animals were necropsied; relative liver and kidney weights were determined, and the liver, kidneys, and occasionally other selected organs (not specified) were processed for histopathological examination. No effects were reported in treated rats, rabbits or guinea pigs. Following the increase in concentration to 1,000 ppm, cats had reduced body weight gain and elevated BUN and serum creatinine levels relative to controls. In the cats at 1,000 ppm, BUN levels were elevated immediately and rose steadily until week 11 (week 24 of the whole study), at which time they reached a peak level that was approximately three-fold greater than the control level. Blood creatinine levels showed a parallel but less dramatic increase. No increases were noted in the activities of serum ALT or AST. One cat was removed from exposure due to poor general condition after 10 weeks at 1,000 ppm; for the remaining animals exposure terminated at week 13. Histopathological examination of the kidneys revealed renal tubular dilation and degeneration in 3 of the 4 treated cats. No information was provided regarding effects at the portal of entry (that is, pulmonary effects). 1,2-Dichloroethane, which was also tested in this study, appeared to be considerably more toxic than 1,1-dichloroethane. Identification of **NOAEL** and **LOAEL** values for renal effects in the cat study is problematic because the kidneys were not examined during the first exposure period. Although serum creatinine and urea were monitored throughout the study, and appearance of increased levels appeared to coincide with raising the exposure concentration from 500 to 1,000 ppm, it is not clear that these parameters are sufficiently sensitive to have revealed subtle renal damage that may have occurred during the 500 ppm exposure. The exposure of 1,000 ppm (4,047 mg/m^3) was a NOAEL for rats, guinea pigs and rabbits." (Reference cited in US EPA, 2006).

The ATSDR review of 1,1-dichloroethane summarised the reproductive toxicity in experimental animals:

> "The potential reproductive toxicity, immunotoxicity, and neurotoxicity of 1,1-dichloroethane have not been examined following inhalation, oral, or dermal exposure." (ATSDR, 2015).

The ATSDR review of 1,1-dichloroethane summarised the developmental toxicity in experimental animals:

> "A single developmental toxicity study reported retarded fetal development (delayed ossification of vertebrae) in rats at 6,000 ppm (7 hours/day on gestation days 6–15); an 11% decrease in maternal body weight gain and a decrease in maternal food consumption were also reported at this concentration." (ATSDR, 2015).

The AICIS review of 1,1-dichloroethane summarised genotoxic potential in experimental animals and *in vitro* test systems:

> "*In vitro* genotoxicity assays with the chemical indicated mixed results (positive or negative) and the single *in vivo* study indicated that the chemical covalently binds to nucleic acids and proteins in rat and mice organs. The weight of evidence from the available data do not indicate that the chemical has genotoxic potential.
>
> "The chemical gave mixed results in several *in vitro* assays (**WHO**, 2003; ATSDR, 2013):
>
> - positive in bacterial reverse mutation assay in several Salmonella typhimurium strains, with or without metabolic activation in Ames assays
> - non-mutagenic in *Saccharomyces cerevisae* in a gene mutation test, with or without metabolic activation
> - negative in mammalian cell assays, with no cell transformations in BALB/C-3T3 cells or induction of chromosomal aberrations in Chinese hamster lung fibroblasts
> - positive in Syrian hamster embryo cell transformation assay, with increased **DNA** viral transformations, and
> - increased DNA repair in rat and mouse hepatocytes.
>
> "Only one *in vivo* study was available. In this study, male rats and mice received a single intraperitoneal (**i.p.**) injection of the chemical at ~1.2 **mg/kg bw**. The chemical was observed to covalently bind to nucleic acids and proteins in the liver, lungs, kidneys and stomach of animals after 22 hours (ATSDR, 2013)." (References cited in AICIS, 2015).

Patlolla *et al*. (2005) reported investigating the genotoxic potential of 1,1-dichloroethane (i.p.) in the bone marrow cells of Swiss-Webster mice, using chromosomal aberrations (**CA**), mitotic index (**MI**), and micronuclei (**MN**) formation as endpoints, with significantly increased number of chromosomal aberrations and frequency of micronucleated cells in the bone marrow with increasing dose (100, 200, 300, 400 and 500 mg/kg b.w. i.p.). However, the test groups consisted of only 3 animals each.

4.2 Cancer

The International Agency for Research on Cancer [**IARC**] has no evaluation on the carcinogenic potential of 1,1-dichloroethane (IARC, 2020).

The US National Toxicology Program [**NTP**] Report on Carcinogens [**RoC**], Fourteenth Edition has no evaluation on the carcinogenic potential of 1,1-dichloroethane (NTP RoC, 2016).

The New Zealand EPA has classified 1,1-dichloroethane as a 6.7B substance – a substance that is a suspected human carcinogen (EPA, 2020).

Humans

The ATSDR review of 1,1-dichloroethane noted that no studies were located regarding exposure and carcinogenicity potential in humans (ATSDR, 2015).

Animals

The ATSDR review of 1,1-dichloroethane summarised the data on exposure and carcinogenicity in experimental animals:

> "The results of the bioassay conducted by **NCI** (1977) suggest carcinogenic effects induced by 1,1-dichloroethane in rats and mice. A significant positive dose-related trend was observed for the incidence of hemangiosarcomas and mammary adenocarcinomas in female rats, hepatocellular carcinoma in male mice, and endometrial stromal polyps in female mice. However, only the incidence of endometrial stromal polyps in female mice exposed to 3,331mg/kg/day, 5 days/week was significantly increased over the corresponding control animals. When only male mice surviving at least 52 weeks were examined, there was a significant increase in the incidence of hepatocellular carcinomas in the 2,885mg/kg/day group. There are several limitations to this study. Survival was poor in both treated and control animals, thereby limiting the validity of these results. Although survival was significantly lower in the exposed groups, it is not clear that the increase in mortality was treatment-related. Furthermore, there were no other treatment-related effects on body weight, clinical signs, or the incidence of non-neoplastic lesions. Because of the high mortality in both the treated and control animals, the authors concluded that not enough animals survived to be at risk for late-developing tumors. Thus, though the results of this bioassay suggest that 1,1-dichloroethane is carcinogenic to rats and mice, the evidence is not conclusive.
>
> "The carcinogenicity of 1,1-dichloroethane was also examined in mice exposed to 155 or 465mg/kg/day of the compound in the drinking water for 52 weeks (Klaunig *et al*. 1986). A two-stage carcinogenesis protocol was also employed in this study to assess the ability of 1,1-dichloroethane to act as a tumor promoter. Neither 1,1-dichloroethane-treated animals initiated with diethylnitrosamine (**DENA**) or animals treated with 1,1-dichloroethane without initiation showed a significant increase in the incidence of lung or liver tumors over their corresponding controls. However, the conclusion that 1,1-dichloroethane is not a tumor promoter may not be entirely justified since a maximal response was observed in terms of tumor incidence in the DENA-alone-treated mice (100% tumor incidence at 52 weeks). Therefore, an increase in the incidence of liver tumors due to 1,1-dichloroethane following DENA initiation, if it existed, could not have been detected. Furthermore, since measurement of water consumption and replenishment were only done once a week, there was no way to determine the extent, if any, evaporation contributed to loss of the test chemical and affected the reported level of exposure. However, precautions were taken to minimize the loss of test chemical during the 1-week period; amber bottles with Teflon stoppers and double sipper tubes were used. Since 1,1-dichloroethane is a volatile chemical, this may present a limitation to the interpretation of results obtained from drinking water administration.
>
> "The difference in results (for example, induction of liver tumors) between the NCI (1977) and Klaunig *et al*. (1986) studies may be due to the method of administration, vehicle, and/or doses used. The pharmacokinetics of 1,1-dichloroethane may vary considerably when administered in drinking

water *ad libitum* over a week as compared to bolus doses given in corn oil. Evidence obtained with carbon tetrachloride indicates that corn oil likely acts as a reservoir in the gut to delay and diminish the systemic absorption of the lipophilic chemical, while such a chemical is probably rapidly absorbed when ingested in water (Kim *et al*. 1990a, 1990b). Furthermore, the doses given to mice by gavage were approximately 6 times higher than the drinking water concentrations. Sufficient information is not available to assess the contributions of these factors to the apparently disparate responses.

"Milman *et al*. (1988) examined the carcinogenic potential of 1,1-dichloroethane in initiation and promotion assays. In partially hepatectomized Osborne-Mendel rats receiving a single gavage dose of 700 **mg/kg** 1,1-dichloroethane in corn oil followed by dietary exposure to phenobarbitol for 7 weeks, there were no alterations in gamma-glutamyltranspeptidase (**GGT**)-altered foci. However, in the promotion assay in which partially hepatecomized [sic] Osborne-Mendel rats received an intraperitoneal dose of diethylnitrosamine followed by gavage administration of 700 mg/kg 1,1-dichloroethane in corn oil 5 days/week for 7 weeks, there was an increase in the total number of GGT-altered foci." (References cited in ATSDR, 2015).

4.3 Absorption, distribution, metabolism and excretion

The AICIS review of 1,1-dichloroethane summarised the absorption, distribution, metabolism and excretion (**ADME**):

"Oral toxicity studies in animals and information from the chemical's former use as a gaseous anaesthetic agent in humans provide evidence that the chemical can be absorbed following oral and inhalation exposure. Following absorption in rats and mice, the chemical is distributed preferentially to the adipose tissues and to a lesser extent to the liver, kidneys, lungs and stomach tissues. The chemical's former use as an anaesthetic also indicates that it can be distributed to the central nervous system (CNS) (WHO, 2000; ATSDR, 2013).

"The chemical was administered orally at 700 mg/kg bw in rats and 1,800 mg/kg bw in mice. After 48 hours, the chemical was excreted (7% for rats and 29% for mice) in the urine and in expired air as carbon dioxide. A study in rats showed that the primary metabolic route was via the hepatic microsomal cytochrome P450 system. The metabolites identified were acetic acid (major metabolite), 2,2-dichloroethanol and mono- and dichloroacetic acid (WHO, 2000; ATSDR, 2013).

"Although no specific dermal absorption studies were available, the chemical was reported to penetrate the shaved abdominal skin of rabbits (ATSDR, 2013). The rate of absorption was not reported." (References cited in AICIS, 2015).

The ATSDR review of 1,1-dichloroethane noted that there are limited data to identify the critical targets of 1,1-dichloroethane toxicity or to elucidate the mode of action for the observed effects (ATSDR, 2015).

5.0 Exposure standards

IN THIS SECTION:

5.1 Other exposure standards

Table 3 below shows 1,1-dichloroethane exposure standards from around the world, as published by the Institute for Occupational Safety and Health of the German Social Accident Insurance (IFA, 2020).

JURISDICTION OR ADVISORY BODY	8-HOUR LIMIT VALUE		SHORT-TERM LIMIT VALUE	
	ppm	mg/m³	ppm	mg/m³
Australia	100	412		
Austria	100	400	400	1,600
Belgium[1]	100	412		
Canada – Ontario	100			
Canada – Québec	100	405		
Denmark[5]	100	412	200[3]	824[3]
European Union[2]	100	412		
Finland	100	410	250[3]	1,000[3]
France[4]	100	412		
Germany – AGS	100	410	200[3]	820[3]
Germany – DFG[5]	100	410	200[3]	820[3]
Hungary		412		
Ireland	100	412		
Italy[5]	100	412		
Japan – JSOH	100	400		
Latvia	100	412		
New Zealand	200	810	250	1,010
Poland		400		
Romania	100	412		
Singapore	100	405		
South Korea	100	405		
Spain[5]	100	412		
Sweden	100	412		
Switzerland	100	400	200	800
The Netherlands		400		800
Turkey	100	412		
USA – NIOSH	100	400		
USA – OSHA	100	400		
UK	100			

TABLE 3: Exposure standards for 1,1-dichloroethane from around the world

It is noted that the only organisations from whom we obtained information as to how and why they set occupational exposures standards on 1,1-dichloroethane were ACGIH®, **SEG**, DFG and Safe Work Australia.

[1] Additional indication "D" means that the absorption of the agent through the skin, mucous membranes or eyes is an important part of the total exposure. It can be the result of both direct contact and its presence in the air.

[2] Indicative Occupational Exposure Limit Value (**IOELV**).

[3] 15 minutes average value.

[4] Indicative statutory limit values **Skin**.

[5] Skin.

5.2 ACGIH®

The ACGIH® review of 1,1-dichloroethane concluded with recommendations that a **TLV-TWA** of 100 ppm (405 mg/m³) for occupational exposure to 1,1-dichloroethane, would minimise the potential for ocular and upper respiratory tract irritation and possible liver and kidney injury from chronic exposure (ACGIH®, 2001).

Rationale:

> "1,1-Dichloroethane has a low acute and chronic toxicity. Only after inhalation of high concentrations have anesthetic effects been observed in rats and mice (Mueller, 1925; Muralidhara *et al*., 1986). 1,1-Dichloroethane appears to have very limited potential to cause liver injury in rats, guinea pigs, rabbits, cats, and dogs (American Industrial Hygiene Association, 1971; Hofmann *et al*., 1971; Klaunig *et al*., 1986; Story *et al*., 1986; Milman *et al*., 1988). The CNS, liver, and kidneys are the target organs for 1,1-dichloroethane, with cats appearing as the most sensitive species (Hofmann *et al*., 1971). The lowest **NOEL** that has been established in a 13-week inhalation study is 500 ppm (Hofmann *et al*., 1971). From the existing data, it is not clear whether 1,1-dichloroethane is mutagenic, although a number of genotoxicity studies have reported positive results, such as sister-chromatid exchanges and DNA repair. The same is true for some short-term carcinogenicity tests that revealed equivocal results. In the only long-term carcinogenicity bioassay, there was no conclusive evidence for the carcinogenicity of 1,1-dichloroethane in rats and mice. 1,1-Dichloroethane is not teratogenic to rats and only slightly fetotoxic after exposure to 3,800 ppm.
>
> "Very limited data are available to estimate the toxicity of 1,1-dichloroethane to humans. It is reported to irritate the eyes and respiratory tract, producing salivation, sneezing and coughing. Anesthetic effects have been observed with associated dizziness, nausea, and vomiting. In severe and fatal cases at unknown concentrations, hepatic and renal injury occurred.
>
> "Based on the data from the animal studies with repeated inhalations, a TLV-TWA of 100 ppm is recommended. This value should minimize the potential risk for possible hepatic and renal injury and eye and upper respiratory tract irritation that may occur after acute and chronic exposure to 1,1-dichloroethane. The evidence of carcinogenicity in inconclusive for 1,1-dichloroethane (U.S. National Cancer Institute, 1978; Klaunig *et al*., 1986; Story *et al*., 1986).
>
> "Accordingly, a carcinogenicity notation of A4, Not Classifiable as a Human Carcinogen, is assigned. Sufficient data were not available to recommend Skin or **SEN** notations or a **TLV-STEL**." (References cited in ACGIH®, 2001).

5.3 SEG

The Scientific Expert Group on Occupational Exposure Limits [SEG] review of 1,1-dichloroethane recommended an 8-hour TWA of 100 ppm (412 mg/m³), but no **STEL**, with a "skin" notation as percutaneous absorption was expected to significantly increase total body burden (SEG, 1996).

Rationale:

> "Little is known of the toxicokinetics of 1,1-dichloroethane. The log $P_{o/w}$ of 1,2-dichloroethane, which has a skin notation (log $P_{o/w}$ = 1.5; Banerjee *et al*., 1980) and 1,1-dichloroethane (log $P_{o/w}$ = 1.9) are comparatively similar. Moreover, a high skin absorption has been calculated for 1,1-dichloroethane (Fiserova-Bergerova *et al*., 1990). Metabolism results in dechlorination and formation of conjugates that are excreted in the urine. A significant proportion

of inhaled 1,1-dichloroethane is exhaled unchanged. The acute toxicity of 1,1-dichloroethane is low, with reported 8h-LC50 values in mice and rats of 17,300 ppm (71,276 mg/m^3) for 2 hours and 16,000 ppm (65,900 mg/m^3) for 8 hours, respectively (Henschler, 1970).

"Exposure of rats, guinea pigs, rabbits and cats to atmospheres of 500 ppm (2,060 mg/m^3), 6 **h/d**, 5 **d/w** for 13 weeks, resulted in no overt adverse effects (Hofmann *et al*., 1971).

"Subsequent exposure of the same groups of animals to 1,000 ppm (4,120 mg/m^3) for an additional 13 weeks was tolerated by rats, guinea pigs and rabbits. In cats, a decrease in body weight gain and macroscopic and microscopic evidence of kidney damage were observed. This was attributed to biotransformation of dichloroethane to oxalic acid, resulting in formation of calcium oxalate crystals in the kidney tubules (Hofmann *et al*., 1971). In a study conducted by Dow Chemical (1971) no evidence of gross or haematological or histological changes were seen in rats, guinea pigs, rabbits and dogs exposed to 500 and 1,000 ppm (2,060 and 4,120 mg/m^3), 7h/d, 5d/w for 6 months.

"1,1-Dichloroethane was found to be mutagenic in the Ames test modified for volatile substances (Riccio *et al*., 1983; Milman *et al*., 1988) whereas the standard Ames assay and tests with *Asp. nidulans* gave a negative results (Simmons *et al*., 1977; Crebelli *et al*., 1988). DNA-repair was induced in hepatocytes isolated from rats and mice (Milman *et al*., 1988; Williams *et al*., 1989). Microsomes from liver, lung and stomach of rats and mice catalyse the formation of DNA-adducts *in vitro* and after i.p. application of 1,1-dichloroethane to rats and mice DNA-adducts were detected in liver, lung and stomach (Colacci *et al*., 1985). DNA-fragmentation was not detected in livers of BALB/c-mice after i.p. application (Taningher *et al*., 1991).

"1,1-Dichloroethane did not increase the transformation of BALB/c-3T3 cells (Tu *et al*., 1985; Milman *et al*., 1988) but enhanced the transformation rate induced by SA7-adenovirus in **SHE** cells (Hatch *et al*., 1983). In a short-term rat liver foci assay, 1,1-dichloroethane did not induce GGT-foci but enhanced the foci initiated by diethylnitrosamine (Milman *et al*., 1988).

"Gavage carcinogenicity studies have been performed in both rats and mice (NCI, 1978; Weisburger, 1977). The data are not considered to be adequate to allow the carcinogenicity of 1,1-dichloroethane to be evaluated.

"The data on mutagenicity and carcinogenicity are not considered to be adequate to draw a conclusion on these toxicological endpoints.

"1,1-Dichloroethane has been shown to be embryotoxic only at exposure levels in excess of those necessary to cause maternal toxicity (Schwetz *et al*., 1974).

"There are no human data available that are relevant for establishing occupational exposure levels.

"The study of Hofmann *et al*. (1971), indicating a NOAEL of 500 ppm (2,060 mg/m^3) for kidney damage in animals, was considered to be the best available basis for proposing occupational exposure limits. In view of the absence of human data, an uncertainty factor of 5 was considered appropriate." (References cited in SEG, 1996).

5.4 DFG

The Deutsche Forschungsgemeinschaft [DFG, German Research Foundation] re-evaluation of 1,1-dichloroethane confirmed a **MAK** value of 100 **mL/m³** (410 mg/m³), **Peak Limitation Category II** (excursion factor 2), with a **Carcinogen Category 4** notations, while changing the **Pregnancy Risk Group from D** to **C** (DFG MAK, 2007; DFG MAK, 2001; DFG MAK, 1972).

5.5 Safe Work Australia

In their draft proposal, Safe Work Australia has recommended an 8-hour TWA of 100 ppm (412 mg/m³) for 1,1-dichloroethane to protect for eye and upper respiratory tract irritation, liver and kidney injury in exposed workers (Safe Work Australia, 2019).

Rationale:

> "The critical effects are irritation of the eyes and upper respiratory tract and adverse effects on liver and kidney. No toxicological data in humans are available. In a 13 week inhalation study involving multiple animal species exposed six hours a day, five days a week, a NOEC of 500 ppm was reported based on hepatoxicity and kidney effects in cats (noted to be the sensitive species). Another similar study was conducted in rats, rabbits, dogs and guinea pigs for six months, reporting no adverse effects at up to 1,000 ppm (ACGIH, 2018).
>
> "A TWA of 100 ppm was derived by applying an uncertainty factor of five for interspecies difference to the **NOAEC** of 500 ppm in cats. The recommended TWA is considered sufficiently low to minimise the potential for ocular and upper respiratory tract irritation, liver and kidney injury in exposed workers." (Safe Work Australia, 2019).

6.0 Analytical methods for the assessment of airborne 1,1-dichloroethane

A common method to measure 1,1-dichloroethane exposure is using NIOSH Method 1003, Issue 3 (NIOSH, 2003).

Using this method an air sample of 0.5 to 15 litres is collected onto a solid sorbent tube of coconut shell charcoal using a flow rate of 0.01 to 0.2 litres per minute. Following desorption of the analyte using carbon disulphide, the sample is analysed using gas chromatography and flame ionisation detection.

With a limit of quantitation of 5.1µg per sample, this method can reliably determine 8-hour exposures as low as 1ppm.

7.0
Discussion

WorkSafe's WES for 1,1-dichloroethane has been unchanged since adoption in 1994.

The toxicological database reviewed above indicates that 1,1-dichloroethane is locally and systemically toxic to humans, causing skin, eye and respiratory tract irritation, CNS depression, and organ damage. 1,1-Dichloroethane is systemically toxic to experimental animals, causing CNS effects, liver and kidney damage, and tumours in female rats and male and female mice.

Based on the aforementioned documentation, informed by the conclusions of the ACGIH®, SEG and DFG reviews, and in particular the findings listed below, WorkSafe considers its current WES-TWA of 200 ppm (810 mg/m³) and WES-STEL of 250 ppm (1,010 mg/m³) for 1,1-dichloroethane, to be inadequate to manage health risks from possible workplace exposure:

- 1,1-Dichloroethane is a potential irritant and toxicant in exposed workers inducing skin, eye and respiratory tract irritation, and liver and kidney damage (ACGIH®, 2001).
- The ACGIH® review of 1,1-dichloroethane concluded with a recommendation that a TLV-TWA of 100 ppm (405 mg/m³) would minimise the potential for ocular and upper respiratory tract irritation and possible liver and kidney injury from chronic exposure (ACGIH®, 2001).
- The SEG review of 1,1-dichloroethane recommended an 8-hour TWA of 100 ppm (412 mg/m³), but no STEL, with a "skin" notation as percutaneous absorption was expected to significantly increase total body burden. The TWA was based on the study of Hofmann *et al*. (1971), indicating a NOAEL of 500 ppm (2,060 mg/m³) for kidney damage in animals, with an uncertainty factor of 5 to extrapolate to humans (SEG, 1996).
- The DFG re-evaluation of 1,1-dichloroethane recommended a MAK value of 100 ppm (410 mg/m³), Peak Limitation Category II (excursion factor 2), with a Carcinogen Category 4 notations, while changing the Pregnancy Risk Group from D to C (DFG MAK, 2007; DFG MAK, 2001; DFG MAK, 1972). The MAK value was based on the NOAEL of 500 ppm from Hofmann *et al*. (1971) (DFG MAK, 1972).
- The Safe Work Australia draft review of 1,1-dichloroethane recommended a TWA of 100 ppm (412 mg/m³) to protect for eye and respiratory tract irritation, liver and kidney injury in exposed workers, based on a NOAEC of 500 ppm in exposed cats (SafeWork, 2019).
- The proposed WES-TWA of 100 ppm (405 mg/m³) of 1,1-dichloroethane is intended to protect exposed workers from potential ocular and upper respiratory tract irritation, and possible liver, kidney, and CNS effects (ACGIH®, 2001).
- A ***skin*** notation does not appear justified for 1,1-dichloroethane, due to the lack of reported data on the potential significance of dermal absorption from contact with 1,1-dichloroethane (ACGIH®, 2001).
- Available information indicates that 1,1-dichloroethane is not a sensitiser (ACGIH®, 2001), and ***dsen*** and ***rsen*** notations are not warranted.

8.0 Recommendations

WorkSafe considers its current WES-TWA of 200 ppm (810 mg/m^3) to be inadequate to protect workers exposed in the workplace, based on current knowledge.

It is proposed that WorkSafe:

1. adopt a WES-TWA for 1,1-dichloroethane of 100 ppm (405 mg/m^3)
2. retain a WES-STEL for 1,1-dichloroethane of 250 ppm (1,010 mg/m^3).

Noting that the proposed WES-TWA for 1,1-dichloroethane may not eliminate all risk, due to the limited toxicological database, so workplace exposures should be minimised.

Appendices

IN THIS SECTION:

Appendix 1: Glossary

TERM	MEANING
ACGIH®	The American Conference of Governmental Industrial Hygienists (ACGIH®) is a member-based organisation, established in 1938, that advances occupational and environmental health. Examples of this include their annual edition of the TLVs® and BEIs® book and work practice guides. Store at: https://portal.acgih.org/s/store#
ADME	Absorption, Distribution, Metabolism and Excretion.
AGS	Ausschuss für Gefahrstoffe [Committee for Hazardous Substances] is a consultative body of the German Federal Ministry of Labour and Social Affairs on issues of the Ordinance on Hazardous Substances. Administered by the BAuA.
AICIS	Australian Industrial Chemicals Introduction Scheme – the regulatory scheme that administers the Australian law regulating the importation and manufacture of industrial chemicals in Australia. AICIS replaced the National Industrial Chemicals Notification and Assessment Scheme (**NICNAS**) on 1 July 2020.
ALT; ALAT	Alanine transaminase – also called alanine aminotransferase.
AST; ASAT	Aspartate transaminase – also called aspartate aminotransferase.
ATSDR	Agency for Toxic Substances and Disease Registry is a federal public health agency of the US Department of Health and Human Services.
BAT	Biologische Arbeitsstoff-Toleranzwerte [Biological Tolerance Value], a DFG term.
BUN	Blood urea nitrogen.
CA	Chromosomal aberration.
Carcinogen category 4	DFG MAK designation: Substances that cause cancer in humans or animals or that are considered to be carcinogenic for humans and for which a MAK value can be derived. A nongenotoxic mode of action is of prime importance and genotoxic effects play no or at most a minor part provided the MAK and **BAT** values are observed. Under these conditions no contribution to human cancer risk is expected. The classification is supported especially by evidence that, for example, increases in cellular proliferation, inhibition of apoptosis or disturbances in cellular differentiation are important in the mode of action. The classification and the MAK and BAT values take into consideration the manifold mechanisms contributing to carcinogenesis and their characteristic dose-time-response relationships.
CNS	Central nervous system.
DENA	Diethylnitrosamine.
DFG	Deutsche Forschungsgemeinschaft (German Research Foundation), the Permanent Senate Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area, Federal Republic of Germany. The science-based MAK values are recommended to the German Minister of Labour and Social Affairs for possible adoption under the German Hazardous Substances Ordinance.
DNA	Deoxyribonucleic acid.
dsen	A substance that can 'sensitise' the skin, inducing a state of hypersensitivity to it, so that on subsequent exposures, an allergic reaction can occur (which would not develop in non-sensitised individuals). It is uncommon to become sensitised to a compound after just a single reaction to it. A WorkSafe term.
d/w	Days per week.
EPA	The New Zealand Environmental Protection Authority.
γGT; gamma-GT; GGT	Gamma-glutamyltranspeptidase.
GHS	Globally Harmonized System of Classification and Labelling of Chemicals.
h/d	Hours per day.
HSDB	Hazardous Substances Data Bank, administered by the US National Library of Medicine.
HSNO	Hazardous Substances and New Organisms Act 1996, New Zealand.
IARC	The International Agency for Research on Cancer – an agency of the World Health Organisation.

TERM	MEANING
IFA	Institut für Arbeitsschutz der Deutschen Gestzlichen Unfallversicherung [Institute for Occupational Safety and Health of the German Social Accident Insurance].
IOELV	Indicative Occupational Exposure Limit Value (health-based, **SCOEL** parameter).
i.p.	Intraperitoneal.
JSOH	Japan Society for Occupational Health.
LC_{50}	Lethal Concentration for 50% of the test population.
LOAEL	Lowest Observed Adverse Effect Level.
MAK	Maximale Arbeitsplatz-Konzentration, (maximum workplace concentration) is defined as the maximum concentration of a chemical substance (as gas, vapour or particulate matter) in the workplace air which generally does not have known adverse effects on the health of the employee nor cause unreasonable annoyance (for example, by a nauseous odour) even when the person is repeatedly exposed during long periods, usually for 8 hours daily but assuming on average a 40-hour working week. Values set by the DFG.
mg/kg	Milligrams per kilogram.
mg/kg b.w. or mg/kg bw	Milligram of substance per kilogram body weight.
mg/L	Milligrams of a substance per litre.
mg/m^3	Milligrams of substance per cubic metre of air.
MI	Mitotic Index.
mL/m^3 or ml/m^3	Millilitres of substance per cubic metre (of air).
MN	Micronuclei.
NCI	US National Cancer Institute.
NICNAS	National Industrial Chemicals Notification and Assessment Scheme is the Australian government's regulatory body for industrial chemicals.
NIOSH	The National Institute for Occupational Safety and Health is the United States federal agency responsible for conducting research and making recommendations for the prevention of work-related injury and illness.
NLM	National Library of Medicine, administered by the US National Institutes of Health.
NOAEC	No Observed Adverse Effect Concentration.
NOAEL	No Observed Adverse Effect Level.
NOEL	No Observed Effect Level
NTP	National Toxicology Program, US Department of Health and Human Services.
OASIS-TIMES	OASIS tissue metabolism simulator software.
OECD	Organisation for Economic Co-operation and Development.
OSHA	Occupational Safety and Health Administration, US Department of Labor.
Peak limitation category 2 or II	Substances with systemic effects; Excursion factor = 2 [default]; Duration 15 minutes, average value; Number per shift = 4; Interval = 1 hour. A DFG term.
ppm	Parts of vapour or gas per million parts of air.
Pregnancy Risk Group C	Damage to the embryo or foetus is unlikely when the MAK value or the BAT value is observed. A DFG term.
Pregnancy Risk Group D	Either there are no data for an assessment of damage to the embryo or foetus or the currently available data are not sufficient for classification in one of the groups A – C. A DFG term.

TERM	MEANING
QSAR	Quantitative structure-activity relationship.
Risk criteria	The terms of reference against which the significance of risk is evaluated. It is used to support decision making on health risk management. ISO 31000 2nd edition *Risk Management– guidelines* (2018).
RoC	Report on Carcinogens.
rsen	A substance that can 'sensitise' the respiratory system, inducing a state of hypersensitivity to it, so that on subsequent exposures, an allergic reaction can occur (which would not develop in non-sensitised individuals). It is uncommon to become sensitised to a compound after just a single reaction to it. A WorkSafe term.
SCOEL	The Scientific Committee on Occupational Exposure Limits is a committee of the European Commission, established in 1995 to advise on occupational health limits for chemicals in the workplace within the framework of Directive 98/24/EC, the chemical agents directive, and Directive 90/394/EEC, the carcinogens at work directive.
SEG	The Scientific Expert Group on Occupational Exposure Limits [SEG] was a committee of the European Commission, established in 1995 to advise on occupational health limits for chemicals in the workplace within the framework of Directive 98/24/EC, the chemical agents directive, and Directive 90/394/EEC, the carcinogens at work directive. The Scientific Committee on Occupational Exposure Limits [SCOEL] has replaced SEG.
SEN	A notation indicating the substance is a sensitiser. DSEN and RSEN are used in place of SEN when specific evidence of sensitisation by the dermal or respiratory route, respectively, is confirmed by human or animal data. An ACGIH® term.
SHE	Syrian Hamster Embryo.
skin	Skin absorption – applicable to a substance that is capable of being significantly absorbed into the body through contact with the skin. A WorkSafe term.
Skin	A notation indicating the potential for significant contribution to the overall exposure, by the cutaneous route, including mucous membranes and the eyes, by contact with vapours, liquids and solids. An ACGIH® term.
STEL	Short-Term Exposure Limit. The STEL is a limit value above which exposure should not occur and usually relates to a 15-minute reference period.
TLV®	Threshold Limit Value (see TLV-STEL and TLV-TWA below). An ACGIH® term. Please see the Statement of Position Regarding the TLVs® and BEIs® and Policy Statement on the Uses of TLVs® and BEIs®
TLV-STEL	TLV-Short-Term Exposure Limit; a 15 minute TWA exposure that should not be exceeded at any time during a workday, even if the 8-hour TWA is within the TLV-TWA. An ACGIH® term.
TLV-TWA	TLV – Time-Weighted Average; the TWA concentration for a conventional 8-hour workday and a 40-hour workweek, to which it is believed that nearly all workers may be repeatedly exposed to, day after day, for a working lifetime without adverse effect. An ACGIH® term.
UN	United Nations.
US EPA	United States Environmental Protection Agency.
WES	Workplace Exposure Standard – WESs are values that refer to the airborne concentration of substances, at which it is believed that nearly all workers can be repeatedly exposed to, day after day, without coming to harm. The values are normally calculated on work schedules of five shifts of eight hours duration over a 40 hour week. A WorkSafe term.
WES-STEL	The 15-minute time-weighted average exposure standard. Applies to any 15-minute period in the working day and is designed to protect the worker against adverse effects of irritation, chronic or irreversible tissue change, or narcosis that may increase the likelihood of accidents. The WES-STEL is not an alternative to the WES-TWA; both the short-term and time-weighted average exposures apply. Exposures at concentrations between the WES-TWA and the WES-STEL should be less than 15 minutes, should occur no more than four times per day, and there should be at least 60 minutes between successive exposures in this range. A WorkSafe term.
WES-TWA	The average airborne concentration of a substance calculated over an eight-hour working day. A WorkSafe term.
WHO	World Health Organisation, Geneva.

Appendix 2: HSNO health-related hazardous substance classifications

This is the full list of all health-related hazardous substances classifications that are listed by the NZ EPA, including those that apply to this substance.

CLASSIFICATION CODE	MEANING
Acutely toxic	
6.1A	Substances that are acutely toxic – Fatal
6.1B	Substances that are acutely toxic – Fatal
6.1C	Substances that are acutely toxic – Toxic
6.1D	Substances that are acutely toxic – Harmful
6.1E	Substances that are acutely toxic – May be harmful, aspiration hazard
Skin irritant	
6.3A	Substances that are irritating to the skin
6.3B	Substances that are mildly irritating to the skin
Eye irritant	
6.4A	Substances that are irritating to the eye
Sensitisation	
6.5A	Substances that are respiratory sensitisers
6.5B	Substances that are contact sensitisers
Mutagens	
6.6A	Substances that are known or presumed human mutagens
6.6B	Substances that are suspected human mutagens
Carcinogens	
6.7A	Substances that are known or presumed human carcinogens
6.7B	Substances that are suspected human carcinogens
Reproductive/developmental toxicants	
6.8A	Substances that are known or presumed human reproductive or developmental toxicants
6.8B	Substances that are suspected human reproductive or developmental toxicants
6.8C	Substances that produce toxic human reproductive or developmental effects on or via lactation
Target organ toxicants	
6.9A	Substances that are toxic to human target organs or systems
6.9B	Substances that are harmful to human target organs or systems
Skin corrosive	
8.2A	Substances that are corrosive to dermal tissue (UN PGI)
8.2B	Substances that are corrosive to dermal tissue (UN PGII)
8.2C	Substances that are corrosive to dermal tissue (UN PGIII)
Eye corrosive	
8.3A	Substances that are corrosive to ocular tissue

Source: www.epa.govt.nz/industry-areas/hazardous-substances/rules-for-hazardous-substances/hazardous-substances-classification-codes

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