MICHIGAN DEPARTMENT OF ENVIRONMENT, GREAT LAKES, AND ENERGY

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INTEROFFICE COMMUNICATION

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TO: File for 1,1,2-Trichloroethane (CAS #79-00-5)

FROM: Doreen Lehner, Toxics Unit, Air Quality Division

DATE: October 6, 2020

SUBJECT: Screening Level for 1,1,2-Trichloroethane (CAS #79-00-5)

Summary

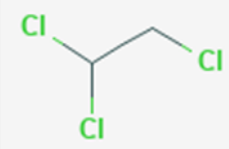
The initial risk screening level (IRSL) for 1,1,2-trichloroethane (CAS #79-00-5) is 0.063 µg/m^3 and the SRSL is 0.63 µg/m^3 based on an annual averaging time. The initial threshold screening level (ITSL) for 1,1,2-trichloroethane (CAS #79-00-5) is 11 µg/m^3 based on an annual averaging time. A second acute ITSL for 1,1,2-trichloroethane of 160 µg/m^3 based on a 24-hour averaging time. The acute ITSL is derived as a short-term exposure and will be used in conjunction with the more chronic ITSL.

Uses and Physical Chemical Properties

1,1,2-Trichloroethane is used as a solvent and as an intermediate in the production of 1,1-dichloroethane.

1,1,2-trichloroethane is an organochlorine compound with a molecular weight of 133.41 g/mol and can dissolve in water and evaporates easily.

Table 1. Physical/Chemical Properties of 1,1,2-Trichloroethane

Structure	
CAS Number	79-00-5
Synonyms	1,1,2-TCE, ethane trichloride, β-trichloroethane, vinyl trichloride
Appearance/Odor	Clear, colorless, sweet-smelling liquid
Melting Point	-36.6°C
Boiling Point	113.8°C
Vapor Pressure	23 mmHg at 25°C
Solubility	4900 mg/L at 25°C
LogP (octanol/water)	1.89

Literature Search

The following references or databases were searched to identify data to determine the screening level: U.S. Environmental Protection Agency (EPA) Integrated Risk Information System (IRIS), Registry for Toxic Effects of Chemical Substances (RTECS), American Conference of Governmental and Industrial Hygienists (ACGIH) Threshold limit Values (TLVs). International Agency for Research on Cancer (IARC) Monographs, Chemical Abstract Service (CAS) SciFinder (searched 5/26/2020), U.S. EPA ChemView, California Office of Environmental Health Hazard Assessment (OEHHA), and the U.S. Department of Health and Human Services Agency for Toxic Substances and Disease Registry (ATSDR).

Toxicokinetics

ATSDR (2019) reviewed the available data on the comparison of the absorption efficiency of 1,1,2-trichloroethane in the GI tract and lung. ATSDR (2019) stated that, "Studies in humans indicate that 1,1,2-trichloroethane is absorbed rapidly after inhalation exposure (Morgan et al. 1970, 1972). In one of the studies (Morgan et al. 1970), a volunteer took one breath of radiolabeled 1,1,3-trichloroethane and expired 2-3% of the inspired dose in the alveolar air after 12 seconds and about 0.5% after 40 seconds of breath-holding. More than 90% of the administered dose was retained in the body after 50 minutes. These data indicate that 1,1,2-trichloroethane was extensively absorbed into the bloodstream, supported by a blood-air partition coefficient (K_D) of 44.2. Gargas et al. (1989) determined a blood:air partition coefficient in humans of 35.7."

ATSDR (2019) stated that, "Rats and mice exposed to 1,1,2-trichloroethane under closed chamber conditions at 100 ppm 6 hours/day, 5 days/week for 4 weeks showed significant concentrations of 1,1,2-trichloroethane in the blood, indicating that 1,1,2-trichloroethane is extensively absorbed in both species (Sapphire Group, 2003). This is supported by the identification of a partition coefficient for rats of 58.0 (Gargas et al. 1989)."

1,1,2-Trichloroethane absorption via inhalation above was compared against the absorption in the GI tract. "…[R]ats were administered 1,1,2-trichloroethane via gavage in corn oil at 92 mg/kg/day or in water at 1.7 mg/kg/day for 5 days. Similarly, mice were treated at 390 mg/kg/day in corn oil or 10 mg/kg/day in water. Significant concentrations of 1,1,2-trichloroethane were detected in the blood, indicating that 1,1,2-trichloroethane is well absorbed in both species. Maximal blood concentrations were observed in rats on day 1 (up to 17 µg/g) and in mice on days 3 and 5 (8.5 to 25 µg/g) (Sapphire Group 2003). The only other data available in animals showed that oral doses near the maximum tolerated dose in mice (300 mg/kg) or rats (70 mg/kg) were 81% metabolized, indicating that at least this amount was absorbed (Mitoma et al. 1985). This suggests that 1,1,2-trichloroethane, like other structurally related halocarbons is well absorbed from the gastrointestinal tract of animals, and probably humans as well" (ATSDR, 2019).

Carcinogenicity

Genotoxicity

1,1,2-Trichloroethane has been tested for genotoxicity *in vivo*. The table below was taken from ATSDR (2019) and shows that there were mixed results in the available *in vivo* tests. Foureman et al. (1994) showed a negative result in sex-linked recessive lethal mutations in *Drosophila melanogaster* after fed 1,1,3-trichloroethane, but Vogel and Nivard (1993) showed a positive result in mitotic recombination in *D. melanogaster* after inhalation. Crebelli et al. (1999) found no effect of 1,1,2-trichloroethane in mouse micronuclei in bone marrow cells after intraperitoneal (i.p.) injection and no DNA damage (unwinding of the DNA) was seen in mouse hepatocytes after i.p. injection of 1,1,2-trichloroethane (Taningher et al., 1991). However, Mazzullo et al., 1986 found DNA adduct formation in mouse liver DNA after i.p. injection which was seen to a greater extent in mice than in rats given i.p. 1,1,2-trichloroethane. Mirsalis et al. (1989) showed that mice given 1,1,2-trichloroethane via gavage showed induction of S-phase DNA synthesis in hepatocytes and Miyagawa et al. (1995) found an increase in replicative DNA synthesis in mouse hepatocytes after gavage with 1,1,3-trichloroethane (ATSDR, 2019). Taken as a whole, 1,1,2-trichloroethane is genotoxic.

Table 2. Genotoxicity of 1,1,2-Trichloroethane *In Vivo**

Species (exposure route)	Endpoint	Results	Reference
Drosophila melanogaster (feed)	Sex-linked recessive lethal	–	Foureman et al. 1994
D. melanogaster (inhalation)	Mitotic recombination	+	Vogel and Nivard 1993
Mouse (intraperitoneal)	Micronuclei in bone marrow cells	–	Crebelli et al. 1999
Mouse (intraperitoneal)	DNA damage (unwinding) in hepatocytes	–	Taningher et al. 1991
Rat (intraperitoneal)	DNA adduct formation with liver DNA	+	Mazzullo et al. 1986
Mouse (intraperitoneal)	DNA adduct formation with liver DNA	+	Mazzullo et al. 1986
Mouse (gavage)	Unscheduled DNA synthesis in hepatocytes	–	Mirsalis et al. 1989
Mouse (gavage)	S-phase DNA synthesis in hepatocytes	+	Mirsalis et al. 1989
Mouse (gavage)	Replicative DNA synthesis in hepatocytes	+	Miyagawa et al. 1995

+ = positive results; – = negative results; DNA = deoxyribonucleic acid
*Table 2-4 from ATSDR (2019), page 48.

Key Study

The EPA (1987) set an inhalation unit risk of 1.6×10^{-5} per µg/m^3 based on a National Cancer Institute (NCI, 1978) study in rats and mice. Groups of 50 male and female B6C3F1 mice and Osborne-Mendel rats were exposed to technical grade (92.7%) 1,1,2-trichloroethane by gavage 5 days/week for 78 weeks. During the study, the doses for the rats and mice were increased.

"Low and high dose mice received 150 and 300 mg/kg body weight, respectively, for 8 weeks, followed by 200 and 400 mg/kg, respectively, for 70 weeks, followed by 12-13 weeks without treatment, after which the experiment was terminated. The time-weighted doses were 195 and 390 mg/kg, respectively. Untreated control and vehicle control groups were included (20 animals/sex/group)" (OEHHA, 2009).

"Low and high dose rats received 35 and 70 mg/kg body weight, respectively, for 20 weeks, followed by 50 and 100 mg/kg, respectively, for 58 weeks, followed by 34-35 weeks without treatment, after which the experiment was terminated. The time-weighted average doses were 46 and 92 mg/kg, respectively. Untreated control and vehicle control groups were included (20 animals/sex/group)" (OEHHA, 2009).

Critical Effect

"No statistically significant increase in 1,1,2-trichloroethane-induced tumor incidence was noted in either male or female rats. Increases in hepatocellular carcinoma incidence were noted in all male and female mouse 1,1,2-trichloroethane-exposed treatment groups. The Fisher exact test comparing tumor incidences of dosed to control groups and the Cochran-Armitage test for positive dose-related trend indicated a highly significant association ($p < 0.001$) between hepatocellular carcinomas and 1,1,2-trichloroethane exposure. A positive dose-related association between 1,1,2-trichloroethane exposure and adrenal gland pheochromocytoma incidence in male and female mice was also indicated by the Cochran-Armitage test ($p = 0.003$ for males, $p < 0.001$ for females). Fisher exact tests confirmed these results for high dose female mice ($p= 0.006$) but not for other mouse treatment groups" (OEHHA, 2009).

Derivation of IRSL/SRSL

The inhalation unit risk factor was derived from the male mouse hepatocellular carcinoma tumor data. The doses were adjusted from 5 days/week to 7 days/week. Then a linearized multistage model was used to calculate an oral slope factor of 5.7E-2 per (mg/kg)/day and derive an inhalation unit risk value of 1.6×10^{-5} per µg/m^3 based on a route-to-route extrapolation (Sapphire Group, 2004).

Rule 231(1) is used to develop an IRSL for 1,1,2-trichloroethane. The equation is below:

$$IRSL = \frac{1\ x\ 10^{-6}}{Unit\ risk} = \frac{1\ x\ 10^{-6}}{1.6\ x\ 10^{-5}} = 0.0625\ {}^{\mu g}/_{m^3} \approx 0.063\ {}^{\mu g}/_{m^3}$$

Rule 231(3) states that the averaging time for IRSLs and SRSLs is an annual averaging time. The initial risk screening level (IRSL) for 1,1,2-trichloroethane (CAS #79-00-5) is 0.063 µg/m^3 and the SRSL is 0.63 µg/m^3 based on an annual averaging time.

Non-Carcinogenic Response

Key Study (Annual ITSL)

ATSDR developed an intermediate inhalation exposure Minimal Risk Level (MRL) of 0.002 ppm based on a study by Kirkpatrick (2002). In the Kirkpatrick (2002) study, ten F344 CDF Crl:BR rats/sex/dose group (with satellite groups for pharmacokinetic evaluations) were exposed whole body to either 0,15,40, or 100 ppm for 6 hours/day 5 days/week for 13 weeks. The "rats exposed at ≥ 40 ppm for 13 weeks showed significantly increased incidences of lesions in the olfactory epithelium of the nasal turbinates, including atrophy, vacuolization and microcyst formation, and respiratory epithelial metaplasia compared to control rats" (ATSDR, 2019). Rats exposure at 100 ppm showed histopathological changes in the liver (hepatocellular vacuolization).

Critical Effect

ATSDR performed benchmark dose modeling (BMD) to identify a point of departure (POD) using "concentrations adjusted for intermittent exposure and incidence data for vacuolization/ microcyst formation and atrophy in the olfactory epithelium. Concentrations of 0, 15, 40, and 100 ppm were adjusted for intermittent exposure (6 hours/24 hours and 5 days/ 7 days) resulting in adjusted concentrations of 0, 2.7, 7.1, and 17.9 ppm. The data were fit to all available dichotomous models in EPA's Benchmark Software (BMDS version 2.6.0). A BMR of 10% was selected in the absence of data that would support a lower BMR. In accordance with EPA (2012) guidance, BMCs and BMCLs (95% lower confidence limit on the benchmark concentration) associated with an extra risk of 10% are calculated for all models" (ATSDR, 2019). The Log-Logistic model was selected as the best model for vacuolization/microcyst formation data. The POD value of 0.57 ppm was converted to a human equivalent concentration of 0.07 ppm. A total uncertainty factor (UF) of 30 (UF of 3 for extrapolation from animal to humans and an UF of 10 for human variability) was used. This results in an intermittent inhalation exposure MRL of 0.002 ppm.

Derivation of ITSL (Annual)

ATSDR states that "an MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified route and duration of exposure" (ATSDR, 2019). ATSDR derived the MRL from the most sensitive endpoint which are lesions of the olfactory epithelium and an intermediate MRL is derived to be protective for 15 to 364 days. According to Rule 229(2)(b), which allows AQD to use the best available science to derive a screening level, the intermediate MRL of 0.002 ppm is appropriate and will be adopted as an ITSL. 1,1,2-Trichloroethane concentrations in ppm were converted to µg/m^3 using equation 4-1b on page 4-20 in EPA (1994) with the assumptions that the testing was performed at 25°C and 760 mmHg, and that 1-g/mole of a perfect gas occupies 24.45 L.

$$mg/m^3 = \frac{ppm \times MW}{24.45}$$

The molecular weight of 1,1,2-trichloroethane is 133.405 g/mol. Using the equation above:

$$mg/m^3 = \frac{0.002\,ppm \times 133.405\ g/mol}{24.45} = 0.010912474\ mg/m^3 = 10.912474\ \mu g/m^3$$
$$\approx 11\ \mu g/m^3$$

According to Rule 232(2)(b) the averaging time is annual. Therefore, the initial threshold screening level (ITSL) for 1,1,2-trichloroethane (CAS# 79-00-5) is 11 µg/m^3 based on an annual averaging time.

Key Study (Short-term ITSL)

ATSDRs acute-duration inhalation MRL is 0.03 ppm. It is based on Kirkpatrick (2001):"F344 rats(5/sex/group) were exposed whole-body to 1,1,2-trichloroethane (purity 99.56%) as a vapor at target concentrations of 0, 50, 200, and 1,500 ppm for 4 hours and sacrificed 24 hours after cessation of exposure. Actual (measured) concentrations were 0, 58, 181, and 1,527 ppm. Animals were monitored for mortality and clinical signs of toxicity. The response to a stimulus

(noise) was assessed at (approximately) the midpoint of exposure. One day following exposure, detailed physical examinations were performed, and body weights were measured. At necropsy, organ weights (of the adrenals, brain, kidneys, liver, lungs, ovaries, and testes) were recorded. Respiratory tract tissues were examined microscopically in animals from all exposure groups; the liver, kidneys, and stomach were additionally examined in animals in the control and high-exposure groups" (ATSDR, 2019).

Critical Effect

ATSDR used the lowest observed adverse effect level (LOAEL) of 58 ppm for necrosis of the olfactory epithelium in rats. Benchmark Dose (BMD) was not used because the lowest tested concentration of 58 ppm showed incidence of necrosis in 70% of the animals, which is substantially higher than the benchmark response (BMR) of 10%. The LOAEL of 58 ppm was adjusted to a human equivalent concentration (HEC) of 7.5 ppm and an uncertainty factor of 270 was applied (3 for extrapolation from animals to humans; 10 for human variability; 3 for extrapolation from a minimal LOAEL – the study authors classified the severity of necrosis as minimal to mild because the necrosis affected a small number of cells; and 3 as a modifying factor for database deficiency as the only acute exposure data are from a single 4-hour exposure study) (ATSDR, 2019).

The acute inhalation MRL from ATSDR was adopted as the acute ITSL (160 ug/m^3, 24-hr AT) because it is recently derived, it is well documented and justified, and it provides an appropriate level of protection. Also, the ATSDR MRL was derived using a 1,1,2-trichloroethane inhalation study, unlike the ACGIH TLV-TWA, which used the similarity of effects of 1,1,2-trichloroethane to 1,1,2,2-tetrachloroethane and chloroform to develop a value. Table 3 provides a useful comparison of this and other acute benchmarks discussed above, and the associated candidate acute ITSLs.

According to Rule 229(2)(b), the acute-duration inhalation MRL of 0.03 ppm is appropriate based on toxicological grounds and is supported by the scientific data, therefore the acute-duration MRL will be adopted as an ITSL. 1,1,2-Trichloroethane concentrations in ppm were converted to µg/m^3 using equation 4-1b on page 4-20 in EPA (1994) with the assumptions that the testing was performed at 25°C and 760 mmHg, and that 1-g/mole of a perfect gas occupies 24.45 L.

$$mg/m^3 = \frac{ppm \times MW}{24.45}$$

The molecular weight of 1,1,2-trichloroethane is 133.405 g/mol. Using the equation above:

$$mg/m^3 = \frac{0.03\ ppm \times 133.405\ g/mol}{24.45} = 0.163687117\ mg/m^3 = 163.687117\ \mu g/m^3$$
$$\approx 160\ \mu g/m^3$$

ATSDR states that acute MRLs are applicable to exposure of 14 days or less, the most appropriate and practical averaging time for AQD to assign to this acute MRL value is 24 hours. The acute inhalation MRL from ATSDR was adopted as the acute ITSL (160 ug/m^3, 24-hr AT) because it is recently derived, it is well documented and justified, and it provides an appropriate level of protection.

Other Relevant Information

ACGIH set a TLV-TWA of 10 ppm (55,000 µg/m^3) for 1,1,2-trichloroethane "to minimize the potential for central nervous system depression that can lead to narcosis, eye and upper respiratory tract irritation, and liver damage" (ACGIH, 2001). "The TLV-TWA is based on toxicological similarity of 1,1,2-TCE to 1,1,2,2-tetrachloroethane and by analogy to chloroform" (ACGIH, 2001). "The TLV-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, day after day, for a working lifetime without adverse effect." (ACGIH, 2001). The TLVs are derived for workers who are typically healthy adults that are exposed during work hours and do not consider long-term exposures or their effects on susceptible subpopulations such as infants, children, the elderly, sensitive individuals, or those with illnesses. Therefore, TLVs may generally be divided by 100 to derive an acute ITSL that may be presumed to be protective of the general population including sensitive subgroups.

OSHA set a PEL of 10 ppm (45,000 ug/m^3) for 1,1,2-trichloroethane (OSHA, 2020). The 8-hour PEL is the highest level of exposure an employee may be exposed to without incurring the risk of adverse health effects. These values are for workers and may be divided by 100 to be presumed to be protective for the general population including sensitive subgroups. NIOSH set a REL of 10 ppm (45,000 µg/m^3) for 1,1,2-trichloroethane (NIOSH, 2020). NIOSH RELs are time-weighted average concentrations for up to a 10-hour workday during a 40-hour workweek. As with the ACGIH TLVs above, these values are derived for workers and may be divided by 100 to be presumed to be protective for the general population including sensitive subgroups.

Table 3. 1,1,2-Trichloroethane Acute Toxicity Benchmarks and Candidate Acute ITSLs

Available benchmark type	Value (µg/m^3)	Candidate acute ITSL (in descending order) (µg/m^3)	Candidate ITSL Averaging Time
ACGIH TLV-TWA	55,000	TLV/100 = 550	8-hour
OSHA PEL	45,000	PEL/100 = 450	8-hour
NIOSH REL	45,000	REL/100 = 450	8-hour
ATSDR acute MRL	160	160	24-hour

Summary

The initial risk screening level (IRSL) for 1,1,2-trichloroethane (CAS #79-00-5) is 0.063 µg/m^3 and the SRSL is 0.63 µg/m^3 based on an annual averaging time. The initial threshold screening level (ITSL) for 1,1,2-trichloroethane (CAS #79-00-5) is 11 µg/m^3 based on an annual averaging time. A second acute ITSL for 1,1,2-trichloroethane of 160 µg/m^3 based on a 24-hour averaging time. The acute ITSL is derived as a short-term exposure and will be used in conjunction with the more chronic ITSL.

References

ACGIH. 2001. 1,1,2-Trichloroethane. CAS number: 79-00-5. TLVs and BEIs Based on the Documentation of the Threshold Limit Values for Chemical Substances and Physical Agents & Biological Exposure Indices. ACGIH Worldwide Signature Publications.
Act 451 of 1994. Part 55, Air Pollution Control, of the Natural Resources and Environmental Protection Act, 1994 PA 451, as amended.

ATSDR. 2019. Toxicological Profile for 1,1,2-Trichloroethane. Draft for Public Comment. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Available online at: https://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=796&tid=155

Crebelli R, Carere A, Leopardi P, Conti L, Fassio F, Raiteri F, Barone D, Ciliutti P, Cinelli S, and Vericat JA. 1999. Evaluation of 10 aliphatic halogenated hydrocarbons in the mouse bone marrow micronucleus test. Mutagenesis 14(2):207-215.

EPA. 1987. Integrated Risk Information System (IRIS). 1,1,2-Trichloroethane; CASRN 79-00-5. National Center for Environmental Assessment. Available online at: https://cfpub.epa.gov/ncea/iris2/chemicalLanding.cfm?substance_nmbr=198

EPA. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office. EPA/600/8-90/066F.

EPA. 2012. Drinking water standards and health advisories. Washington, DC: U.S. Environmental Protection Agency, Office of Water. EPA822S12001.

Foureman P, Mason JM, Valencia R, and Zimmering S. 1994. Chemical mutagenesis testing in Drosophila. X. Results of 70 coded chemicals tested for the National Toxicology Program. Environ Mol Mutagen 23(3):208-227.

Gargas ML, Burgess RJ, Voisard DE, Cason GH, and Andersen ME. 1989. Partition coefficients of low-molecular-weight volatile chemicals in various liquids and tissues. Toxicol Appl Pharmacol. 98(1):87-99.

Kirkpatrick DT. 2001. Acute inhalation toxicity (with histopathology) study of 1,1,2-trichloroethane (1,1,2-TCE) in rats. WIL Research Laboratories, Inc. HAP Task Force. EPA-HQ-OPPT-2002-0056-0039. WIL-417001.

Kirkpatrick DT. 2002. A 90-day inhalation toxicity study of 1,1,2-trichloroethane (1,1,2-TCE) in rats (with satellite groups for pharmacokinetic evaluations in rats and mice). WIL Research Laboratories, Inc. HAP Task Force. EPA-HQ-OPPT-2002-0046-003. WIL-417002.

Mazullo M, Colacci A, Grilli S, Prodi G, and Arfellini G. 1986. 1,1,2-Trichloroethane: evidence of genotoxicity from short-term tests. Jpn J Cancer Res 77(6):532-539.

Mirsalis JC, Tyson CK, Steinmetz KL, Lih EK, Hamilton CM, Bakke JP, and Spalding JW. 1989. Measurement of unscheduled DNA synthesis and S-phase synthesis in rodent hepatocytes following in vivo treatment: Testing of 24 compounds. Environ Mol Mutagen 14(3):155-164.

Mitoma C, Tyson CA, Riccio ES. 1985. Investigation of the species sensitivity and mechanism of carcinogenicity of halogenated hydro-carbons final report EPA Contract 68-01-5079. EPA/OTS; Document #40-842-8424225.

Miyagawa M, Takasawa H, Sugiyama A, Inoue Y, Murata T, Uno Y, and Yoshikawa K. 1995. The in vivo-in vitro replicative DNA synthesis (RDS) test with hepatocytes prepared from male B6C3F1 mice as an early prediction assay for putative nongenotoxic (Ames-negative) mouse hepatocarcinogens. Mutat Res 343(2-3):157-183.

Morgan A, Black A, Belcher DR. 1970. The excretion in breath of some aliphatic halogenated hydrocarbons following administration by inhalation. Ann Occup Hyg 13:219-233.

Morgan A, Black A, Belcher DR. 1972. Studies on the absorption of halogenated hydrocarbons and their excretion in breath using ^{38}Cl tracer techniques. Ann Occup Hyg 15:273-282.

NCI. 1978. National Cancer Institute. Bioassay of 1,1,2-trichloroethane for possible carcinogenicity. U.S. DHEW Tech. Rep. Ser. 74. Publ. No. NIH 78-1324.

NIOSH. 2020. 1,1,2-Trichloroethane. NIOSH Pocket Guide to Chemical Hazards. The National Institute for Occupational Safety and Health. Available online at: https://www.cdc.gov/niosh/npg/npgd0628.html

OEHHA. 2009. 1,1,2-Trichloroethane (Vinyl Trichloride) CAS No: 79-00-5. Air Toxics Hots Spots Program Technical Support Document for Cancer Potencies. Appendix B. Chemical-specific summaries of the information used to derive unit risk and cancer potency values. Updated 2011. Available online at: https://oehha.ca.gov/media/downloads/crnr/appendixb.pdf

OSHA. 2020. 1,1,2-Trichloroethane. Occupational Safety and Health Administration. OSHA Annotated PELs. Standard 1910.1000 Table Z-1. Part Z. Toxic and Hazardous Substances.

Sapphire Group. 2003. Physiologically based pharmacokinetic model development, simulations, and sensitivity analysis for repeated exposure to 1,1,2-trichloroethane. The Sapphire Group. HAP Task Force. EPA-HQ-OPPT-2002-0046-0007. https://www.regulations.gov/document?D=EPA-HQ-OPPT-2002-0046-0007

Sapphire Group. 2004. Route-to-route extrapolation of 1,1,2-trichloroethane studies from the oral route to inhalation using physiologically based pharmacokinetic models: Carcinogenicity. The Sapphire Group. HAP Task Force. EPA-HQ-OPPT-2002-0046-0009.

Taningher M, Parodi S, Grilli S, Colacci A, Mazzullo M, Bordone R, and Santi L. 1991. Lack of correlation between alkaline DNA fragmentation and DNA covalent binding induced by polychloroethanes after in vivo administration. Problems related to the assessment of a carcinogenic hazard. Cancer Detection and prevention 15(1):35-39.

Vogel EW and Nivard MJ. 1993. Performance of 181 chemicals in a Drosophila assay predominantly monitoring interchromosomal mitotic recombination. Mutagenesis 8(1):57-81.

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