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**Chemical Name: Vinyl Chloride**

**CAS: 75-01-4**

**Synonyms:** Chloroethene; chloroethylene; ethylene monochloride; Monochloroethene;  
Monochloroethylene

**Acute Non-Cancer Health Risk Limit (nHRL<sub>acute</sub>) = Not Derived (Insufficient data)**

**Short-term Non-Cancer Health Risk Limit (nHRL<sub>short-term</sub>) = Not Derived (Insufficient data)**

**Subchronic Non-Cancer Health Risk Limit (nHRL<sub>subchronic</sub>) = 80 ug/L**

$$= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Subchronic intake, L/kg/d})}$$

$$= \frac{(0.03 \text{ mg/kg/d}) \times (0.2) \times (1000 \text{ ug/mg})}{(0.077 \text{ L/kg-d})}$$

$$= 77.92 \text{ rounded to } \mathbf{80 \text{ ug/L}}$$

Toxicity value: 0.03 (laboratory animal)  
Source of toxicity value: MDH 2007  
Point of Departure: 10 ppm (NOAEL, CMA 1998 as cited by EPA 2000)  
Human Equivalent Dose Adjustment: 1 mg/kg-d  
Total uncertainty factor: 30  
UF allocation: 10 for intraspecies and 3 for interspecies extrapolation because PBPK modeling decreases uncertainty for animal to human extrapolation but does not account for toxicodynamic differences.  
Critical effect(s): increased liver weight, hypertrophy and hepatocellular foci.  
Co-critical effect(s): none  
Additivity endpoint(s): Hepatic (liver) system  
Secondary effect(s): none

**Chronic Non-Cancer Health Risk Limit (nHRL<sub>chronic</sub>) = 10 ug/L**

$$\begin{aligned} &= \frac{(\text{Reference Dose, mg/kg/d}) \times (\text{Relative Source Contribution}) \times (\text{Conversion Factor})}{(\text{Chronic intake rate, L/kg/d})} \\ &= \frac{(0.003 \text{ mg/kg/d}) \times (0.2) \times (1000 \text{ ug/mg})}{(0.043 \text{ L/kg-d})} \\ &= 13.98 \text{ rounded to } \mathbf{10 \text{ ug/L}} \end{aligned}$$

Toxicity value:	0.003 (laboratory animal)
Source of toxicity value:	MDH, 2007
Point of Departure:	0.13 mg/kg-d (NOAEL, Til et al, 1991 as cited by EPA 2000)
Human Equivalent Dose Adjustment:	0.09 mg/kg-d
Total uncertainty factor:	30
UF allocation:	10 for intraspecies and 3 for interspecies extrapolation because PBPK modeling decreases uncertainty for animal to human extrapolation but does not account for toxicodynamic differences
Critical effect(s):	liver cell polymorphism and cyst formation
Co-critical effect(s):	none
Additivity endpoint(s):	Hepatic (liver) system
Secondary effect(s):	none

**Cancer Health Risk Limit (cHRL) = 0.2 ug/L**

The lifetime oral slope factor from IRIS was used as a chemical-specific slope factor:

$$\begin{aligned} &= \frac{(\text{Additional Lifetime Cancer Risk, } 1 \times 10^{-5}) \times (\text{Conversion Factor, } 1000 \text{ ug/mg})}{(\text{Slope Factor, per mg/kg-d}) \times (\text{Lifetime Adjustment Factor}) \times (\text{Lifetime Intake Rate, L/kg})} \\ &= \frac{(1E-5) \times (1000 \text{ ug/mg})}{(1.4 \text{ (mg/kg-d)}^{-1} \times (1) \times 0.043 \text{ L/kg-d})} \\ &= 0.166 \text{ rounded to } \mathbf{0.2 \text{ ug/L}} \end{aligned}$$

Cancer classification:	A (a known human carcinogen)
Oral Slope factor:	1.4 (mg/kg-d) <sup>-1</sup> (laboratory animal)
Source of slope factor:	IRIS 2000
Tumor site(s):	Liver, and blood vessels (primary sites); Kidney, stomach and skin cancers (secondary sites)

**Volatile: Yes (highly volatile)**

**Summary of changes since 1993/1994 HRL promulgation:**

Since no non-cancer HRL was previously calculated, the short-term, subchronic, and chronic nHRLs represent new values.

The cancer HRL (0.2 ug/L) is the same as the 1993/94 cancer HRL (0.2 ug/L) as the result of: 1) the utilization of the continuous lifetime exposure from birth cancer slope factor (1.4 per mg/kg/day), 2) the use of a lifetime time-weighted average of water consumption rate of 0.043 L/kg-d and 3) rounding to one significant digit.

**Summary of toxicity testing for health effects identified in the Health Standards Statute:**

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity
Tested?	No	Yes	Yes	Yes	Yes
Effects?	Yes <sup>1</sup>	Yes <sup>2</sup>	Yes <sup>3</sup>	Yes <sup>4</sup>	Yes <sup>5</sup>

Note: Even if testing for a specific health effect was not conducted for this chemical, information about that effect might be available from studies conducted for other purposes. Most chemicals have been subject to multiple studies in which researchers identify a dose where no effects were observed, and the lowest dose that caused one or more effects. A toxicity value based on the effect observed at the lowest dose across all available studies is considered protective of all other effects that occur at higher doses.

**Comments on extent of testing or effects:**

Note: Many reported effects occur via the inhalation route of exposure. Vinyl chloride is readily and rapidly absorbed via all routes of exposure and effects via all routes occur systemically.

<sup>1</sup>A study of workers exposed to vinyl chloride in PVC manufacturing plants reported that most workers who presented with scleroderma were shown to have thyroid insufficiency. No histopathology effects on the adrenals were reported in guinea pigs exposed to 400,000 ppm for 30 minutes. Rats were found to have colloid goiter and markedly increased numbers of perifollicular cells.

<sup>2</sup>Stimulation of spontaneous lymphocyte transformation was observed in mice following inhalation exposure. There is some evidence to suggest that an adaptive process may lead to a reduction or elimination of this effect over time. Also, it is not clear from the evidence that a clear adverse effect to the immune system is taking place.

<sup>3</sup>Developmental toxicity occurred in inhalation experiments at doses that caused maternal toxicity. These effects occurred at exposure levels significantly higher than those producing liver toxicity (i.e., the basis of the RfD)

<sup>4</sup>Testicular histopathological changes and decreased male fertility have been reported in inhalation studies. These effects occur at exposure levels significantly higher than those producing liver toxicity (i.e., the basis of the RfD).

<sup>5</sup>Nervous system toxicity has been observed in inhalation studies at high exposure levels. Vinyl chloride was once considered for use as an inhalation anesthetic. Investigators studying the effects of vinyl chloride exposure frequently report central nervous system symptoms that are consistent with the anesthetic properties of vinyl chloride. The most commonly reported central nervous system effects are ataxia or dizziness, drowsiness or fatigue, loss of consciousness, and/or headache. Other central nervous system effects that have been reported by vinyl chloride workers include euphoria and irritability, visual and/or hearing disturbances, nausea, memory loss, and nervousness and sleep disturbances.

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