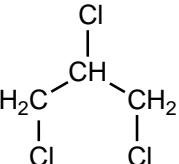


14	CAS No.: 96-18-4	Substance: 1,2,3-Trichloropropane
Chemical Substances Control Law Reference No.: 2-83 (poly(3-5)chloropropene)		
PRTR Law Cabinet Order No.: – (Cabinet Order No. after revision*: 1-289)		
Structural Formula:		
Molecular Formula: C ₃ H ₅ Cl ₃ Molecular Weight: 147.43		
		
*Note: No. according to revised order enacted on October 1, 2009.		
1. General information		
<p>The aqueous solubility of this substance is 1.75×10^3 mg/L (25°C), the partition coefficient (1-octanol/water) ($\log K_{ow}$) is 2.63, and the vapor pressure is 3.69 mmHg (=492 Pa) (25°C). The biodegradability (aerobic degradation) is characterized by a BOD degradation rate of 0%, and bioaccumulation is thought to be nonexistent or low. Its half-life for hydrolysis is 44 years (calculated value).</p>		
<p>Based on a revision of substances regulated by the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management (PRTR Law) (enacted on October 1, 2009), this substance was newly designated as a Class 1 Designated Chemical Substance. It is primarily used in closed systems as a synthetic intermediate for pesticides and other chemical compounds and as a crosslinking agent in polymer manufacture. In addition, it is produced as a by-product in the manufacture of chlorinated compounds such as epichlorohydrin. The production quantity of this substance is approximately 500 t.</p>		
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2. Exposure assessment		
<p>Because this substance was not classified as a Class 1 Designated Chemical Substance prior to revision of substances regulated by the PRTR Law, release and transfer quantities could not be obtained. Predictions of distribution by medium using a Mackay-type level III fugacity model indicated that if equal quantities were released to the atmosphere, water bodies, and soil, the proportions distributed to soil and water bodies would be higher.</p>		
<p>Data for setting the predicted maximum exposure to humans via inhalation could not be obtained. The predicted maximum oral exposure was estimated to be less than around 0.0004 µg/kg/day based on calculations from data for groundwater, and around 0.0012 µg/kg/day based on calculations from data for public freshwater bodies. A predicted maximum oral exposure of around 0.0012 µg/kg/day was adopted for this substance. The risk of exposure to this substance by intake from an environmental medium via food is considered slight.</p>		
<p>The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was 0.03 µg/L for public freshwater bodies and about 0.01 µg/L for seawater.</p>		
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3. Initial assessment of health risk		
<p>This substance is irritating to the eyes and respiratory tract and may cause effects on the liver and kidneys, causing functional hepatic and renal disorders. Loss of consciousness is caused as a result of exposure to high levels of this substance. Inhalation exposure causes, cough, sore throat, headache, lethargy and loss of consciousness while oral exposure causes nausea, headache, diarrhea, lethargy and loss of consciousness. By contact with this substance, redness and painful irritation in the eyes and dryness and redness of and prickling pain in the skin are caused.</p>		
<p>As for its non-carcinogenic effects, information on its general toxicity and reproductive toxicity has been obtained. As for its carcinogenicity, experiments on animals have provided its evidences, so the substance is likely to be carcinogenic</p>		

to humans. Initial assessments have been conducted both on its non-carcinogenic and carcinogenic effects.

A no-observed-adverse-effect-level (NOAEL) of 3 mg/kg/day for increased liver weight, forestomach hyperplasia was obtained for its non-carcinogenic effects through oral exposure, from mid-term and long-term toxicity tests for rats. It was then adjusted for exposure conditions, and divided by 10, as is always the case with LOAEL, to produce 0.21 mg/kg/day as its ‘non-toxic level*’. As for its carcinogenicity, it was assumed that there was no threshold, and 7 (mg/kg/day)⁻¹ was identified as its slope factor for tumors at multiple sites, from experiments on rats.

As for its inhalation exposure, its no-observed-adverse-effect-level (NOAEL) of 6.1 mg/m³ (for the degeneration of the olfactory epithelium) obtained for its non-carcinogenic effects through inhalation exposure from mid-term and long-term toxicity tests for rats. It was then adjusted for exposure conditions to provide 1.2 mg/m³. This was divided by 10 due to their short test periods to produce 0.12 mg/m³ as its ‘non-toxic level*’. As for its carcinogenicity, its unit risk could not be obtained when it was assumed that there was no threshold.

As for its oral exposure, the predicted maximum exposure was estimated to be 0.0012 µg/kg/day, when intakes of freshwater from public water supply and also food intakes were assumed. Its margin of exposure (MOE) would be 1,800, when calculated from its ‘non-toxic level*’ of 0.2 mg/kg/day and the predicted maximum exposure, then divided by 10 due to the fact that the ‘non-toxic level*’ was obtained from animal experiments, and divided again by 10 when its carcinogenicity was considered. On the other hand, the excess incidence rate of its carcinogenicity for the predicted maximum exposure would be 8.4×10^{-6} when calculated from the slope factor. Since risk associated with exposure to this substance through food intakes from the environment is presumed to be minimal, this exposure will not increase MOE significantly, and no further action will be required at the moment to assess health risk from oral exposure to this substance.

As for inhalation exposure to this substance, lack of information on its exposure concentration did not allow its risk assessment. Its half-life in the atmosphere is as long as 15 to 150 days. When released to the atmosphere, most of it is expected to remain in the ambient air. Collection of information on its inhalation exposure to assess health risk associated with its exposure in the ambient air would be required.

Exposure Path	Information of toxicity			Exposure assessment		Result of risk assessment			Judgment	
	Criteria for risk assessment		Animal	Criteria for diagnoses (endpoint)	Exposure medium					
Oral	‘Non-toxic level’	0.21 mg/kg/day	Rats	Increase in liver weight, forestomach hyperplasia, etc.	Drinking water	— µg/kg/day	MOE excessive incidence	— —	✗ ✗	▲
	Slope factor	7 (mg/kg/day) ⁻¹	Rats	Tumors at multiple sites	Fresh water	0.0012 µg/kg/day	MOE excessive incidence	1,800 8.4×10^{-6}	○ ▲	
Inhalation	‘Non-toxic level’	0.12 mg/m ³	Rats	Degeneration of the olfactory epithelium	Ambient air	— µg/m ³	MOE excessive incidence	— —	✗ ✗	(▲)
	Unit risk	— (µg/m ³) ⁻¹	—	—	Indoor air	— µg/m ³	MOE excessive incidence	— —	✗ ✗	✗

Non-toxic level *

- When a LOAEL is available, it is divided by 10 to obtain a level equivalent to NOAEL.
- When an adverse effect level is available for the short-term exposure, it is divided by 10 to obtain a level equivalent to an adverse effect level for the long-term exposure.

4. Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 48-h median effective concentration (EC₅₀) of 4,130 µg/L for swimming inhibition in the crustacean *Ceriodaphnia cf. dubia*, and a 96-h median lethal concentration (LC₅₀) of 66,500 µg/L for the fish species *Pimephales promelas* (fathead minnow). Accordingly, based on these acute

toxicity values and an assessment factor of 1,000, a predicted no effect concentration (PNEC) of 4.1 µg/L was obtained. No data is available regarding chronic toxicity, and on this account, the acute toxicity to the crustacean of 4.1 µg/L was adopted as the PNEC for this substance.

The PEC/PNEC ratios were 0.007 for freshwater bodies and 0.002 for seawater. Accordingly, further work is thought to be unnecessary at this time.

Hazard assessment (basis for PNEC)			Assessment factor	Predicted no effect concentration PNEC (µg/L)	Exposure assessment		PEC/PNEC ratio	Result of assessment
Species	Acute/chronic	Endpoint			Water body	Predicted environmental concentration PEC (µg/L)		
Crustacean (water flea)	Acute	EC ₅₀ Swimming inhibition	1,000	4.1	Freshwater	0.03	0.007	○
					Seawater	0.01	0.002	

5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Collection of information required.	▲
	Inhalation exposure	Further information collection would be required for risk characterization.	(▲)
Ecological risk	No need for further work.		○

[Risk judgments] ○: No need for further work ▲: Requiring information collection

■: Candidates for further work ×: Impossibility of risk characterization

(○) : Though a risk characterization cannot be determined, there would be little necessity of collecting information.

(▲) : Further information collection would be required for risk characterization.