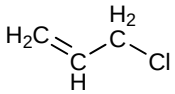
8	CAS No.: 107-05-1	Substance: 3-Chloropropene

Chemical Substances Control Law Reference No.: 2-123

PRTR Law Cabinet Order No.: 1-123

Molecular Formula: C_3H_5Cl Structural Formula:

Molecular Weight: 76.52



1. General information

The aqueous solubility of this substance is 4×10^3 mg/1,000 g (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 2.1, and the vapor pressure is 367 mmHg (=4.89×10^4 Pa) (25°C). Biodegradability (aerobic degradation) is good, and bioaccumulation is judged to be non-existent or low. Its half-life for hydrolysis is 6.9 days (20°C) or 14 days (25°C).

This substance is designated as a Class 1 Designated Chemical Substance under the Law Concerning Reporting, etc. of Releases to the Environment of Specific Chemical Substances and Promoting Improvements in Their Management (PRTR Law). The main use of this substance is almost entirely as a raw material for epichlorohydrin, which itself is a raw material for epoxy resin. The production and import quantity in fiscal 2010 was 60,000 t. The production and import category under the PRTR Law is more than 100 t.

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2. Exposure assessment

Total release to the environment in fiscal 2010 under the PRTR Law was approximately 87 t of which approximately 87 t or more than 99% of overall releases were reported. The major destination of reported releases was the atmosphere. In addition, 24 t was transferred to waste materials and 0.10 t to sewage. The main source of reported releases was the chemical industry. The largest release among the releases to the environment including those unreported was to the atmosphere. A multi-media model used to predict the proportions distributed to individual media in the environment indicated that in regions where the largest quantities were estimated to have been released to the environment in general or to the atmosphere in particular, the predicted proportion distributed to the atmosphere was 98.8%. In regions where the largest estimated releases were to public water bodies, the predicted proportion distributed to the atmosphere was 97.6%.

The maximum expected concentration of exposure to humans via inhalation for the general environmental atmosphere was reported to be 0.0085 μg/m^3 (annual average), although this value is smaller than the lower detection limit. Furthermore, a maximum detected level of 0.52 μg/m^3 was reported in a study of the general environmental atmosphere for a limited area. The mean annual value for atmospheric concentration in fiscal 2010 was calculated by using a plume-puff model on the basis of reported releases to the atmosphere according to the PRTR Law; this model predicted a maximum level of 7.1 μg/m^3.

The maximum expected oral exposure could not be obtained. Note that albeit past data, a maximum expected oral exposure of around 0.0004 μg/kg/day was calculated from groundwater data. However, when reported releases to public freshwater bodies in fiscal 2010 according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers while taking into consideration only dilution gave a maximum value of 29 μg/L. Using this estimated concentration for rivers to calculate oral exposure gave 1.2 μg/kg/day. The risk of exposure to this substance by intake from an environmental medium via food is considered slight, based on estimates of oral exposure obtained by using estimated concentrations in fish species.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, could not be obtained. However, past data yielded less than 0.01 μg/L for public freshwater bodies and around less than

0.01 μg/L for seawater. The maximum river concentration was estimated to be 29 μg/L from reported releases to public freshwater bodies under the PRTR Law.

3.Initial assessment of health risk

This substance may cause irritation to eyes, skin and respiratory tract, and it may affect the central nervous system. Inhalation exposure to the substance may cause coughing, sore throat, headache, dizziness, weakness, labored breathing, vomiting and loss of consciousness, while its oral exposure may cause abdominal pain, burning sensation and vomiting. Contact of the substance with skin may cause redness, burning sensation and pain, while its contact with eyes may cause redness, pain and blurred vision. Pulmonary edema may occur if exposed to its vapor of high concentrations. Its minimum lethal dose for human is reported to be 3,000 ppm (9,390 mg/m^3).

Although sufficient information was not available to evaluate carcinogenic potential of the substance, an initial assessment was conducted on the basis of information on its non-carcinogenic effects.

With regard to oral exposure to the substance, a LOAEL of 55 mg/kg/day (for suppressed body weight increase, etc.) obtained from its mid-term and long-term toxicity tests on rats was adjusted for their durations to provide 39 mg/kg/day for its intermittent to continuous exposure, and divided by a factor of 10 for the use of a LOAEL. 3.9 mg/kg/day was identified to be the reliable lowest dose of the substance as its 'non-toxic level*'. With regard to its inhalation exposure, a NOAEL of 17 mg/m^3 (for effects on peripheral nerves, liver and kidneys) obtained from its mid-term and long-term toxicity tests on rabbits was adjusted for their durations to provide 3.6 mg/m^3 for its intermittent to continuous exposure, and divided by a factor of 10 due to their short test periods. 0.36 mg/m^3 was identified to be the reliable lowest dose of the substance as its 'non-toxic level*'.

With regard to oral exposure to the substance, as its exposure concentrations were not known, its health risk could not be assessed. In addition, oral exposure to the substance was estimated to be below 0.0004 μg/kg/day from its maximum concentration in groundwater reported in 1999. The MOE would be above 980,000 when calculated from its 'non-toxic level*' of 3.9 mg/kg/day from animal experiments and divided by a factor of 10 to convert animal data to human. In addition, its maximum exposure was calculated to be 1.2 μg/kg/day from its concentrations in river water with effluents from operators discharging high concentrations of the substance, reported in FY 2010 under the PRTR Law. The MOE would be 330 when calculated from this value as its reference. As exposure to the substance in the environment through food intakes would be limited, the MOE would not change significantly even when this exposure was included. Therefore, collection of further information would not be required to assess health risk from its oral exposure.

With regard to inhalation exposure to the substance, its maximum exposure concentration in the ambient air was predicted to be below the detection limit, although its highest annual mean concentration of 0.0085 μg/m^3 had been reported. The MOE would be 4,200 when calculated from its 'non-toxic level*' of 0.36 mg/m^3 and the maximum exposure concentration predicted from animal experiments, and divided by a factor of 10 to convert animal data to human. However its maximum concentration of 0.52 μg/m^3 has been reported for some area. The MOE would be 69 when calculated from this value as its reference. In addition, its maximum (annual mean) concentration in the ambient air near the operators discharging high concentrations of the substance was calculated to be 7.1 μg/m^3 from its emissions reported in FY 2010 under the PRTR Law. The MOE would be 5 when calculated from this value as its reference. Therefore, collection of further information would be required at this moment to assess health risk from its inhalation in the ambient air.

Toxicity				Exposure assessment		Result of risk assessment			Judgment
Exposure Path	Criteria for risk assessment	Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure dose and concentration				
Oral	'Non-toxic level*' 3.9 mg/kg/day	Rat	Suppressed body weight increase, etc.	Drinking water	— μg/kg/day	MOE	—	×	(○)
				Groundwater	— μg/kg/day	MOE	—	×	
Inhalation	'Non-toxic level*' 0.36 mg/m^3	Mouse	Effects on peripheral nerves, liver and kidneys	Ambient air	0.0085 μg/m^3	MOE	4,200	○	(▲)
				Indoor air	— μg/m^3	MOE	—	×	×

Non-toxic level *

・When a LOAEL is available, it is divided by 10 to obtain a NOAEL-equivalent level.

・When an adverse effect level for the short-term exposure is available, 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

The predicted environmental concentration (PEC) of this substance could not be obtained. Furthermore, a predicted no effect concentration (PNEC) could not be set for this substance because toxicity data applicable to initial assessment could not be obtained. As such, a judgment on ecological risk could not be made. A 14-d LC_{50} of 1,210 μg/L from extended toxicity tests with *Poecilia reticulata* (guppy) suggests that the acute toxicity value for the guppy exceeds 1,210 μg/L. Accordingly, if this chronic toxicity value is divided by an assessment factor of 1,000, a preliminary PNEC in excess of 1.2 μg/L is obtained.

Albeit past data, the concentration of this substance in public water bodies is around less than 0.01 μg/L, and the ratio of this concentration to the preliminary PNEC is smaller than 0.1. However, the river concentration was estimated to be 29 μg/L from reported emissions under the PRTR Law. There may be locations where the ratio to the preliminary PNEC exceeds 0.1.

Accordingly, efforts to collect data on this substance are needed, as are measurements of environmental concentrations by taking PRTR data into consideration. Furthermore, there is a need to consider the augmentation of toxicity data by taking into account the findings of these environmental concentration measurements.

Hazard assessment (basis for PNEC)			Assessment factor	Predicted no effect concentration PNEC (μg/L)	Exposure assessment		PEC/PNEC ratio	Judgment based on PEC/PNEC ratio	Assessment result
Species	Acute/ chronic	Endpoint			Water body	Predicted environmental concentration PEC (μg/L)			
—	—	—	—	—	Freshwater	—	—	×	▲
					Seawater	—	—		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Although risk to human health could not be identified, collection of further information would not be required.	(○)
	Inhalation exposure	Collection of further information would be required.	(▲)
Ecological risk	Requiring information collection.		▲

［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.