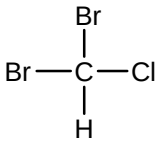
8	CAS No.: 124-48-1	Substance: Dibromochloromethane

Chemical Substances Control Law Reference No.:

PRTR Law Cabinet Order No.: 1-209

Molecular Formula: $CHBr_2Cl$ Structural Formula:



Molecular Weight: 208.28

1. General information

The aqueous solubility of this substance is 2.51×10^3 mg/1,000 g (30°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 2.16, and the vapor pressure is 76 mmHg (1.0×10^4 Pa) (20°C). The mean biodegradability (aerobic degradation) as determined by BOD, TOC, and GC is 25%. Its half-life for hydrolysis is 27.4–274 years (calculated assuming a pH of 8–7).

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). This substance is a component of trihalomethane, which is formed during the process of water purification by the aqueous reaction of organic substances such as humins with the chlorine in disinfectants. It is unintentionally formed in the chlorination processes of wastewater and cooling water.

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2. Exposure assessment

Total release to the environment in fiscal 2013 under the PRTR Law was approximately 54 t and all emissions were unreported. The majority of unreported emissions were to water bodies. 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 overall or public water bodies in particular, the predicted proportion distributed to the atmosphere was 79.5%, and that distributed to water bodies was 19.8%. In regions where the largest quantities were estimated to have been released to the atmosphere, the predicted proportion distributed to the atmosphere was 86.9%.

The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was around 0.029 μg/m^3. In addition, the predicted maximum concentration of exposure for indoor air was 12 μg/m^3. However, past general environmental atmospheric data from an environmental study that surveyed a limited area indicated around 0.49 μg/m^3. The maximum expected oral exposure was 1.6 μg/kg/day when calculated from potable water data. Furthermore, the predicted maximum exposure calculated from potable water data and past data for food from a study that surveyed a limited area was also 1.6 μg/kg/day.

Information to determine the predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, could not be obtained. However, past data yielded a value of around 0.41 μg/L for public freshwater and a value of around 0.04 μg/L for seawater. However, the PEC was 10 μg/L at maximum in freshwater bodies on the basis of measurements of raw water from the surface of lakes, including dam lakes.

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3. Initial assessment of health risk

No information on acute symptoms in humans was available. The observed symptoms caused by this substance were piloerection, sedation, muscular relaxation, ataxia and exhaustion in rats and sedation and paralysis in mice.

As sufficient information on the carcinogenicity of the substance was not available, the initial assessment was conducted on the basis of information on its non-carcinogenic effects.

The NOAEL of 30 mg/kg/day for oral exposure (based on hepatocyte degeneration), determined from

medium-term and long-term toxicity tests in rats, was adjusted for exposure conditions to obtain 21 mg/kg/day, and subsequently divided by a factor of 10 to account for extrapolation from sub-chronic to chronic exposure. The obtained value of 2.1 mg/kg/day was deemed to be the lowest reliable dose and was identified as the 'non - toxic level*' of the substance for oral exposure.

The 'non-toxic level*' for inhalation exposure could not be identified.

With regard to oral exposure, assuming the substance is absorbed via drinking water, the predicted maximum exposure level was 1.6 μg/kg/day. The MOE (Margin of Exposure) would be 130, when calculated from the predicted maximum exposure level and its 'non-toxic level*' of 2.1 mg/kg/day, and subsequently divided by 10 to account for extrapolation from animals to humans.

For comparison, assuming the substance is ingested via drinking water and food, the predicted maximum exposure level and the derived MOE would still be 1.6 μg/kg/day and 130 respectively, since the estimated maximum exposure level via food is 0.032 μg/kg/day on the basis of the food data in limited area reported in 1996. Therefore, no further work would be required at present to assess the health risk of this substance via oral exposure.

With regard to inhalation exposure, owing to lack of identified 'non-toxic level *', the health risk could not be assessed. For comparison, assuming that 100% of the ingested substance is absorbed, the 'non-toxic level*' of inhalation exposure, derived by converting that of oral exposure, would be 7 mg/m^3. The MOE would be 24,000, when calculated from this level and the predicted maximum exposure concentration in ambient air of approximately 0.029 μg/m^3, and subsequently divided by a factor of 10 to account for extrapolation from animals to humans. In addition, the MOE would be 1,400, calculated from the maximum concentration of 0.49 μg/m^3 based on the data in limited area reported in 2003. On the other hand, the MOE would be 58, when calculated from the predicted maximum concentration of 12 μg/m^3 in indoor air. Therefore, while collection of further information would not be required to assess the health risk of this substance via inhalation in ambient air, it would be necessary for indoor 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*' 2.1 mg/kg/day	Rat	Hepatocyte degeneration	Drinking water	1.6 μg/kg/day	MOE	130	○	○
				Groundwater	— μg/kg/day	MOE	—	×	
Inhalation	'Non-toxic level*' — mg/m^3	—	—	Ambient air	0.029 μg/m^3	MOE	—	×	(○)
				Indoor air	12 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.

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4. Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 72-h EC_{50} of 9,610 μg/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*, a 48-h EC_{50} of 26,500 μg/L for swimming inhibition in the crustacean *Daphnia magna*, and a 96-h LC_{50} of 79,300 μg/L for the fish species *Oryzias latipes* (medaka). Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 96 μg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 4,500 μg/L for growth inhibition in the green algae *P. subcapitata*, a 21-d NOEC of 63.2 μg/L for reproductive inhibition in the

crustacean *D. magna*, and a 41-d NOEC of 1,100 μg/L for growth inhibition in the fish species *O. latipes* (medaka). Accordingly, based on these chronic toxicity values and an assessment factor of 10, a PNEC of 6.3 μg/L was obtained.

The value of 6.3 μg/L obtained from the chronic toxicity to the crustacean was used as the PNEC for this substance.

Information to determine the PEC of this substance could not be obtained. As such, a judgment on ecological risk could not be made. If we assume that the value of 10 μg/L for freshwater bodies, calculated on the basis of measurements of raw water from the surface of lakes including dam lakes, is used as the PEC for seawater, the PEC/PNEC ratio is greater than 1. Moreover, estimates of emissions unreported under the PRTR Law do not estimate emissions of trihalomethane unintentionally formed in sewage treatment processes. Accordingly, efforts to collect data on this substance are needed, as are measurements of environmental concentrations.

Hazard Assessment（Basis for PNEC）			Assessment Coefficient	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)			
Crustacean *Daphnia magna*	Chronic	NOEC reproductive inhibition	10	6.3	Freshwater	－	－	×	▲
					Seawater	－	－		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	No need for further work at present.	○
	Inhalation exposure (atmosphere)	Although risk to human health could not be confirmed, collection of further information would not be required.	(○)
	Inhalation exposure (room air)	Further information collection would be required for risk characterization.	(▲)
Ecological risk	Requiring information collection.		▲

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

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

（○）: Although risk to human health could not be confirmed, collection of further information would not be required.

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