

12	CAS No.: 75-27-4	Substance: Bromodichloromethane
<p>Chemical Substances Control Law Reference No.:</p> <p>PRTR Law Cabinet Order No.: 1-381</p> <p>Molecular Formula: CHBrCl_2 Structural Formula:</p> <p>Molecular Weight: 163.83</p> <div style="text-align: center;"> $\begin{array}{c} \text{Cl} \\ \\ \text{Br}-\text{C}-\text{Cl} \\ \\ \text{H} \end{array}$ </div>		
<p>1. General information</p> <p>The aqueous solubility of this substance is 3.00×10^3 mg/1,000 g (30°C), the partition coefficient (1-octanol/water) ($\log K_{ow}$) is 2.00, and the vapor pressure is 50 mmHg (6.7×10^3 Pa) (20°C). The mean biodegradability (aerobic degradation) as determined by BOD, TOC, and GC is 35%. Its half-life for hydrolysis is 13.7–137 years (pH=8–7, calculated values).</p> <p>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 chlorination processes of wastewater and cooling water.</p> <hr/> <p>2. Exposure assessment</p> <p>Total release to the environment in fiscal 2013 under the PRTR Law was approximately 57 t, and all emissions were unreported. The majority of unreported emissions were 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 overall or the atmosphere and public water bodies in particular, the predicted proportion distributed to the atmosphere was 80.4%.</p> <p>The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was around $0.033 \mu\text{g}/\text{m}^3$. In addition, the predicted maximum exposure for indoor air was around $0.48 \mu\text{g}/\text{m}^3$ based on past data from a survey of a limited area. The maximum expected oral exposure was $1.1 \mu\text{g}/\text{kg}/\text{day}$ when calculated from potable water data and less than $0.00016 \mu\text{g}/\text{kg}/\text{day}$ when calculated from public freshwater data. Furthermore, the predicted maximum exposure calculated from potable water data and past food data from a survey of a limited area was $1.2 \mu\text{g}/\text{kg}/\text{day}$.</p> <p>The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was reported to be less than $0.004 \mu\text{g}/\text{L}$ for public freshwater bodies and generally $0.011 \mu\text{g}/\text{L}$ for seawater. Values of generally $0.067 \mu\text{g}/\text{L}$ for public freshwater bodies and $0.038 \mu\text{g}/\text{L}$ for seawater have been reported in an environmental survey of a limited area. However, the PEC was $11 \mu\text{g}/\text{L}$ at maximum in freshwater bodies on the basis of measurements of raw water from the surface of lakes, including dam lakes.</p> <hr/> <p>3. Initial assessment of health risk</p> <p>No information on acute symptoms in humans was available. The observed symptoms caused by this substance were piloerection, sedation, muscular relaxation, ataxia, exhaustion and soiled fur in rats, and sedation and sensory anesthesia in mice.</p> <p>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.</p> <p>The LOAEL of $6.1 \text{ mg}/\text{kg}/\text{day}$ for oral exposure (based on fatty degeneration in the liver), determined from medium-term and long-term toxicity tests in rats, was divided by a factor of 10 to account for uncertainty in</p>		

using LOAEL. The obtained value of 0.61 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 NOAEL of 1 ppm for inhalation exposure (based on tubular degeneration and some other effects), determined from medium-term and long-term toxicity tests in mice, was adjusted for exposure conditions to obtain 0.25 ppm (1.7 mg/m³), and subsequently divided by a factor of 10 to account for extrapolation from sub-acute to chronic exposure. The obtained value of 0.17 mg/m³ was deemed to be the lowest reliable concentration and was identified as the ‘non-toxic level*’ of the substance for inhalation exposure.

With regard to oral exposure, assuming the substance is ingested via drinking water, the predicted maximum exposure level was 1.1 µg/kg/day. The MOE (Margin of Exposure) would be 11, when calculated from the predicted maximum exposure level and the ‘non-toxic level*’ of 0.61 mg/kg/day, and subsequently divided by a factor of 10 to account for extrapolation from animals to humans and another factor of 5 to account for carcinogenicity.

On the other hand, assuming the substance is absorbed via public freshwater bodies, the predicted maximum exposure level was less than 0.00016 µg/kg/day and the derived MOE would be over 76,000.

The maximum exposure level via food was 0.052 µg/kg/day on the basis of the data reported in some areas in 1996. For comparison, assuming the substance is ingested via food and drinking water, the maximum exposure level was 1.2 µg/kg/day and the derived MOE would be 10. Therefore, collection of information on oral exposure to this substance would be required to assess the health risk of this substance via oral exposure.

With regard to inhalation exposure, the predicted maximum exposure concentration in ambient air was 0.033 µg/m³, approximately. The MOE would be 100, when calculated from this value and the ‘non-toxic level*’ of 0.17 mg/m³, and subsequently divided by a factor of 10 to account for extrapolation from animals to humans and another factor of 5 to account for carcinogenicity. On the other hand, the maximum exposure concentration in indoor air was 0.48 µg/m³ approximately, on the basis of the data reported in some areas in 1994, and the derived MOE would be 7.

Therefore, while no further work would be required to assess the health risk of this substance via inhalation in ambient air, it would be necessary for indoor air.

Exposure Path	Toxicity			Exposure assessment		Result of risk assessment			Judgment
	Criteria for risk assessment	Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure dose and concentration	MOE			
Oral	‘Non-toxic level*’ 0.61 mg/kg/day	Rat	fatty degeneration in the liver	Drinking water	1.1 µg/kg/day	MOE	11	▲	▲
				Public Freshwater bodies	<0.00016 µg/kg/day	MOE	>76,000	○	
Inhalation	‘Non-toxic level*’ 0.17 mg/m ³	Mouse	tubular degeneration and some other effects	Ambient air	0.033 µg/m ³	MOE	100	○	○
				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

With regard to acute toxicity, the following reliable data were obtained: a 72-h EC₅₀ of 11,600 µg/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*, a 48-h EC₅₀ of 29,000 µg/L for swimming inhibition in the crustacean *Daphnia magna*, a 96-h LC₅₀ of 28,200 µg/L for the fish species *Oryzias latipes* (medaka), and a 96-h EC₅₀ of 64,000 µg/L for teratogenicity in embryos of the African clawed frog *Xenopus laevis*. Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect

concentration (PNEC) of 116 µg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 802 µg/L for growth inhibition in the green algae *P. subcapitata*, and a 21-d NOEC of 2,170 µg/L for reproductive inhibition in the crustacean *D. magna*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a PNEC of 8.0 µg/L was obtained.

The value of 8.0 µg/L obtained from the chronic toxicity to the alga was used as the PNEC for this substance.

The PEC/PNEC ratio is less than 0.0005 for freshwater bodies and 0.001 for seawater. If the value of 11 µg/L calculated on the basis of measurements of raw water from the surface of lakes including dam lakes is used as the PEC for freshwater bodies, 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)			
Green algae	Chronic	NOEC Growth Inhibition	100	8.0	Freshwater	<0.004	<0.0005	○	▲
					Seawater	0.011	0.001		

5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Requiring information collection.	▲
	Inhalation exposure (atmosphere)	No need for further work at present.	○
	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.