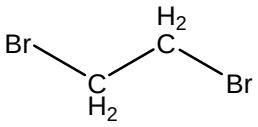
4	CAS No.: 106-93-4	Substance:1,2-Dibromoethane

Chemical Substances Control Law Reference No.: 2-59 (α,ω-Dibromoalkane (C=2–4))

PRTR Law Cabinet Order No.: 2-45

Molecular Formula: $C_2H_4Br_2$ Structural Formula:



Molecular Weight: 187.86

1. General information

The aqueous solubility of this substance is 4.12×10^3 mg/1,000 g (20°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 1.96, and the vapor pressure is 11.6 mmHg (=1.55×10^3 Pa) (25°C). Biodegradability (aerobic degradation) is characterized by a BOD degradation rate of 0% and bioaccumulation is judged to be non-existent or low. Its half-life for hydrolysis is 2.2 years (pH=7.5, 25°C).

This substance is designated as a Class 2 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 uses of this substance are in analytical chemistry and as a raw material. The production and import category under the PRTR Law is 1 to t<100 t. The production and import quantity as *α,ω*-dibromoalkane (C=2–4) in fiscal 2011 was less than 1,000 t.

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2. Exposure assessment

Because this substance is not classified 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), release and transfer quantities could not be obtained. Predictions of proportions distributed to individual media by 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 were largest.

The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was generally 0.0069 μg/m^3. Furthermore, a maximum level of generally 0.054 μg/m^3 was reported in a study of general environmental atmospheric data for a limited area.

The maximum expected oral exposure was estimated to be less than 0.00015 μg/kg/day on the basis of calculations from data for public freshwater bodies. However, a level of less than 0.002 μg/kg/day was reported in a study of potable water for a limited area. The risk of exposure to this substance by intake from an environmental medium via food is considered slight, based on its low bioaccumulation.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was less than around 0.0037 μg/L for both public freshwater bodies and seawater.

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3. Initial assessment of health risk

This substance may cause irritation to eyes, skin and respiratory tract. Inhalation exposure may cause burning sensation, coughing, labored breathing and loss of consciousness. Contact of the substance with skin may cause pain, redness and blisters, while contact with eyes may cause pain, redness and severe burns.

With regard to the substance's non-carcinogenic health risk, information on its general, reproductive and developmental toxicities on animal was available. As for carcinogenicity of the substance, there were evidences of its carcinogenic effects on animals observed in their experiments. Therefore, an initial assessment was conducted on both non-carcinogenic and carcinogenic potential risks of the substance, since it would be carcinogenic to humans as well.

With regard to oral exposure to the substance, a LOAEL of 38 mg/kg/day (for symptoms such as suppressed

weight increase, effects on liver and adrenal cortex) obtained from its mid-term and long-term toxicity tests on rats, was adjusted for their durations to provide 27 mg/kg/day for its intermittent to continuous exposure, and divided by a factor of 10 for conservative use of the LOAEL. Outcome of 2.7 mg/kg/day would be the reliable lowest dose. As no information was available on the threshold for its carcinogenicity, 2.7 mg/kg/day was deemed to be its 'non-toxic level*' on the basis of its non-carcinogenic effects. No threshold was assumed for its carcinogenicity, and 3.6 $(mg/kg/day)^{-1}$ (for forestomach tumor, etc.) obtained from experiments on rats and mice was identified as its slope factor.

As for inhalation exposure to the substance, a NOAEL of 3 ppm (for nasal cavity tumor and increased relative liver weight) obtained from its mid-term and long-term toxicity tests on rats was adjusted for their durations to provide 0.54 ppm (4.1 mg/m^3) for its intermittent to continuous exposure, and divided by a factor of 10 due to their short test periods. Outcome of 0.41 mg/m^3 was considered to be the reliable lowest dose of the substance. As no information on the threshold for its carcinogenicity was available, 0.41 mg/m^3 would be its 'non-toxic level*' on the basis of its non-carcinogenic effects. No threshold was assumed for its carcinogenicity, and 6×10^{-4} $(\mu g/m^3)^{-1}$ obtained from tests on rats (for nasal cavity tumor, hemangiosarcoma and mesothelioma) was identified as its unit risk.

With regard to oral exposure to the substance, both its mean and maximum exposure levels were predicted to be below about 0.00015 μg/kg/day, when its intakes through freshwater from public water bodies were assumed. The MOE (Margin of Exposure) would be above 180,000 when calculated from its 'non-toxic level*' of 2.7 mg/kg/day and its maximum exposure concentration predicted from animal experiments, and divided by a factor of 10 to convert animal data to human data and further divided by a factor of 10 to extrapolate animal data to the carcinogenic hazard to human. As for carcinogenicity of the substance to human, its excess incidence rate would be below 5.4×10^{-7} from the slope factor for the predicted maximum exposure concentration. As exposure to the substance in the environment through food intakes would be limited, neither the MOE nor excess incidence would not change significantly even when this exposure was included. Therefore, no further action would be required at this moment 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 approximately 0.0069 $\mu g/m^3$. The MOE would be 590 when calculated from this predicted maximum exposure concentration and its 'non-toxic level*' of 0.41 mg/m^3 from the animal experiments and divided by a factor of 10 to convert animal data to human data, and further divided by a factor of 10 to extrapolate animal data to the carcinogenic hazard to human. Its excess incidence rate would be 4.6×10^{-6} from the unit risk for the predicted maximum exposure concentration. Additionally, its maximum concentration of 0.054 $\mu g/m^3$ was reported for some area. The MOE would be 76 when calculated from this for reference, while its excess incidence rate would be 3.2×10^{-5}. Therefore, collection of further information would be required to assess health risk from inhalation exposure to the substance 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*'	2.7 mg/kg/day	Rat	Suppressed weight increase, effects on liver and adrenal cortex, etc.	Drinking water	— μg/kg/day	MOE	—	×	○
							Excess incidence rate	—	×	
					Fresh water	<0.00015 μg/kg/day	MOE	>180,000	○	
	Slope factor	3.6 $(mg/kg/day)^{-1}$	Rat/mouse	Forestomach tumor, etc.			Excess incidence rate	$<5.4\times10^{-7}$	○	

Inhalation	'Non-toxic level*' 0.41 mg/m^3		Rat	Nasal cavity tumor and increased relative liver weight	Ambient air	0.0069 μg/m^3	MOE	590	○	▲
							Excess incidence rate	4.1×10^{-6}	▲	
	Unit risk	6×10^{-4} $(mg/m^3)^{-1}$	Rat	Nasal cavity tumor, hemangiosarcoma and mesothelioma	Indoor air	— μg/m^3	MOE	—	×	×
							Excess incidence rate	—	×	

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 48-h LC_{50} of 3,610 μg/L for the crustacean *Ceriodaphnia dubia*, a 96-h LC_{50} of 4,300 μg/L for the fish species *Pimephales promelas* (fathead minnow), and a 48-h TLm of more than 40,000 μg/L for the mayfly *Cloeon dipterum*. Accordingly, based on this acute toxicity value and an assessment coefficient of 1,000, a predicted no effect concentration (PNEC) of 3.6 μg/L was obtained.

The value of 3.6 μg/L obtained from the acute toxicity to the crustacean was used as the PNEC for this substance because reliable chronic toxicity data could not be obtained.

The PEC/PNEC ratio was less than 0.001 for both freshwater bodies and seawater. Accordingly, further work is considered unnecessary at this time.

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)			
Crustacean *Ceriodaphnia dubia*	Acute	LC_{50} mortality	1,000	3.6	Freshwater	<0.0037	<0.001	○	○
					Seawater	<0.0037	<0.001		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	No need of further work at present.	○
	Inhalation exposure	Requiring information collection.	▲
Ecological risk	No need of further work at present.		○

［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.