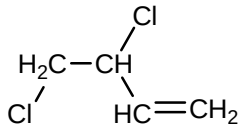
8	CAS No.: 760-23-6	Substance: 3,4-Dichloro-1-butene

Chemical Substances Control Law Reference No.: 2-118 (Dichlorobutene)

PRTR Law Cabinet Order No.:

Molecular Formula: $C_4H_6Cl_2$ Structural formula:

Molecular Weight: 125.00



1. General information

The aqueous solubility of this substance is 1.6×10^3 mg/L (20°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 2.37, and the vapor pressure is 21.9 mmHg (=2.9×10^3 Pa) (25°C). The biodegradability (aerobic degradation) is characterized by a BOD degradation rate of 11% (mean value), and bioaccumulation is thought to be nonexistent or low. Its half-life for hydrolysis is 20.9 days (25°C, pH4), 33.3 days (25°C, pH7), and 35.0 days (25°C, pH9).

In Japan, this substance is manufactured and used in closed systems as a manufacturing intermediate for chloroprene. Commercially sold polychloroprene does not contain this substance as an impurity. The production quantity of this substance in 1998 was approximately 50,000 t.

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2. Exposure assessment

Because this substance is not 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 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 greater.

Data for setting the predicted maximum exposure to humans via inhalation could not be obtained. Based on past general environmental atmospheric data, the predicted maximum exposure was less than around 0.06 μg/m^3.

Data for estimating maximum oral exposure could not be obtained. Further, albeit past data, public freshwater body data indicated an exposure of less than around 0.00044 μg/kg/day.

General environmental atmosphere and public freshwater body data recorded more than 10 years ago exist, and the manufactured quantity of the major use (polychloroprene) has remained almost constant. Taking into consideration trends in production facilities, concentrations of this substance in the general environmental atmosphere and public freshwater bodies are not believed to have changed markedly. The risk of exposure to this substance by intake from an environmental medium via food is considered slight based on estimates of oral exposure using estimated concentrations in fish species.

Data for setting the predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, could not be obtained. Further, albeit past data, general environmental atmospheric data indicated a value of less than around 0.011 μg/L for both public freshwater bodies and seawater. Data exists from measurements of public freshwater bodies taken more than 10 years ago, but concentrations are not believed to have changed markedly.

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3. Initial assessment of health risk

Protracted contact with this substance can cause dermatitis and blistering. Exposure to high concentrations of vapour can produce delayed toxic effects on the eyes, causing irritation and lacrimation several hours after the exposure.

As sufficient information was not available on the carcinogenicity of the substance, and an initial assessment was

conducted on the basis of information on its non-carcinogenic effects.

With regard to oral exposure to the substance, a NOAEL of 10 mg/kg/day（for increased liver weight, incidence of liver hypertrophy observed）obtained from mid-term and long-term toxicity tests in rats was divided by 10 due to the short test periods, and 1 mg/kg/day derived was deemed as a plausible value for the lowest dose of the substance and was identified as its 'non-toxic level*'. As for inhalation exposure, a NOAEL of 96 mg/m^3 (for increased relative liver weight and degenerative conditions in liver cells) obtained from mid-term and long-term toxicity tests in rats was adjusted to 17 mg/m^3 according to exposure conditions and then divided by 10 due to the short test periods. 1.7 mg/m^3 derived was deemed as a plausible value for the lowest dose of the substance and was identified as its 'non-toxic level*'.

As for oral exposure, the absence of information available on and exposure concentrations did not allow for a health risk assessment. For reference, however, intakes of freshwater from public water bodies would result in oral exposure of approximately less than 0.00044 μg/kg/day when calculated from the maximum concentration reported in 1997 for river water. For reference, a MOE of greater than 230,000 would be derived when calculated from oral exposure of approximately less than 0.00044 μg/kg/day and its 'non-toxic level*' of 1 mg/kg/day divided by 10 due to the need to convert the 'non-toxic level*' obtained from the animal experiments to a human equivalent dose. As historical production and usage trends of polychloroprene, or the major product of the substance, were not indicative of considerable increases in concentrations in the environment, the MOE would not be greatly affected. Also, as exposure to the substance from food intakes in the environment was estimated minor, even when the exposure through food intakes was combined, remarkable changes in the MOE would not be likely. Therefore, collection of information on oral exposure to the substance to assess health risk would not be required.

With regard to inhalation exposure to the substance, the absence of information available on and exposure concentrations did not allow for a health risk assessment. For reference, however, the maximum concentration in the ambient air reported in 1998 was approximately less than 0.06 μg/m^3. The MOE would be above 2,800 when calculated from its 'non-toxic level' of 1.7 mg/m^3 and divided by 10 due to the need to convert the 'non-toxic level*' obtained from the animal experiments to a human equivalent dose. As mentioned above, considerable increases in concentrations in the ambient air would not be likely, and, thus, the MOE would not be greatly affected. Therefore, collection of information would not be required to assess health risk from inhalation exposure to this substance in the ambient air.

Information of toxicity				Exposure assessment		Result of risk Exposure assessment			Judgment
Exposure Path	Criteria for risk assessment	Animal	Criteria for diagnoses（endpoint）	Exposure medium	Predicted maximum exposure quantity and concentration				
Oral	'Non-toxic level * ' 1 mg/kg/day	Rats	Increased liver weight, incidence of liver hypertrophy	Drinking water	－ μg/kg/day	MOE	－	×	
				Groundwater	－ μg/kg/day	MOE	－	×	(○)
Inhalation	'Non-toxic level * ' 1.7 mg/m^3	Rats	Relative liver weight increased, change in liver cell morphology	Ambient air	－ μg/m^3	MOE	－	×	(○)
				Indoor air	－ μg/m^3	MOE	－	×	×

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 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 58,100 μg/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*, a 48-h EC_{50} of 10,000 μg/L for swimming inhibition in the crustacean *Daphnia magna*, and a 96-h LC_{50} of 7,170 μg/L for the fish species *Pimephales promelas* (fathead minnow). Accordingly, based on these acute toxicity values and an assessment coefficient of 100, a predicted no effect

concentration (PNEC) of 72 μg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 10,400 μg/L for growth inhibition in the green algae *P. subcapitata* and a 21-d NOEC of 830 μg/L for reproductive inhibition in the crustacean *D. magna*. Accordingly, based on these chronic toxicity values and an assessment coefficient of 100, a predicted no effect concentration (PNEC) of 8.3 μg/L was obtained. The value of 8.3 μg/L obtained from the chronic toxicity to the crustacean was used as the PNEC for this substance.

The risk of this substance could not be judged because the predicted environmental concentration (PEC) could not be set. However, past data for public freshwater bodies and seawater indicate less than around 0.011 μg/L. The ratio of this public freshwater body concentration with PNEC is less than 0.001 for both freshwater bodies and seawater. Public freshwater body data recorded more than 10 years ago exists for this substance but concentrations are not believed to have changed markedly. Accordingly, there is minimal need to collect further data regarding this substance.

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	End point			Water body	Predicted environmental concentration PEC (μg/L)			
Crustacean *Daphnia magna*	Chronic	NOEC reproductive inhibition	100	8.3	Freshwater	－	－	×	○
					Seawater	－	－		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Though a risk characterization cannot be determined, there would be little necessity of collecting information.	(○)
	Inhalation exposure	Though a risk characterization cannot be determined, there would be little necessity of collecting information.	(○)
Ecological risk	Need to collect further data considered minimal.		○

［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.