16	CAS No.: 108-90-7	Substance: Monochlorobenzene

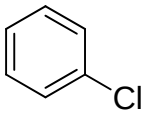
Chemical Substances Control Law Reference No.:3-31

PRTR Law Cabinet Order No.*: 1-125

Molecular Formula: C_6H_5Cl

Molecular Weight: 112.56

Structural formula:



Note: No. in Revised Cabinet Order enacted on October 1, 2009

1. General information

The water solubility of this substance is 498 mg/L (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 2.84, and the vapor pressure is 12 mmHg (=1.6×10^3 Pa) (25°C). In the aerobic biodegradation test, BOD degradation rate was 0 %. This substance is judged as a non- or low bioaccumulative. The hydrolysis half-life is more than 897 days (25°C, pH=7, extrapolated value).

This substance is designated as a Class 1 Designated Chemical Substances 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 are as a raw material for other chemical substances (raw material for vitamins, pharmaceuticals and agricultural chemicals, etc.), as a solvent for organic synthesis reactions, as an agricultural chemical adjuvant, and as a solvent for paints, inks and electronic equipment washing. The production and import quantity of this substance in FY 2009 was 6,134 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 FY 2009 under the PRTR Law was approximately 320 t, of which approximately 250 t or 78% of overall releases were reported. Among reported release destinations, the atmosphere was the largest. In addition, approximately 1,500 t was transferred to waste materials. The chemical industry reported large releases to the atmosphere and public water bodies. The largest release among releases to the environment including unreported ones was to the environment. A multi-media model used to predict the distribution into each medium in the environment indicated that in regions where the largest quantities were estimated to have been released to the environment or to the atmosphere, the proportion distributed to the atmosphere would be 94.1 %. In regions where the largest estimated releases were to public water bodies, the predicted proportions distributed to soil and water bodies would be 63.6 % and 34.9 %, respectively.

The predicted maximum exposure to humans via inhalation, based on general environmental atmospheric data, was around 0.079 μg/m^3. In addition, the predicted maximum exposure for indoor air was around 0.37 μg/m^3. Meanwhile, the annual mean value of atmospheric concentration estimated from reported releases to the atmosphere under the PRTR Law was a maximum of 32 μg/m^3. The predicted maximum oral exposure was estimated to be generally less than 0.004 μg/kg/day based on calculations from data for groundwater, and around 0.008 μg/kg/day based on calculations from data for public freshwater bodies. A predicted maximum oral exposure of around 0.008 μg/kg/day was used for this substance. Meanwhile, the maximum river concentration was 0.51 μg/L based on reported releases to public freshwater bodies under the PRTR Law. Using this estimated concentration for rivers to calculate oral exposure gives 0.02 μg/kg/day. Further, the predicted maximum oral exposure calculated from past data for food and from data for public freshwater bodies was around more than 0.008 μg/kg/day to around less than 0.2 μg/kg/day.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was around 0.2 μg/L for public freshwater bodies and around 0.03 μg/L for seawater. The maximum river concentration was estimated to be 0.51 μg/L from reported releases to public freshwater bodies under the PRTR Law.

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3. Initial assessment of health risk

This substance is irritating to eyes and skin. When orally taken, it may cause chemical pneumonitis through its pulmonary aspiration, and it may lower consciousness through its effects on the central nervous system. When inhaled it may cause drowsiness, headache, nausea, sore throat and loss of consciousness, and when orally taken, it may also cause abdominal pain. Contact of skin to this substance makes it red and dry, and contact of eyes to it makes them red and cause pain to them.

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

As for oral exposure to the substance, a NOAEL of 27.25 mg/kg/day (for symptoms such as bile duct hyperplasia in liver) obtained from mid- and long-term toxicity tests on dogs was divided by 10 due to their rather short test periods. Its outcome of 2.7 mg/kg/day was deemed to be the lowest reliable dose without any effect, and this was identified as its 'non-toxic level*'. As for inhalation exposure to the substance, a NOAEL of 50 ppm (for symptoms such as hypertrophy of hepatocytes, expansion of renal tubules) was obtained from mid- and long-term toxicity tests on rats. It was then adjusted to 13 ppm (60 mg/m^3) against exposure conditions, and divided by 10 due to their short test periods. Final outcome of 6 mg/m^3 was deemed to be the lowest reliable concentration of the substance without any effect and was identified as its 'non-toxic level*'.

As for its oral exposure, its mean exposure would be less than about 0.004 μg/kg/day and its predicted maximum exposure would be around 0.008 μg/kg/day, respectively, if its intakes through freshwater from public water bodies were assumed. The MOE would be from 34,000 when calculated from its 'non-toxic level*' of 2.7 mg/kg/day and the predicted maximum exposure, and further divided by 10 for conversion of the 'non-toxic level*' from animal experiments to an equivalent dose for humans. For reference, its concentrations in receiving river water around the major sources of its discharges estimated on the basis of its releases to public water bodies in FY2009 reported under the PRTR Law suggests that its maximum exposure would be 0.02 μg/kg/day and associated MOE would be 14,000. For information, although somewhat old, data for food intakes reported in 1999 suggest that its oral exposure would be no less than about 0.008 μg/kg/day but around less than 0.2 μg/kg/day, and associated MOE would be 1,400 to 34,000. Therefore, further actions would not be required at the moment to assess health risk from oral exposure to this substance.

As for its inhalation exposure, its mean exposure would be about 0.022 μg/m^3 and its predicted maximum exposure would be around 0.079 μg/m^3 respectively, when its concentrations in the ambient air are considered. The MOE would be 7,600 when calculated from the 'non-toxic level*' of 6 mg/m^3 and the predicted maximum exposure, and divided by 10 for conversion of the 'non-toxic level*' from animal experiments to an equivalent dose for humans. On the other hand, its releases to the ambient air reported in FY2009 under the PRTR Law suggests that its maximum annual mean concentration in the ambient air around its major sources of emissions would be 32 μg/m^3 and associated MOE would be 19. When its concentrations in the indoor air are considered, its mean exposure would be about 0.01 μg/m^3 and its predicted maximum exposure would be around 0.37 μg/m^3 respectively. The predicted maximum exposure concentration would provide MOE of 1,600. Therefore, collection of information would be required to assess health risk from inhalation exposure to this substance in the ambient air. As a part of such effort, it is desirable to measure its concentrations in the ambient air around its major sources of emissions. On the other hand, for the indoor air, further actions would not be required at the moment to assess health risk from oral exposure to this substance.

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	Dogs	Bile duct hyperplasia in liver, etc.	Drinking water	−	μg/kg/day	MOE	−	×	○
				Freshwater	0.008	μg/kg/day	MOE	34,000	○	
Inhalation	Non-toxic level * ' 6 mg/m^3	Rats	Hypertrophy of hepatocytes, expansion of renal tubule, etc.	Ambient air	0.079	μg/m^3	MOE	7,600	○	(▲)
				Indoor air	0.37	μg/m^3	MOE	1,600	○	○

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.

4.Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 96-h EC_{50} of 12,500 µg/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*; a 48-h EC_{50} of 5,290 µg/L for immobilization in the crustacean *Ceriodaphnia* cf. *dubia*; and a 48-h LC_{50} of 4,100 µg/L in the fish *Oncorhynchus mykiss* (rainbow trout). Also obtained was a 7-d EC_{50} of 353,000 µg/L for growth inhibition in the duckweed *Lemna minor*. Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 41 µg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 16-d NOEC of 320 µg/L for reproductive inhibition in the crustacean *Daphnia magna*; and a 43-d NOEC of 247 µg/L for growth inhibition in the fish *Oryzias latipes* (medaka). Also obtained was a 7-d NOEC of 294,000 µg/L for growth inhibition in the duckweed *L. minor* and fat duckweed *L. gibba*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 2.5 µg/L was obtained. This 2.5 µg/L obtained from the fish chronic toxicity was used as the PNEC for this substance.

The PEC/PNEC ratio was 0.08 for freshwater bodies and 0.01 for seawater. Accordingly, further work is thought to be unnecessary at this time. However, estimating river concentrations using reported releases under the PRTR Law indicated the possibility of locations existing with concentrations that are higher than the PEC. Accordingly, efforts are required to collect more data concerning this substance. Further, measurement of environmental concentrations taking into account PRTR data is considered necessary.

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)			
Fish (medaka)	Chronic	NOEC growth inhibition	100	2.5	Freshwater	0.2	0.08	○	▲
					Seawater	0.03	0.01		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	No need for further work.	○
	Inhalation exposure	Further information collection would be required for risk characterization.	(▲)
Ecological risk	More data collection required. Measuring environmental concentrations taking into account PRTR data considered necessary.		▲

［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.