3	CAS No.: 563-47-3	Substance: 3-Chloro-2-methyl-1-propene

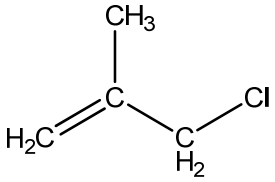
Chemical Substances Control Law Reference No.: 2-117 (monochloropropene), 2-2367

PRTR Law Cabinet Order No.: 1-131

Molecular Formula: C_4H_7Cl

Molecular Weight: 90.55

Structural Formula:



1.**General information**

The aqueous solubility of this substance is 1.4×10^3 mg/L (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 1.98, and the vapor pressure is 102 mmHg (=1.36×10^4 Pa) (20°C). The biodegradability (aerobic degradation) is characterized by a BOD degradation rate of 99%, and degradability is judged to be good. In addition, 2-methylarryl alcohol is formed through the hydrolysis of this compound.

This substance is classified as a Class 1 Designated Chemical Substance under the PRTR Law. The main uses of this substance are as a raw material for acrylic fiber dyestuff modifiers, synthetic resins, and agricultural chemicals. The production and import quantity of monochloropropene in fiscal 2016 was less than 1,000 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 fiscal 2016 under the PRTR Law was approximately 8.2 t; all releases were reported. All reported releases were to the atmosphere. In addition, 0.91 t was transferred to waste. The main source of releases was the chemical industry. A multi-media model used to predict the proportions distributed to individual media in the environment indicates that in regions where the largest quantities were estimated to have been released to the environment overall or the atmosphere in particular, the predicted proportion distributed to the atmosphere was 98.7%.

The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was around 0.025 μg/m^3. The mean annual value for the atmospheric concentration in fiscal 2016 was calculated by use of a plume-puff model on the basis of releases to the atmosphere reported under the PRTR Law; this model predicts a maximum level of 2.0 μg/m^3.

The maximum expected oral exposure could not be determined because of the lack of actual survey data for potable water, ground water, public water bodies and fresh water, food and soil. River concentrations were not estimated because there were no releases to public water bodies reported in fiscal 2016 under the PRTR Law.

The risk of exposure to this substance by intake from an environmental medium via food is considered slight, given the low bioaccumulation of the substance expected on the basis of its physicochemical properties.

Data for setting the predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, could not be obtained. There were no releases to public water bodies reported in fiscal 2016 according to the PRTR Law. On this account, river concentrations could not be calculated.

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3. Initial assessment of health risk

This substance causes lachrymation and is irritating to the eyes, skin and respiratory tract. It may cause effects on the central nervous system. Exposure at high levels may lower consciousness. Inhalation of the substance causes cough, sore

throat, headache and shortness of breath. Contact with the eyes or skin causes redness and pain.

As sufficient information on the carcinogenicity in humans was not available, it could not be determined whether the substance is carcinogenic to humans or not. However, significant and dose-dependent tumorigenesis in forestomach was observed in all dose-groups in the carcinogenesis study by oral administration in mice. Considering the above, assessment of the carcinogenic risk was deemed necessary as well, and initial assessment was conducted for both non-carcinogenic and carcinogenic effects.

The non-carcinogenic LOAEL of 75 mg/kg/day for oral exposure (based on forestomach basal cell hyperplasia and nephrosis), determined from toxicity tests in rats, was adjusted according to exposure conditions to obtain 54 mg/kg/day and subsequently divided by a factor of 10 to account for uncertainty in using a LOAEL. The calculated value of 5.4 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 cancer slope factor for oral exposure of 0.14 $(mg/kg/day)^{-1}$ (based on forestomach tumors), determined from carcinogenicity tests in mice, was adopted assuming no threshold.

The non-carcinogenic LOAELs for inhalation exposure of 50 ppm (based on decrease in the relative weight of kidneys and eosinophilic change in olfactory epithelium) and 50 ppm (based on suppression of body weight gain and eosinophilic change in respiratory epithelium), determined from toxicity tests in rats and mice respectively, were adjusted according to exposure conditions to obtain 8.9 ppm (33 mg/m^3), and subsequently divided by a factor of 10 to account for uncertainty in using a LOAEL. The calculated value of 3.3 mg/m^3 was deemed to be the lowest reliable dose and was identified as the 'non-toxic level*' of the substance for inhalation exposure. The unit risk for cancer assuming no threshold could not be identified.

With regard to oral exposure, owing to the lack of identified exposure levels, the health risk could not be assessed. The total release of the substance to the environment was reported to be approximately 8.2 t in FY 2016 under the PRTR Law. However, the release of the substance into public water bodies was reported to be 0 t, and predictions of the multimedia fugacity model indicated that the proportion distributed to water was little. Therefore, collection of further information would not 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 0.025 $\mu g/m^3$, approximately. The MOE would be 2,600, when calculated from the predicted maximum exposure concentration and the 'non-toxic level*'of 3.3 mg/m^3, and subsequently divided by a factor of 10 to account for extrapolation from animals to humans, and by another factor of 5 to take into consideration the carcinogenicity in animals. On the other hand, the maximum concentration (annual mean) in ambient air near the operators releasing large amount of this substance was estimated to be 2.0 $\mu g/m^3$ based on the releases to air reported in FY 2016 under the PRTR Law. The MOE would be 33, falling below 100, when calculated from this concentration. Therefore, collection of information would be required to assess the health risk of this substance via inhalation in ambient air, starting from data on concentrations near the operators releasing large amount of the 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*' 5.4 mg/kg/day	Rats	Forestomach basal cell hyperplasia and nephrosis	Drinking water	- μg/kg/day	MOE	-	○
						Excess incidence rate	-	
	Slope factor 0.14 $(mg/kg/day)^{-1}$	Mice	Forestomach tumors	Groundwater	- μg/kg/day	MOE	-	
						Excess incidence rate	-	

Inhalation	'Non-toxic level*' 3.3 mg/m^3		Rats Mice	Decrease in the relative weight of kidney etc. Suppression of body weight gain, etc.	Ambient air	0.025 μg/m^3	MOE	2,600	(▲)
						-	Excess incidence rate	-	
	Unit risk - (μg/m^3)$^{-1}$		-	-	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

Insufficient appropriate toxicity data exists for aquatic organisms. On this account, study and augmentation of toxicity data, including observations employing QSAR predicted values, should be implemented with the objective of assessing ecological risk in the future.

Hazard assessment (basis for PNEC)			Assessment coefficient	Predicted no effect concentration PNEC (μg/L)	Exposure assessment		PEC/ PNEC ratio	Assessment result
Species	Acute/ chronic	Endpoint			Water body	Predicted environmental concentration PEC (μg/L)		
—	—	—	—	—	Freshwater	—	—	—
					Seawater	—	—	

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	No need for further work.	○
	Inhalation exposure	Further efforts to collect data required based on comprehensive review of existing relevant data.	(▲)
Ecological risk	Insufficient appropriate toxicity data exists for aquatic organisms. On this account, study and augmentation of toxicity data, including observations employing QSAR predicted values, should be implemented with the objective of assessing ecological risk in a future phase of research.		—

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

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

（▲）: Further efforts to collect data required based on comprehensive review of existing relevant data

（■）: Candidate for further work based on comprehensive review of existing data