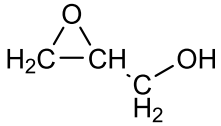
6	CAS No.: 556-52-5	Substance: 2,3-Epoxy-1-propanol

Chemical Substances Control Law Reference No.: 2-2389

PRTR Law Cabinet Order No.: 1-67

Molecular Formula: $C_3H_6O_2$ Structural Formula:

Molecular Weight: 74.08



1. General information

This substance is freely miscible with water, the partition coefficient (1-octanol/water) (log K_{ow}) is -0.95, and the vapor pressure is 0.9 mmHg (=120 Pa) (25°C). This substance is readily biodegradable.

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). The main uses of this substance are as a reactive diluent for epoxy resin or alkyd resin, a resin stabilizer, a modifier for cotton and wool, and a stainability conditioner for dyestuffs. The production and import quantity in fiscal 2009 was 432 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 2010 under the PRTR Law was 8.0 t, and all releases were reported. The major destination of reported releases was public freshwater bodies. In addition, 16 t was transferred to waste materials. The only source of reported releases is the chemical industry. 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 to the atmosphere or public freshwater bodies in particular, the predicted proportion distributed to water bodies was 99.6% in all cases.

The maximum expected concentration of exposure to humans via inhalation could not be obtained. The mean annual value for atmospheric concentration in fiscal 2010 was calculated by using a plume-puff model on the basis of reported releases to the atmosphere according to the PRTR Law; this model predicted a maximum level of 0.023 μg/m^3. The maximum expected oral exposure was reported to be less than 0.00035 μg/kg/day on the basis of calculations from data for public freshwater bodies. The risk of exposure to this substance by intake from an environmental medium via food is considered slight, based on estimates of oral exposure obtained by using estimated concentrations in fish species.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was reported to be less than 0.0087 μg/L for public freshwater bodies and generally less than 0.0087 μg/L for seawater.

3.Initial assessment of health risk

This substance may cause irritation to eyes, skin and respiratory tract. Inhalation exposure to the substance may cause coughing, sore throat, dizziness and lethargy, while oral exposure may cause abdominal pain, diarrhea, nausea and vomiting. In addition, the substance may possibly affect the central nervous system. Its contact with skin may cause redness, while its contact with eyes maycause redness and pain.

There is an evidence of its carcinogenic effects for animals observed in their experiments, and it may be carcinogenic also to human.

With regard to non-carcinogenic effects of the substance through its oral exposure, a LOAEL of 19 mg/kg/day (for suppressed body weight increase) obtained from its mid-term and long-term toxicity tests on mice was

adjusted for their durations to provide 14 mg/kg/day for its intermittent to continuous exposure and divided by a factor of 10 due to their short test periods, and further divided by a factor of 10 for the use of a LOAEL. 0.14 mg/kg/day was considered to be the reliable lowest dose of the substance. As no information on the threshold for carcinogenicity was available, the substance's 'non-toxic level*' was identified as 0.14 mg/kg/day. When no threshold was assumed for its carcinogenicity, a slope factor would be 1.3 $(mg/kg/day)^{-1}$ (for multiple organ tumor) obtained from experiments on rats.

As for non-carcinogenic effects of inhalation exposure to the substance, a LOAEL of 4 ppm (for effects on nasal tissue) obtained from its mid-term and long-term toxicity tests on mice was adjusted for their durations to obtain 0.71 ppm (2.2 mg/m^3) from its intermittent to continuous exposure, and divided by a factor of 10 for the use of a LOAEL. 0.22 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, the substance's 'non-toxic level*' was identified as 0.22 mg/m^3. Its unit risk could not be determined since no threshold was assumed for its carcinogenicity.

As for oral exposure to the substance, its maximum exposure concentration was predicted to be below 0.00035 μg/kg/day, when its intakes through freshwater from public water bodies were assumed. The MOE (Margin of Exposure) would be above 4,000 when calculated from the substance's 'non-toxic level*' of 0.14 mg/kg/day and the predicted maximum exposure concentration from animal experiments, and divided by a factor of 10 to convert animal data to human, and further divided by a factor of 10 to extrapolate animal data to human carcinogenic hazard.

As for carcinogenic potential of the substance to human, its excess incidence rate was calculated to be below 4.6×10^{-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, as its exposure concentrations were not known, its health risk could not be assessed. The MOE would be 96, or almost 100 when calculated from the substance's maximum (annual mean) concentration of 0.023 $\mu g/m^3$ in the ambient air near the operators discharging it in high concentrations, estimated from its emissions reported in FY 2010 under the PRTR Law, and its 'non-toxic level*' of 0.22 mg/m^3 from animal experiments, and divided by a factor of 10 to convert animal data to human, and further divided by a factor of 10 to extrapolate animal data to human carcinogenic hazard. Therefore, collection of further information on its oral exposure would not be required to assess health risk from its inhalation 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*' 0.14 mg/kg/day	Mouse	Suppressed body weight increase	Drinking water	— μg/kg/day	MOE	—		○
						Excess incidence rate	—	×	
	Slope factor 1.3 $(mg/kg/day)^{-1}$	Rat	Multiple organ tumor			MOE	> 4,000	○	
				Groundwater	< 0.00035 μg/kg/day	Excess incidence rate	$< 4.6\times10^{-7}$	○	
Inhalation	'Non-toxic level*' 0.22 mg/m^3	Mouse	Effects on nasal tissue	Ambient air	— $\mu g/m^3$	MOE	—		(○)
						Excess incidence rate	—	×	
	Unit risk — $(\mu g/m^3)^{-1}$	—	—			MOE	—		
				Indoor air	— $\mu g/m^3$	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

Data for this substance that would enable initial assessment of its ecological risk could not be obtained. For this reason, ecological risk could not be determined because a predicted no effect concentration (PNEC) could not be established. A 14-d LC_{50} value of 50,100 μg/L obtained from extended toxicity tests with *Poecilia reticulata* (guppy) suggested an acute toxicity in excess of this value for the guppy. Hence, when this value is divided by an assessment factor of 1,000, the interim PNEC based on the acute toxicity value exceeds 50 μg/L. A comparison of this value with the predicted environmental concentration (PEC) suggests that the ecological risk is sufficiently low for this substance. Accordingly, the need to collect further data for initial assessment of the ecological risk towards aquatic organisms is considered to be minimal.

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)			
–	–	–	–	–	Freshwater	<0.0087	–	×	○
					Seawater	<0.0087	–		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	No need for further work.	○
	Inhalation exposure	Although risk to human health could not be identified, collection of further information would not be required.	(○)
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.