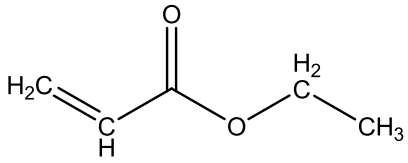
1	CAS No.: 140-88-5	Substance: Ethyl acrylate

Chemical Substances Control Law Reference No.: 2-988

PRTR Law Cabinet Order No.: 1-3

Molecular Formula: $C_5H_8O_2$ Structural Formula:

Molecular Weight: 100.12



1. General information

The aqueous solubility of this substance is 1.50×10^4 mg/1,000 g (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 1.32, and the vapor pressure is 38.6 mmHg (=5.14×10^3 Pa) (25°C). Biodegradability (aerobic degradation) is judged to be good. Its half-life for hydrolysis is 2.8 years (pH = 7).

This substance is designated as a Priority Assessment Chemical Substance and 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 raw materials for adhesives, acrylic-based paints, and acrylic rubbers. The production and import quantity in fiscal 2010 was 22,104 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 approximately 36 t, of which approximately 19 t or 51% of overall releases were reported. The major destination of reported release was the atmosphere. In addition, approximately 110 t was transferred to waste materials, and approximately 0.14 t was transferred to sewage. Industry types with large reported releases were the chemical industry and the warehousing industry for the atmosphere, and the chemical industry alone for public water bodies. The largest release among releases to the environment including those unreported was to the atmosphere. 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 in particular, the predicted proportion distributed to the atmosphere was 89.5%. In regions where the largest estimated releases were to public water bodies, the predicted proportions distributed to the atmosphere and water bodies were 65.0% and 34.4%, respectively.

The maximum expected concentration of exposure to humans via inhalation, based on an annual average of general environmental atmospheric data, was around 0.039 μg/m^3—a value smaller than the lower detection limit. Furthermore, a maximum detected level of 0.018 μg/m^3 was reported in a study of general environmental atmospheric data for a limited area. The mean annual atmospheric concentration in fiscal 2010 was also 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 0.88 μg/m^3. The maximum expected oral exposure could not be obtained. A value of around 0.0012 μg/kg/day was calculated from past data for public freshwater bodies. However, when reported releases to public freshwater bodies in fiscal 2010 according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers taking into consideration only dilution gave a maximum value of 2.7 μg/L. Using this estimated concentration for rivers to calculate oral exposure gave 0.11 μg/kg/day. 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, could not

be obtained. However, past data yielded around 0.03 μg/L for public freshwater bodies and around 0.01 μg/L for seawater. The maximum river concentration was estimated to be 2.7 μg/L from reported releases to public freshwater bodies under the PRTR Law.

3.Initial assessment of health risk

This substance may cause irritation to eyes, skin and respiratory tract. Inhalation exposure to the substance may cause burning sensation, coughing, shortness of breath and sore throat, while oral exposure may cause abdominal pain, diarrhea, nausea and vomiting. Contact of the substance with skin may cause redness and pain, while contact with eyes may cause redness, pain and blurred vision.

With regard to carcinogenic potential of the substance, an initial assessment was conducted on the basis of solely on its non-carcinogenic effects since its carcinogenicity to humans could not be identified though its carcinogenic effects on animals had been reported.

As for oral exposure to the substance, a NOAEL of 17 mg/kg/day (for increased relative stomach weight and hyperplasia of squamous epithelium of forestomach), obtained from its mid-term and long-term toxicity tests on rats, was divided by a factor of 10 due to their short test periods. 1.7 mg/kg/day was identified to be the reliable lowest dose as its 'non-toxic level*'. With regard to inhalation exposure to the substance, a NOAEL of 5 ppm (for hyperplasia of the olfactory epithelium, metaplasia of the respiratory tract, etc.), obtained from its mid-term and long-term toxicity tests on rats and mice, was adjusted for their durations to provide 0.89 ppm (3.6 mg/m^3) for its intermittent to continuous exposure. 0.89 ppm was identified to be the reliable lowest dose as its 'non-toxic level*'.

As for oral exposure to the substance, as its exposure concentrations were not known, its health risk could not be assessed. The maximum exposure was estimated to be approximately 0.0012 μg/kg/day for its oral exposure from historical data on its exposure through freshwater from public water bodies (reported in 2000). A MOE (Margin of Exposure) would be 28,000 from its animal experiments and divided by a factor of 10 to convert the animal data to human and further divided by a factor of 5 to extrapolate animal data to human carcinogenic hazard. The maximum exposure level was calculated to be 0.11 μg/kg/day from concentrations of the substance in river water with effluents from operators discharging high concentrations of the substance, reported in FY 2010 under the PRTR Law. The MOE would be 310 when calculated from this value as its reference. As exposure to the substance in the environment through food intakes would be limited, the MOE would not change significantly even when this exposure is included. Therefore, collection of further information would not be required to assess health risk from the oral exposure to the substance.

As for inhalation exposure to the substance, its maximum exposure concentration in the ambient air was predicted to be below the detection limit, though it was reported to be 0.039 $\mu g/m^3$. The MOE would be 1,800 when calculated from the substance's 'non-toxic level*' of 3.6 mg/m^3 and the maximum exposure concentration predicted from animal experiments and divided by a factor of 10 to convert animal data to human, and further divided by a factor of 5 to extrapolate animal data to human carcinogenic hazard.

In addition, the maximum (annual mean) concentration in the ambient air near the operators discharging high concentrations of the substance was calculated to be 0.88 $\mu g/m^3$ from its emissionsreported in FY 2010 under the PRTR Law. The MOE would be 82 when calculated from this value as its reference. Therefore, collection of information would be required to assess the substance's 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*'	1.7	mg/kg/day	Rat	Increased weight of stomach relative to body weight, and hyperplasia of squamous epithelium of forestomach	Drinking water	—	μg/kg/day	MOE	—	×	(○)
						Freshwater	—	μg/kg/day	MOE	—	×	
Inhalation	'Non-toxic level*'	3.6	mg/m^3	Rat Mouse	Hyperplasia of the olfactory epithelium, epithelial metaplasia of the respiratory tract	Ambient air	0.039	$\mu g/m^3$	MOE	1,800	○	(▲)
						Indoor air	—	$\mu g/m^3$	MOE	—	×	×

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.

4.Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 72-h EC_{50} of 2,260 μg/L for growth inhibition in the green alga *Pseudokirchneriella subcapitata*, a 96-h LC_{50} of 1,860 μg/L for the amphipod crustacean *Gammarus pulex*, and a 96-h LC_{50} of 1,160 μg/L for the fish species *Oryzias latipes* (medaka). Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 12 μg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 961 μg/L for growth inhibition in the green alga *P. subcapitata*, and a 21-d NOEC of 190 μg/L for reproductive inhibition or growth inhibition in the crustacean *Daphnia magna*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a PNEC of 1.9 μg/L was obtained.

The value of 1.9 μg/L obtained from the chronic toxicity to the crustacean was used as the PNEC for this substance.

Ecological risk could not be judged because data concerning environmental concentrations could not be obtained. Albeit past data, the concentration of this substance in public water bodies was around 0.03 μg/L for freshwater bodies and around 0.01 μg/L for seawater. The ratios of these concentrations to PNEC are less than 0.1 for both freshwater bodies and seawater. However, the river concentration estimated by using reported releases under the PRTR Law is 2.7 μg/L; this indicates the possibility of locations existing with concentrations that are higher than the PNEC. Accordingly, efforts must be made to collect data on this substance. Furthermore, measurement of environmental concentrations and augmentation of data related to the chronic toxicity to fish will likely need to be considered, while taking PRTR data into consideration.

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 *Daphnia magna*	Chronic	NOEC Reproductive / growth inhibition	100	1.9	Freshwater	—	—	×	▲
					Seawater	—	—		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Although risk to human health could not be confirmed, collection of further information would not be required.	(○)
	Inhalation exposure	Collection of further information would be required.	(▲)
Ecological risk	Requiring information collection.		▲

［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.