1	CAS No.: 79-10-7	Substance: Acrylic acid
Chemical S	ubstances Control Law Re	eference No.:2-984
PRTR Law	Cabinet Order No.*: 1-4 ((acrylic acid and its water-soluble salts)
Molecular I	Formula: C ₃ H ₄ O ₂	Structural formula:
Molecular	Weight: 72.06	
		СОНН
*Note: No.	in Revised Cabinet Order	enacted on October 1, 2009

1. General information

This substance is miscible in water, the partition coefficient (1-octanol/water) (log K_{ow}) is 0.35, and the vapor pressure is 4.0 mmHg (=530 Pa) (25°C). This substance is judged to be readily biodegradable (aerobic degradation). The substance is stable with respect to hydrolysis (pH=3, 7, 11).

This substance is listed as an item requiring study for drinking water quality standards. Acrylic acid and its water-soluble salts are designated as 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 polymer raw material and an acrylic ester raw material. The production (shipments) and import quantity in FY 2007 was 100,000 to <1,000,000 t/y. The production and import category under the PRTR Law is more than 100 t.

2. Exposure assessment

Total release to the environment in FY 2009 under the PRTR Law was 46 t, of which approximately 42 t or 91% of overall releases were reported. Among reported release destinations, the atmosphere was the largest. In addition, approximately 220 t was transferred to waste materials. Industry types with large reported releases were the chemical industry for the atmosphere, and the chemical industry and fiber industry for public water bodies. The largest release among releases to the environment including unreported ones was to the atmosphere. 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, the proportions distributed to soil and water bodies would be 50.1% and 42.0%, respectively.

The predicted maximum exposure to humans via inhalation, based on general environmental atmospheric data, was about 0.13 μ g/m³. Meanwhile, the annual mean value of atmospheric concentration estimated from reported releases to the atmosphere under the PRTR Law was a maximum of 4.1 μ g/m³. The predicted maximum oral exposure was estimated to be less than around 0.08 μ g/kg/day based on data from calculations for drinking water. Further, the predicted maximum exposure calculated from past data for food and data for drinking water was around 20 μ g/kg/day.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was around 2.8 μ g/L for public freshwater bodies and generally less than 0.1 μ g/L for seawater. Meanwhile, the maximum concentration in river water was estimated to be 11 μ g/L based on reported releases to public freshwater bodies under the PRTR Law.

3. Initial assessment of health risk

This substance is corrosive to skin, eyes and respiratory tract. When inhaled, it may cause coughing, sore throat, shortness of breath, burning sensation, difficulty of breathing and pulmonary edema. When orally taken, it may cause

burning sensation, weakness, stomach cramp, diarrhea, shock and loss of consciousness, and it may be corrosive. Contact of skin to the substance may cause redness, blister and pain to it. Contact of eyes to the substance may cause redness, pain, severe burn, and loss of vision.

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

As for oral exposure to this substance, a NOAEL of 53 mg/kg/day (for suppressed increase of body weight in offspring) obtained from reproductive/developmental toxicity tests on rats was deemed to be the lowest reliable dose without any effect, and this was identified as its 'non-toxic level*'. As for its inhalation exposure, a LOAEL of 5 ppm (for degeneration of the olfactory epithelium) was obtained from mid- and long-term toxicity tests on mice. It was then adjusted to 0.89 ppm (2.6 mg/m³) against the exposure condition and divided by 10 as is always the case with LOAEL. It was further divided by 10 due to their short test period. Final outcome of 0.026 mg/m³ was deemed to be the lowest reliable concentration without any effect, and this was identified as its 'non-toxic level*'.

As for its oral exposure, both its mean exposure and its predicted maximum exposure were estimated to be less than about 0.08 μ g/kg/day when its intakes through drinking water were assumed. The MOE would be more than 66,000, when calculated from the 'non-toxic level*' of 53 mg/kg/day 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, oral exposure calculated from data on exposure through food intakes in the 1999 report was around 20 μ g/kg/day, and the MOE would be 270. Therefore, further actions would not be required at the moment to assess health risk from oral exposure to this substance.

As for inhalation exposure to the substance, its mean exposure concentration was approximately $0.045 \ \mu g/m^3$ and its predicted maximum exposure concentration was approximately $0.13 \ \mu g/m^3$, when its concentrations in the ambient air were considered. The MOE would be 20 when calculated from the 'non-toxic level*' of $0.026 \ mg/m^3$ and the predicted maximum exposure concentration, and divided by 10 for conversion of the 'non-toxic level*' from animal experiments to an equivalent dose for humans. Meanwhile, the maximum annual average concentration of the substance in the atmosphere around its major sources would be $4.1 \ \mu g/m^3$ on the basis of emissions reported for FY 2009 under Japanese PRTR, and, thus, the MOE would be 0.6. Therefore, collection of information would be required to assess health risk from inhalation exposure to this substance in the ambient air. As part of such an action, concentrations of the substance in the atmosphere around its major sources should be measured.

	Toxicity Exposure assessm				ire assessmen	assessment						
Exposure Path	Criteria for risk assessment		Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure dose and concentration		Result of risk assessment			Judgment	
	Non-toxic				Suppressed body weight	Drinking water	< 0.08	µg/kg/day	MOE	> 66,000	0	
Oral	level * '	53	mg/kg/day	Rats	increase in generation of offspring	Groundwater	-	µg/kg/day	MOE	-	×	0
Inhalation	Non-toxic	0.026		Mice	Degeneration of olfactory	Ambient air	0.13	µg/m ³	MOE	20		
minalation	level * '	0.026 mg/m ³	whice	epithelium	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.

4.Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 72-h EC₅₀ of 750 μ g/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*; a 48-h EC₅₀ of 47,000 μ g/L for immobilization in the crustacean *Daphnia magna*; and a 96-h LC₅₀ of 62,000 μ g/L for the fish *Oryzias latipes* (medaka). Also obtained was a 96-h LC₅₀ of 5,487,800 μ g/L for the African clawed frog *Xenopus laevis*. Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 7.5 μ g/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 30 μ g/L for growth inhibition in the green algae *P. subcapitata*; and a 21-d NOEC of 3,800 μ g/L for reproductive inhibition in the crustacean *D. magna*. Also obtained was a 2-d NOEC of 6,250 μ g/L for reproductive inhibition in the marine rotifer *Brachionus calyciflorus*. No chronic value for fish was obtained, but because the algae was thought to have the highest sensitivity, an assessment factor of 10 was applied and a predicted no effect concentration (PNEC) of 3 μ g/L was obtained. This 3 μ g/L obtained from algae chronic toxicity was used as the PNEC for this substance.

The PEC/PNEC ratio was 0.9 for freshwater bodies and less than 0.03 for seawater. Accordingly, more data collection is considered required. More details need to be understood regarding this substance, including efforts to understand the transition of production, import quantities and PRTR data, and prevalent concentrations in public water bodies.

Hazard A	Assessment (Basis for I	PNEC)		Predicted no	E	Exposure Assessment		Judgment based	
Species	Acute/ chronic	Endpoint	Assessment factor	effect concentration PNEC (µg/L)	Water body	Predicted environmental concentration PEC (µg/L)	PEC/PNEC ratio	on PEC/PNEC ratio	Assessment result
Green algae	Chronic	NOEC	10	3	Freshwater	2.8	0.9		
uigue	Sinc	growth inhibition		-	Seawater	<0.1	< 0.03		

5. Conclusions

		Judgment						
Health risk	Oral exposure	No need for further work.	0					
	Inhalation exposure	Requiring information collection.						
Ecological risk	Data collection considered necessary.							
[Risk judgmen	ts] O: No nee	d for further work A: Requiring information collection						
■: Candidates for further work ×: Impossibility of risk characterization								
 (○) : Though a risk characterization cannot be determined, there would be little necessity collecting information. (▲) : Further information collection would be required for risk characterization. 								