

1. General information

The aqueous solubility of this substance is 320 mg/1,000 g (25° C), the partition coefficient (1-octanol/water) (log Kow) is 2.95, and the vapor pressure is 6.1 mmHg (= 810 Pa) (25° C). Biodegradability (aerobic degradation) is judged to be good. The substance does not have any hydrolyzable groups.

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 use of this substance is as a raw material for synthetic resins (polystyrene resin, ABS resin, AS resin, unsaturated polyester, etc.). The production and import quantity in fiscal 2012 was 2,429,955 t. The production and import category under the PRTR Law is more than 100 t.

2. Exposure assessment

Total release to the environment in fiscal 2012 under the PRTR Law was approximately 3,600 t, of which 2,200 t or 61% of overall releases were reported. The major destination of reported releases was the atmosphere. In addition, approximately 0.46 t was transferred to sewage and approximately 1,900 t was transferred to waste materials. Industry types with large reported releases were plastic product manufacturing, the chemical industry, electrical machinery manufacturing, shipbuilding and repair, ship engine manufacturing, and transportation equipment manufacturing for the atmosphere, and the chemical industry alone for public water bodies. The largest release among releases to the environment including unreported releases 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 97.3%. In regions where the largest estimated releases were to soil, the predicted proportion distributed to the atmosphere was 96.9%.

The maximum expected concentration of exposure to humans via inhalation, based on ambient air, was around 2.8 μ g/m³. In addition, the maximum expected concentration of exposure for indoor air was around 130 μ g/m³. The mean annual value for atmospheric concentration in fiscal 2012 was calculated by using a plume-puff model on the basis of releases to the atmosphere reported according to the PRTR Law; this model predicted a maximum level of 33 μ g/m³. The maximum expected oral exposure was around 0.0016 μ g/kg/day when calculated from potable water data and around less than 0.0016 μ g/kg/day when calculated from public freshwater body data. Furthermore, when releases to public freshwater bodies in fiscal 2012 reported according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers by taking into consideration only dilution gave a maximum value of 0.1 μ g/L. Using this estimated concentration for rivers to calculate oral exposure gave 0.004 μ g/kg/day. However, the maximum

expected exposure calculated from potable water or public freshwater body data and past data for food was around 0.4 μ g/kg/day in either case. The exposure level to this substance by intake from an environmental medium via food is considered slight, given the low bioaccumulation of the substance expected on basis of its physicochemical properties.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was less than around 0.04 μ g/L for both public freshwater bodies and seawater. However, albeit past data, values of 1 μ g/L for public freshwater bodies (1998) and around 0.34 μ g/L for seawater (1986) have been reported. When reported releases to public freshwater bodies in fiscal 2012 according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers while taking into consideration only dilution gave a maximum value of 0.1 μ g/L.

3. Initial assessment of health risk

This substance causes irritation to the eyes, skin and respiratory tract. Chemical pneumonitis may occur if the substance is swallowed in its liquid form and reaches the lungs. The substance may possibly affect the central nervous system. When inhaled, dizziness, lethargy, headache, nausea, vomiting, enervation and loss of consciousness may occur, while vomiting and nausea may occur when ingested. Contact of the substance with the eyes or skin may cause redness and pain.

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

With regard to the oral exposure to the substance, the NOAEL of 12 mg/kg/day (based on inhibition of body weight gain), obtained for mid-term and long-term toxicity tests on rats, was identified to be the reliable lowest dose as its 'non-toxic level*'. As for the inhalation exposure to the substance, the NOAEL of 4 ppm (based on color vision deficiency), resulted from observation of effects on humans, was adjusted according to the test conditions to obtain the exposure of 0.8 ppm (3.4 mg/m³) .This value was considered to be the reliable lowest dose of the substance and was identified as its 'non-toxic level*'.

Regarding the oral exposure to the substance, the predicted maximum exposure level was approximately 0.0016 μ g/kg/day, assuming drinking water was ingested. The MOE of 150,000 was derived from the substance's 'non-toxic level*' of 12 mg/kg/day and the predicted maximum exposure, and after the division by a factor of 10 to convert animal data to human data and further by a factor of 5 due to the carcinogenic properties of the substance. Assuming the ingestion of water from public water bodies and freshwater, the predicted maximum exposure level was approximately 0.0016 μ g/kg/day, and the MOE would be more than 150,000. In addition, the MOE of 60,000 was derived from the maximum exposure level of 0.004 μ g/kg/day, derived itself from the estimations of concentrations in effluents from high discharging plants, based on the emissions reported in FY 2012 under the PRTR Law. The exposure level in the environment through diet was considered as low, and the oral exposure level was approximately 0.4 μ g/kg/day, according to the maximum value of the exposure concentrations through diet, as reported in FY 1997; and the MOE derived from this level would be 600. Therefore, no further action would be required at present to assess the health risk for the oral exposure to this substance.

Concerning inhalation exposure to the substance, the predicted maximum exposure concentration in ambient air was approximately 2.8 μ g/m³. The MOE of 240 was derived from the substance's 'non-toxic level*' of 3.4 mg/m³ and the predicted maximum exposure concentration, and after the division by a factor of 5 due to the carcinogenic properties of the substance. The maximum concentration in the atmosphere near the high discharging plants area was 33 μ g/m³ (annual mean), estimated from the reported emissions into the environment in FY 2012 under the PRTR Law, and the MOE derived from this concentration was 21. As for concentrations in

indoor air, the predicted maximum exposure concentration was approximately 130 μ g/m³, and the corresponding MOE was 5. Therefore, collection of further information would be required to assess the health risk for the inhalation exposure to this substance in ambient air, and the substance is considered to be a candidate for further work concerning inhalation exposure in indoor air.

Toxicity						Exposure assessment						
Exposure Path	Criteria fo	Criteria for risk assessment Animal Criteria for diagnoses (endpoint) Exposure medium Predicted maxim exposure dose a concentration			l maximum e dose and ntration	Result of risk assessment			Judgment			
Oral	'Non-toxic	12	mg/kg/day	Rat	Inhibition of body weight gain	Drinking water	0.0016	µg/kg/day	MOE	150,000	0	0
	level*'	12				Groundwater	< 0.0016	µg/kg/day	MOE	>150,000	0	
Inhalation	'Non-toxic	3.4	mg/m ³	Human	Color vision deficiency	Ambient air	2.8	µg/m ³	MOE	240	0	(▲)
	level*'					Indoor air	130	μg/m ³	MOE	5		

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 96-h EC₅₀ of 720 μ g/L for growth inhibition in the green alga *Pseudokirchneriella subcapitata*, a 96-h LC₅₀ of 2,990 μ g/L in the crustacean *Gammarus pseudolimnaeus* (northern spring amphipod), a 96-h LC₅₀ of 4,020 μ g/L for the fish species *Pimephales promelas* (fathead minnow), and a 96-h LC₅₀ of 97,000 μ g/L for the gastropod *Amphimelania holandri* (freshwater snail). Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 7.2 μ g/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 96-h NOEC of 63 μ g/L for growth inhibition in the green alga *P. subcapitata*, and a 21-d NOEC of 1,010 μ g/L for reproductive inhibition in the crustacean *Daphnia magna*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a PNEC of 0.63 μ g/L was obtained.

The value of 0.63 μ g/L obtained from the chronic toxicity to the alga was used as the PNEC for this substance.

The PEC/PNEC ratio is less than 0.06 for both freshwater bodies and seawater. When releases to public freshwater bodies in fiscal 2012 reported according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers by taking into consideration only dilution gave a maximum value of $0.1 \mu g/L$, suggesting that the PEC/PNEC ratio may exceed 0.1 at certain locations. Accordingly, efforts to collect data on this substance are needed, as are measurements of environmental concentrations by taking PRTR data into consideration.

Hazard A		Predicted no	Ε	Exposure Assessment		Judgment			
Species	Acute/ chronic	Endpoint	Assessment Coefficient	effect concentration PNEC (µg/L)	Water body	Predicted environmental concentration PEC (µg/L)	PEC/PNEC ratio	based on PEC/PNEC ratio	Assessment result
Green algae	Chronic	NOEC growth inhibition	100	0.63	Freshwater	<0.04	<0.06	0	•
					Seawater	<0.04	<0.06	Ŭ	

5. Conclusions							
	Conclusions						
	Oral exposure	No need for further work at present.	0				
Health risk	Inhalation exposure (Ambient air)	Collection of further information would be required for risk characterization.	(▲)				
	Inhalation exposure (Indoor air)The substance is considered to be a candidate for further work.						
Ecological risk	Requiring information collection.						
[Risk judgmer	nts] O: No need f	or further work A: Requiring information collection					
■: Candidates for further work ×: Impossibility of risk characterization							
 (○) : Although risk to human health could not be confirmed, collection of furth information would not be required. (▲) : Further information collection would be required for risk characterization. 							