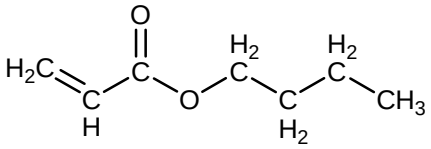
2	CAS No.: 141-32-2	Substance: Butyl acrylate

Chemical Substances Control Law Reference No.: 2-989 (alkyl acrylates (C=3–4))

PRTR Law Cabinet Order No.: 1-7

Molecular Formula: $C_7H_{12}O_2$ Structural Formula:

Molecular Weight: 128.17



1. General information

The aqueous solubility of this substance is 1.4×10^3 mg/L (20°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 2.36, and the vapor pressure is 5.48 mmHg (=731 Pa) (25°C). Biodegradability (aerobic degradation) is judged to be good. Furthermore, the degradation rate by hydrolysis is less than 2% after 28 d (pH 7) and the half-life is 1,100 d (calculated value).

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 acrylic esters are acrylic fiber, fiber processing, paints, paper processing, adhesives, leather processing, and acrylic rubber. The production and import quantity in fiscal 2010 was 136,495 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, and all releases were reported. The major destination of reported release was the atmosphere. In addition, approximately 240 t was transferred to waste materials, and 0.082 t was transferred to sewage. Industry types with large reported releases were the chemical industry and the plastic products manufacturing industry for the atmosphere, and the chemical industry alone for public water bodies. 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 proportion distributed to the atmosphere was 92.9%. In regions where the largest estimated releases were to public water bodies, the proportion distributed to water bodies was 90.3%.

The maximum expected concentration of exposure to humans via inhalation, based on an annual average of general environmental atmospheric data, was around 0.042 μg/m^3. Furthermore, a past report of 0.075 μg/m^3 for a limited area exists (Tokyo Prefecture). 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 1.1 μg/m^3. The maximum expected oral exposure could not be obtained. However, a value of around less than 0.0004 μg/kg/day was calculated from past groundwater data. 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 dilution only gave a maximum value of 1.4 μg/L. Using this estimated concentration for rivers to calculate oral exposure gave 0.056 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 less than 0.01 μg/L for both public freshwater bodies and seawater. The maximum river concentration was estimated to be 1.4 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 a burning sensation, coughing, shortness of breath and sore throat, while its oral exposure may cause abdominal pain, nausea, vomiting and diarrhea. Redness and pain may be caused by contact of the substance with skin or eyes. Chemical pneumonia may occur when liquid of the substance is swallowed to lungs.

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

With regard to oral exposure to the substance, a NOAEL of 84 mg/kg/day (for increased relative liver weight), 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. 8.4 mg/kg/day was considered to be the reliable lowest dose of the substance, and this was identified as its 'non-toxic level*'.

With regard to inhalation exposure, a LOAEL of 14 ppm (for atrophy/hyperplasia of olfactory epithelium, etc.), obtained from its mid-term and long-term toxicity tests on rats, was adjusted for their durations to provide 2.5 ppm (13 mg/m^3) for its intermittent to continuous exposure, and divided by a factor of 10 for the use of a LOAEL. 1.3 mg/m^3 was identified to be the reliable lowest dose as its 'non-toxic level*'.

With regard to oral exposure to the substance, as its exposure concentrations were not known, its potential health risk could not be assessed. As for oral exposure to the substance, the maximum exposure concentration was estimated to be below 0.0004 μg/kg/day from historical data (reported in 2000) on its exposure through groundwater. The MOE (Margin of Exposure) would be above 2,100,000 when calculated from this value as its reference and the substance's 'non-toxic level*' of 8.4 mg/kg/day from its animal experiments divided by a factor of 10 to convert animal data to human. In addition, the maximum exposure level was calculated to be 0.056 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 15,000 when calculated from this value. 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 potential health risk from oral exposure to the substance.

With regard to inhalation exposure to the substance, its maximum exposure concentration in the ambient air was predicted to be around 0.042 $\mu g/m^3$. The MOE would be 3,100 when calculated from the substance's 'non-toxic level*' of 1.3 mg/m^3 and the maximum exposure concentration predicted from its animal experiments and divided by a factor of 10 to convert animal data to human. The maximum exposure level was estimated to be 0.075 mg/m^3 from historical data (reported in 1999) for some locality. The MOE would be 1,700 when calculated from this value as its reference. In addition, the maximum (annual mean) concentration in the ambient air near the operators discharging high concentrations of the substance was calculated to be 1.1 $\mu g/m^3$ from its emissions reported in FY 2010 under the PRTR Law. The MOE would be 120 when calculated from this value as its reference. Therefore, no further action would be required at this moment 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*' 8.4 mg/kg/day	Rat	Increased relative liver weight	Drinking water	— μg/kg/day	MOE	—	×	(○)
				Freshwater	— μg/kg/day	MOE	—	×	
Inhalation	'Non-toxic level*' 1.3 mg/m^3	Rat	Atrophy/hyperplasia of olfactory epithelium, etc.	Ambient air	0.042 μg/m^3	MOE	3,100	○	○
				Indoor air	— μg/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 96-h EC_{50} of 2,650 μg/L for growth inhibition in the green alga *Pseudokirchneriella subcapitata*, a 48-h EC_{50} of 5,230 μg/L for swimming inhibition in the crustacean *Daphnia magna*, and a 96-h LC_{50} of 2,100 μg/L for the fish species *Cyprinodon variegatus* (sheepshead minnow). Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 21 μg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 96-h NOEC of less than 1,800 μg/L for growth inhibition in the green alga *P. subcapitata*, and a 21-d NOEC of 1,000 μg/L for reproductive inhibition in the crustacean *D. magna*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a PNEC of 10 μg/L was obtained.

The value of 10 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 is less than 0.01 μg/L for both freshwater bodies and seawater. The ratios of these concentrations to PNEC are less than 0.001. In addition, the river concentration estimated by using reported releases according to the PRTR Law and taking only dilution into consideration gives 1.4 μg/L, resulting in a ratio to PNEC that only slightly exceeds 0.1. Accordingly, further work is considered unnecessary at this time.

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 inhibition	100	10	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	No need for further work.	○
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.