23	CAS No.: 61788-33-8	Substance: Polychlorinated terphenyls

Chemical Substances Control Law Reference No.:

PRTR Law Cabinet Order No.:

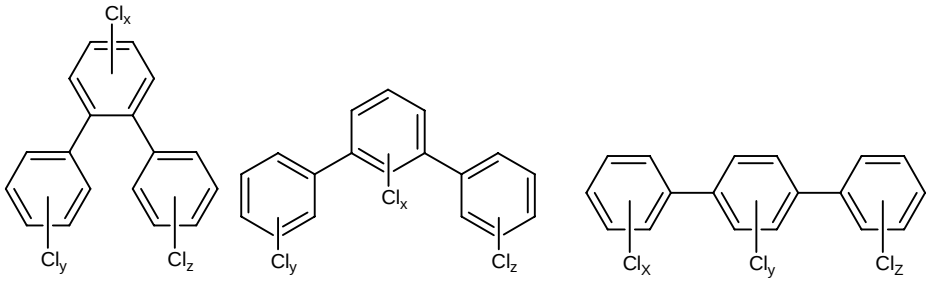
Structural Formula: (x + y + z = 1 - 14)

Molecular Formula:

$C_{18}H_{14-n}Cl_n$(n=1～14)

Molecular Weight:

264.75～712.53



o-Polychlorinated terphenyls　*m*-Polychlorinated terphenyls　*p*-Polychlorinated terphenyls

1. General information

This substance is considered to be insoluble in water. There is a report that the degradability is expected to be persistent.

The production and shipping of this substance was commanded to stop on July 20, 1972, and the instruction was made to withdraw this substance already shipped. The accumulated quantities of production and import were 2,620 tons and 140 tons, respectively. Regard to the destinations, 1/4 of total quantity was for electric apparatus, and 3/4 was for the use in the open system as glue, paint and ink. It was never used for pressure-sensitive paper.

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2. Exposure assessment

As this substance is not 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), release and transfer quantities could not be obtained. The composition of Polychlorinated terphenyls has not been known well, the properties were different depending on the rate of chloridezation. Therefore, the estimation of distribution ratio into individual medium was not carried out.

The predicted maximum exposure concentration for inhalation exposure to human beings was 0.000005 μg/m^3. The predicted maximum oral exposure was estimated to be less than 0.111217 μg/kg/day.

The predicted environmental concentration (PEC) that indicates exposure to aquatic organisms was estimated to be generally less than 0.00042 μg/L for freshwater and approximately less than 0.00042 μg/L for seawater public water bodies.

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3. Initial assessment of health risk

There was no information of the acute toxic symptoms in human beings. In animal testing, its acute toxicity was lower than that of PCB. The acute toxicity of both this substance and PCB tended to decrease in parallel with the increase in the number of chlorine in the molecule by the oral administration in rats. However, there was no such correlation between acute toxicity and number of chlorine in the experiment of percutaneous exposure in rabbits.

There was insufficient information regarding the carcinogenicity of the substance. For this reason, an initial assessment of the substance was conducted based on information of non-carcinogenic effects.

As the ‘Non-toxic level’ for oral exposure, the LOAEL of 2.6 mg/kg/day (nodular hyperplasia in liver) was obtained from the medium- and long-term toxicity testing for mice. As this is a NOAEL, it is divided by 10, and a value of 0.26 mg/kg/day was derived as the ‘Non-toxic level’. For inhalation exposure, the ‘Non-toxic level’ could not be estimated.

With regard to oral exposure, in case of intakes of freshwater public water bodies and food, the predicted maximum

exposure was less than 0.11 μg/kg/day. The MOE of exceeding 240 was derived from the 'Non-toxic level' of 0.26 mg/kg/day divided by the predicted maximum dose, and divided by 10, because the 'Non-toxic level' was established by means of animal testing. Accordingly, further action for assessment of its health risk from oral exposure to this substance would not be required at present.

For the inhalation, because its 'Non-toxic level' was not determined, and the exposure concentrations were not estimated, its health risk could not be identified. As a reference, assuming that the absorption rate is 100 %, and the 'Non-toxic level' for the oral exposure is converted to the 'Non-toxic level' for the inhalation, the value is 0.77 mg/m^3. The MOE determined from this figure and the predicted maximum concentration of inhalation is 15,000,000. Accordingly, there would be low necessity of collecting information on inhalation exposure to this substance in the ambient air for its health risk assessment at present.

Information of toxicity				Exposure assessment		Result of risk assessment			Judgment
Exposure path	Criteria for risk assessment	Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure quantity and concentration				
Oral	'Non toxic level' 0.26 mg/kg/day	Mice	Nodular hyperplasia in liver	Drinking water / food	– μg/kg/day	MOE	–	×	○
				Freshwater / food	< 0.11 μg/kg/day	MOE	> 240	○	
Inhalation	'Non toxic level' – mg/m^3	–	–	Ambient air	0.000005 $μg/m^3$	MOE	–	×	×
				Indoor air	– $μg/m^3$	MOE	–	×	×

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4. Initial assessment of ecological risk

For each species, reliable information has not been obtained, and the PNEC value of this substance could not be calculated.

At this point, the ecological risk could not be determined. Over 30 years have been passed since the production of this substance was stopped, and the environmental concentration is generally below 0.00042 μg/L. Accordingly, there is thought to be little need to give priority for the ecotoxicity tests to fill up the information.

Hazard assessment (basis for PNEC)			Assessment factor	Predicted no effect concentration PNEC (μg/L)	Exposure assessment		PEC/ PNEC ratio	Result of assessment
Species	Acute / chronic	Endpoint			Water body	Predicted environmental concentration PEC (μg/L)		
–	–	–	–	–	Freshwater	< 0.00042	–	×
					Seawater	< 0.00042	–	

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	No need of further work.	○
	Inhalation exposure	Impossible of risk characterization. There is thought to be comparatively little need to collect the information, etc.	×
Ecological risk	Impossible of risk characterization. There is thought to be little need to give priority for the ecotoxicity tests to fill up the information.		×

［Risk judgments］ ○: No need of further work ▲: Requiring information collection
■: Candidates for further work ×: Impossible of risk characterization

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.