1 9 CAS No.: 115-86-6

Substance: Triphenyl phosphate

Chemical Substances Control Law Reference No.: 3-2522 (as triphenyl [or monomethylphenyl, dimethylphenyl, nonylphenyl] phosphate) and 3-3363 (as tris [phenyl, monomethylphenyl, dimethylphenyl, ethylphenyl, nonylphenyl mixture] phosphate)

PRTR Law Cabinet Order No.:

Molecular Formula: C<sub>18</sub>H<sub>15</sub>O<sub>4</sub>P Structural Formula: Molecular Weight: 326.28



## 1. General information

The aqueous solubility of this substance is 1.9 mg/L (25°C), and the partition coefficient (1-octanol / water) (log Kow) is 4.59. The vapor pressure is 6.28 x  $10^{-6}$  mmHg (= 8.37 x  $10^{-4}$  Pa) (25°C, extrapolated value). Degradability is judged to be good. In terms of hydrolyzability, the half-life at 27°C is 30 - 300 days (pH = 8 - 7). The half-life at 21 ± 2°C is 7.5 days (pH = 8.2) and 1.3 days (pH = 9.5).

The primary applications for this substance are as a plasticizer to provide flame retardant capability, transparency, water resistance, softness and non-adhesive properties to cellulose acetate film, as a plasticizer to provide tensile strength to nitrate cellulose film, as a plasticizer to provide softness, gasoline resistance and mineral oil resistance to synthetic rubber, as a flame retardant plasticizer for phenol resin laminate, and as a flame retardant and plasticizing agent for engineering plastics. Production and import quantities in FY2001 came to 1,000 - 10,000 tons as triphenyl (or monomethylphenyl, dimethylphenyl, nonylphenyl) phosphate.

\_\_\_\_\_

## 2. Exposure assessment

As triphenyl phosphate 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 to the environment could not be obtained. When predictions of distribution ratios by medium were made using the Mackay-Type Level III Fugacity Model, in the event of equal release to the atmosphere, water and soil, the distribution ratio was highest for soil.

It was not possible to obtain the data needed to establish a predicted maximum exposure concentration for inhalation exposure to human beings. However, it has been reported that when the data for a limited area (Kawasaki City) was used, the predicted maximum exposure concentration was approximately 0.0035  $\mu$ g/m<sup>3</sup>. In addition, it has been reported that the value based on data for indoor air was approximately 0.0151  $\mu$ g/m<sup>3</sup>. When data for groundwater and food are used, the predicted maximum oral exposure was estimated to be more than 0.0016  $\mu$ g/kg/day but less than 0.022  $\mu$ g/kg/day.

The predicted environmental concentration (PEC) that indicates exposure to aquatic organisms was estimated to be  $0.06 \mu g/L$  for freshwater and less than  $0.01 \mu g/L$  for seawater public water bodies.

-----

## 3. Initial assessment of health risk

No information could be obtained with regard to acute symptoms in humans. However, it has been reported that, when administered orally to rat, mouse and cat, the substance caused diarrhea, tremors, drowsiness, hyperactivity, changes in righting reflex, muscle weakness and other symptoms.

There is insufficient information regarding the carcinogenicity of the substance, and it is not possible to make a judgment as to whether it causes cancer in human beings. For this reason, an initial assessment of the substance was

conducted based on information of non-carcinogenic effects.

As the level at which no adverse effect, etc. was observed, used to estimate the margin of exposure (MOE), a no observed adverse effect level (NOAEL) of 161 mg/kg/day (suppression of weight increase), obtained from rat medium- and long-term toxicity testings, was obtained for oral exposure. As the test period was short, this value was divided by 10 to establish a value of 16 mg/kg/day. For inhalation exposure, a NOAEL value for humans of 3.5 mg/m<sup>3</sup> (concentration at which no health effects were observed) was obtained. This value was corrected to match the exposure circumstances, with the result that a value of  $0.7 \text{ mg/m}^3$  was established.

With regard to oral exposure, when intake of groundwater and food was postulated, the maximum predicted exposure was estimated to be more than 0.0016  $\mu$ g/kg/day but less than 0.022  $\mu$ g/kg/day. As the 'Non-toxic level' of 16 mg/kg/day and the maximum predicted exposure were established by means of animal testing, the value was divided by 10 to derive an MOE of more than 73,000 but less than 1,000,000. Accordingly, assessment of the health risk from oral exposure to this substance is thought to be unnecessary at this time.

With regard to inhalation exposure, data on a national level could not be obtained, and so the health risk could not be determined. When calculations were performed for reference purposes using general environmental data that had been reported for local areas, the predicted maximum value was estimated at 0.0035  $\mu$ g/m<sup>3</sup>, and the MOE derived from the 'Non-toxic level' of 0.7 mg/m<sup>3</sup> and the predicted maximum value was 200,000. When calculations were performed in the same manner using indoor air data for local areas, the predicted maximum value was estimated at 0.015  $\mu$ g/m<sup>3</sup>, and the MOE was 47,000. Although there was a high incidence of detection of the substance in local areas in both the ambient air and indoor air, as the MOE derived for reference purposes is sufficiently high, there is thought to be comparatively little need to determine the concentrations of the substance in ambient air and indoor air.

	К	nowledge of toxici	ty		Expo	sure assessmer	nt				
Exposure	Guidelir	nes for risk	Animal	Impact	Exposure	Predicted	maximum				
path	asse	essment		assessment	medium	exposure q	uantity and	F	Result of risk assessmen	t	Judgment
				guideline (endpoint)		concer	ntration				
Oral	No observed	16 mg/kg/dov	Bot	Suppression	Drinking water / food	-	$\mu$ g/kg/day	MOE	—	×	0
Orai	adverse effect level	16 mg/kg/day	Rai	increase	Groundwater / food	$0.0016 \sim$ $0.022$	$\mu$ g/kg/day	MOE	73,000 $\sim$ 1,000,000	0	0
Inholation	No observed	$0.7 \text{ mg/m}^3$	Humon	Concentration at which no health	Ambient air	-	$\mu$ g/m <sup>3</sup>	MOE	-	×	×
malation	adverse effect level	0.7 mg/m	numan	effects were observed	Indoor air	_	$\mu$ g/m <sup>3</sup>	MOE	_	×	×

## 4. Initial assessment of ecological risk

With regard to acute toxicity, reliable information of a 96-hour  $EC_{50}$  growth inhibition value of 2,000 µg/L was found for the algae *Pseudokirchneriella subcapitata*, a 96-hour  $EC_{50}$  value of 250 µg/L was found for the crustacea *Gammarus pseudolimnaeus* (amphipod), a 96-hour  $LC_{50}$  value of 400 µg/L was found for the fish *Oncorhynchus mykiss* (rainbow trout), and a 48-hour  $EC_{50}$  growth inhibition value of 360 µg/L was found for the chironomid *Chironomus riparius*. Accordingly, an assessment factor of 100 was used, and a predicted no effect concentration (PNEC) of 3 µg/L was obtained based on the acute toxicity values. With regard to chronic toxicity, reliable information of a 72-hour no observed effect concentration (NOEC) growth inhibition value of 980 µg/L was found for the crustacea *D. magn*, and a 30-day NOEC of 87 µg/L at death was found for the fish *Pimephales promelas* (fathead minnow). Accordingly, an assessment factor of 10 was used, and a PNEC value of 9 µg/L was obtained based on the chronic toxicity values. As the PNEC for the substance, a value of 3 µg/L obtained from the acute toxicity for the crustacea was used. The PEC/PNEC ratio was 0.02 for freshwater bodies and less than 0.003 for seawater bodies. Accordingly,

Hazard asse	essment (l	basis for PNEC)		Due diete due e	Exposure	assessment			
Species c	Acute / chronic	Endpoint	Assessment factor	effect concentration PNEC (µg/L)	Water body	Predicted environmental concentration PEC (µg/L)	PEC/PNEC ratio	Result of assessment	
Crustacea	Acute	FC	100	3	Freshwater	0.06	0.02	0	
Clusiacea	Acule		100	5	Seawater	< 0.01	< 0.003	0	
Conclusio	ns			Conclusion	18			Judgment	
Conclusio	ns Ora	l exposure	Assessm	Conclusion ent is thought	ns t to be unne	cessary at thi	s time.	Judgment	
<b>Conclusio</b> Health risk	ns Ora Inha	l exposure alation exposur	Assessm Risk cou re compara this subs	Conclusion ent is thought ild not be dete tively little ne tance.	ns t to be unne ermined. Th eed to deter	ccessary at thi ere is though mine concent	is time. It to be trations for	Judgment	