

1. General information

The aqueous solubility of this substance is 2×10^{-3} mg/1000 g (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 5.73, and the vapor pressure is 6.23×10^{-9} mmHg (= 8.3×10^{-7} Pa) (25°C). The biodegradability (aerobic degradation) is characterized by a BOD degradation rate of 6% (mean) and 0% (mean). Furthermore, the substance does not have any hydrolyzable groups.

The main uses of materials that may contain chrysene are as tar products raw materials, rust prevention coatings, fishing net dyestuffs, lamp black, and fuel in the case of coal tar; road paving in the case of paving tar; and roof coatings, cast iron pipe coatings, waterproof coatings, electrode caking materials, and fuel in the case of prepared tar. The production, export and import quantities for Japan in 2009 were 1,359,425 t, 103,412 t, and 9,467 t, respectively.

2. Exposure assessment

Because 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. Predictions of distribution by medium using a Mackay-type level III fugacity model indicated that if equal quantities were released to the atmosphere, water bodies, and soil, the proportion distributed to soil would be greater.

Data for setting the predicted maximum exposure to humans via inhalation could not be obtained. Further, albeit past data, general environmental atmospheric data indicated a value of around $0.0028 \,\mu g/m^3$.

The predicted maximum oral exposure was estimated to be less than around 0.0008 μ g/kg/day based on calculations from data for groundwater. Further, there is a report of 0.013 μ g/kg/day calculated from food data for a limited area.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was less than around $0.02 \mu g/L$ for both freshwater bodies and seawater.

3. Initial assessment of health risk

No information was available on acute poisoning via the oral or inhalation routes, but an LD_{50} of 320 mg/kg of intraperitoneal administration to mice has been reported.

There was no sufficient information available on the non-carcinogenicity of the substance. There was sufficient evidence of its carcinogenicity from animal experiments, and its potential carcinogenicity on human has been indicated. However, these findings of the carcinogenicity were from subcutaneous, intraperitoneal and intrapulmonary administrations, not from oral or inhalation administrations. Neither slope factor nor unit risk could be identified for the substance. Therefore, neither its 'non-toxic level' of exposure on the assumption that there was a threshold for its toxicity, nor its slope factor or unit risk on the assumption that there was no threshold for its carcinogenicity could be identified as a result.

With regard to oral and inhalation exposure to the substance, its 'non-toxic levels*' could not be identified, its

predicted maximum exposure concentrations could not be identified, and, thus, its potential health risk could not be identified.

According to the draft assessment by U.S.EPA, a cancer potency factor of the substance will be 0.04 to 0.2 when the cancer potency factor of benzo(a)pyrene (BaP) is presumed to be 1. Since the slope factor and unit risk of BaP are $7.3(mg/kg/day)^{-1}$ and $8.7 \times 10^{-2} (\mu g/m^3)^{-1}$ respectively, according to Vol. 5 of the Initial Assessment, its slope factor and unit risk will be as follows;

Slope factor: $2.9 \times 10^{-1} (mg/kg/day)^{-1}$ to $1.5(mg/kg/day)^{-1}$

Unit risk: $3.5 \times 10^{-3} (\mu g/m^3)^{-1}$ to $1.7 \times 10^{-2} (\mu g/m^3)^{-1}$

As for oral exposure to the substance, the predicted maximum exposure was less than approximately 0.0008 μ g/kg/day when intakes through groundwater were assumed, and the excess cancer incidence associated with this would be less than 2.3×10⁻⁷ to less than 1.2×10⁻⁶. The maximum exposure of 0.013 μ g/kg/day would be obtained from intakes through food at some location, and the excess cancer incidence associated with this would be 3.8×10⁻⁶ to 1.6×10⁻⁵. Similarly, for reference, the excess cancer incidence associated with its inhalation exposure would be 3.8×10⁻⁶ to 1.6×10⁻⁵ based on the maximum concentration of 0.0028 μ g/m³ in the ambient air reported in 1999. Although the exposure concentrations of the substance were not up to date, historical production and usage trends of the substance were not indicative of considerable increases in concentrations in the environment, and, thus, remarkable changes in the excess cancer incidence would not be likely. Therefore, collection of information would be necessary to assess health risk from oral and inhalation exposure to the substance. Rather than the carcinogenicity of chrysene alone, its relative carcinogenic potency compared to other PAHs congeners presently under discussion in foreign countries would need to be identified.

	Information of tox	icity		Exposu					
Exposure Path	Criteria for risk assessment	Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure quantity and concentration	Re	esult of risk Expo assessment	sure	Judgment
Oral	'Non-toxic — mg/kg/day level*'	-	_	Drinking water Groundwater	— μg/kg/day < 0.0008 μg/kg/day	MOE MOE		×××	(▲)
Inhalation	'Non-toxic – mg/m ³	-	_	Ambient air Indoor air	— μg/m ³ — μg/m ³	MOE 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 96-h EC₅₀ of 0.63 μ g/L for growth inhibition in the algae *Phaeodactylum tricornutum* and a 48-h EC₅₀ of 3.97 μ g/L for swimming inhibition in the crustacean *Daphnia magna*. Accordingly, based on these acute toxicity values and an assessment coefficient of 100, a predicted no effect concentration (PNEC) of 0.0063 μ g/L was obtained. Because reliable chronic toxicity data could not be obtained, the value of 0.0063 μ g/L obtained from the acute toxicity to the algae was used as the PNEC for this substance.

The PEC/PNEC ratio was less than 3 for both freshwater bodies and seawater. Accordingly, ecological risk cannot be judged. Lowering of the detection limit and elucidating the environmental concentration for this substance is considered necessary, as are efforts to augment toxicity data.

Hazard as	ssessment (basis for PNEC)			Predicted no	Exposure assessment			Judgment	
Species	Acute/ chronic	End point	Assessment	essment effect efficient concentration PNEC (µg/L)	Water body	Predicted environmental concentration PEC (µg/L)	PEC/ PNEC ratio	based on PEC/PNEC ratio	Assessment result
Green		EC ₅₀			Freshwater	< 0.02	<3		
algae	Acute	growth inhibition	100	0.0063	Seawater	< 0.02	<3	×	

5. Conclusions

		Judgment				
	Oral exposure	Further information collection would be required for risk				
	Orar exposure	characterization.	(▲)			
Health risk	Inhalation	Further information collection would be required for risk				
	exposure	characterization.	(▲)			
Ecological	Lowering of de					
risk	considered nec					
[Risk judgment	ts] (): No need	l for further work A: Requiring information collection				
Candidates for further work X: Impossibility of risk characterization						
(\bigcirc) : Though a risk characterization cannot be determined, there would be little necessity of						
collecting information.						
(\blacktriangle) : Further information collection would be required for risk characterization.						