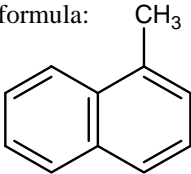


12	CAS No.: 90-12-0	Substance: 1- Methyl-naphthalene
<p>Chemical Substances Control Law Reference No.:4-80 (mono and dimethyl-naphthalene) PRTR Law Cabinet Order No.*: 1-438 (methyl-naphthalene)</p> <p>Molecular Formula: C₁₁H₁₀ Structural formula: </p> <p>Molecular Weight: 142.20</p> <p>Note: No. in Revised Cabinet Order enacted on October 1, 2009</p>		
<p>1. General information</p> <p>The water solubility of this substance is 28.1 mg/1000g (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 3.87, and the vapor pressure is 0.067-0.07 mmHg (=8.9-9 Pa) (25°C). This substance is judged not to be biodegradable, and not to be bioaccumulative. Furthermore, the substance does not have any hydrolyzable groups.</p> <p>Methyl-naphthalene is designated as 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 naphthoic acid, fluorescent whitening agents, and surfactants. The main uses of methyl-naphthalene are as a raw material for dyestuff dispersants and heat transfer oils, and as a solvent for agricultural chemical. The production and import quantity of this substance in FY 2009 was 13,641 t.</p> <p>-----</p> <p>2. Exposure assessment</p> <p>Because methyl-naphthalene was not classified as a Class 1 Designated Chemical Substance prior to revision of substances regulated by the 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.</p> <p>The predicted maximum exposure to humans via inhalation, based on general environmental atmospheric data, was reported to be 0.14 µg/m³. The predicted maximum oral exposure was estimated to be around 0.00018 µg/kg/day based on data from public freshwater bodies. The risk of exposure to this substance by intake from an environmental medium via food is considered slight based on estimates of oral exposure using estimated concentrations in fish.</p> <p>The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was around 0.0046 µg/L for public freshwater bodies and around 0.0031 µg/L for seawater.</p> <p>-----</p> <p>3. Initial assessment of health risk</p> <p>This substance is irritating to eyes. Contact of skin with the substance makes it red, and contact of eyes to it makes them red and causes pain to them. When mice were forced to inhale the substance for 6 minutes, their respiratory rates decreased against its concentrations, and its RD₅₀, or concentration to reduce their respiratory rates by 50%, was 129 mg/m³.</p> <p>As sufficient information was not available on carcinogenicity of the substance, an initial assessment was conducted on the basis of the information on its non-carcinogenic effects.</p> <p>As for oral exposure to the substance, a LOAEL of 72 mg/kg/day (for pulmonary alveolar proteinosis) was obtained from mid- and long-term toxicity tests on mice. It was then divided by 10 as is always the case with LOAEL. Final outcome of 7.2 mg/kg/day was deemed to be the lowest reliable dose without any effect, and this was identified as its</p>		

‘non-toxic level*’. As for inhalation exposure, its ‘non-toxic level*’ could not be identified.

As for its oral exposure, its mean exposure would be about 0.000072 µg/kg/day and its predicted maximum exposure would be around 0.00018 µg/kg/day, respectively, if its intakes through freshwater from public water bodies were assumed. The MOE would be 4,000,000 when calculated from the ‘non-toxic level*’ of 7.2 mg/kg/day and the predicted maximum exposure, and divided by 10 for conversion of the ‘non-toxic level*’ from animal experiments to an equivalent dose for humans. Since risk of exposure to this substance through food intakes from the environment would be limited, even when this exposure were combined, significant changes in the MOE would not be likely. Therefore, further actions would not be required at the moment to assess health risk from oral exposure to this substance.

As for its inhalation exposure, lack of available information on its ‘non-toxic levels*’ did not allow its health risk assessment. For reference, if 100% absorption were assumed, its ‘non-toxic level*’ for oral exposure would be converted to its ‘non-toxic level*’ of 24 mg/m³ for inhalation exposure. The MOE would be 17,000 when calculated from its ‘non-toxic level’ of 1.3 mg/m³ and its predicted maximum concentration of 0.14 µg/m³. Therefore, collection of information would not be required to assess health risk from inhalation exposure to the substance.

Exposure Path	Toxicity			Exposure assessment			Result of risk assessment			Judgment
	Criteria for risk assessment	Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure dose and concentration					
Oral	Non-toxic level * * 7.2 mg/kg/day	Mice	Pulmonary alveolar proteinosis	Drinking water	—	µg/kg/day	MOE	—	×	○
				Freshwater	0.00018	µg/kg/day	MOE	4,000,000	○	
Inhalation	Non-toxic level * * — mg/m ³	—	—	Ambient air	0.14	µg/m ³	MOE	—	×	(○)
				Indoor air	—	µg/m ³	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 72-h EC₅₀ of 2,850 µg/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*; a 48-h LC₅₀ of 1,420 µg/L for the crustacean *Daphnia magna*; and a 96-h LC₅₀ of 5,660 µg/L for the fish *Oryzias latipes* (medaka). Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 14 µg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 454 µg/L for growth inhibition in the green algae *P. subcapitata*; and a 21-d NOEC of 223 µg/L for reproductive inhibition in the crustacean *D. magna*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 2.2 µg/L was obtained. This 2.2 µg/L obtained from the crustacean chronic toxicity was used as the PNEC for this substance.

The PEC/PNEC ratio was 0.002 for freshwater bodies and 0.001 for seawater. Accordingly, further work is thought to be 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 <i>Daphnia magna</i>	Chronic	NOEC reproductive inhibition	100	2.2	Freshwater	0.0046	0.002	○	○
					Seawater	0.0031			

5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	No need for further work.	○
	Inhalation exposure	Though a risk characterization cannot be determined, there would be little necessity of collecting information.	(○)
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.