13	CAS No.: 91-57-6	Substance: 2-Methylnaphthalene

Chemical Substances Control Law Reference No.:4-80 (mono and dimethylnaphthalene)

PRTR Law Cabinet Order No.*: 1-438 (methylnaphthalene)

Molecular Formula: $C_{11}H_{10}$

Molecular Weight: 142.20

Structural formula:

Note: No. in Revised Cabinet Order enacted on October 1, 2009

1. General information

The water solubility of this substance is 25 mg/1000g (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 4.00, and the vapor pressure is 0.055 mmHg (=7.3 Pa) (25°C). In the aerobic biodegradation test, degradation rate was 61.9%. Furthermore, the substance does not have any hydrolyzable groups.

Methylnaphthalene is designated as a Class 1 Designated Chemical Substances 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 this substance are as a raw material for Vitamin K3 and β -naphthoic acid. The main uses of methylnaphthalene are as a raw material for dyestuff dispersants and heat transfer oils, and as a solvent for agricultural chemical. The production (shipments) and import quantity of mono- and di-methylnaphthalenes in FY 2007 was 1,000 to <10,000 t.

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2. Exposure assessment

Because methylnaphthalene 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 higher.

The predicted maximum exposure to humans via inhalation, based on general environmental atmospheric data, was reported to be 0.44 µg/m³. The predicted maximum oral exposure was estimated to be around 0.00036 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.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was around 0.009 µg/L for public freshwater bodies and around 0.0047 µg/L for seawater.

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3. Initial assessment of health risk

This substance is irritating to eyes. Inhalation of this substance causes coughing. Contact of eyes with this substance makes them red and causes pain. When mice were forced to inhale the substance for 6 minutes, their respiratory rates decreased against its concentrations, and its RD_{50}, or concentration to reduce their respiratory rates by 50%, was 67 mg/m³. When it was administered intraperitoneally to mice at 200 mg and 400mg, damages were observed in their pulmonary bronchiole (primarily for Clara cells) for mice with 400 mg of administration, but not for those with 200 mg of administration.

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.

As for oral exposure to the substance, a LOAEL of 50.3 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 5 mg/kg/day was deemed to be the lowest reliable dose without any effect, and this was identified as

its '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 less than about 0.00011 µg/kg/day and its predicted maximum exposure would be less than around 0.00036 µg/kg/day, respectively, if its intakes through freshwater from public water bodies were assumed. The MOE would be 1,400,000 when calculated from the 'non-toxic level*' of 5 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 17 mg/m^3 for inhalation exposure. The MOE would be 3,900 when calculated from its 'non-toxic level' of 1.3 mg/m^3 and its predicted maximum concentration of 0.44 µg/m^3 in the ambient air. 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 * '	5 mg/kg/day	Mice	Pulmonary alveolar proteinosis	Drinking water	—	µg/kg/day	MOE	—	×	○
					Freshwater	0.00036	µg/kg/day	MOE	1,400,000	○	
Inhalation	Non-toxic level * '	— mg/m^3	—	—	Ambient air	0.44	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.

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4. Initial assessment of ecological risk

With regard to acute toxicity, the following reliable data were obtained: a 72-h EC_{50} of 1,920 µg/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*; a 48-h EC_{50} of 1,390 µg/L for immobilization in the crustacean *Daphnia magna*; and a 96-h LC_{50} of 1,456 µg/L for the fish *Oncorhynchus mykiss* (rainbow trout). 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 283 µg/L for growth inhibition in the green algae *P. subcapitata*; and a 21-d NOEC of 233 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 2.3 µg/L was obtained µg/L. This 2.3 µg/L obtained from the crustacean chronic toxicity was used as the PNEC for this substance.

The PEC/PNEC ratio was 0.004 for freshwater bodies and 0.002 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 *Daphnia magna*	Chronic	NOEC reproductive inhibition	100	2.3	Freshwater	0.009	0.004	○	○
					Seawater	0.0047	0.002		

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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.