10	CAS No.: 123-31-9	Substance: Hydroquinone								
Chemical S	Substances Control Law Reference	ce No.:3-543 (dihydroxybenzene)								
PRTR Law	PRTR Law Cabinet Order No.*: 1-336									
Molecular	Formula: C ₆ H ₆ O ₂	Structural formula: OH								
Molecular	Weight: 110.11	OH								
Note: No.	Note: No. in Revised Cabinet Order enacted on October 1, 2009									

1. General information

The water solubility of this substance is $7.0 \times 10^4 - 7.33 \times 10^4$ mg/L (25°C), the partition coefficient (1-octanol/water) (log K_{ow}) is 0.59, and the vapor pressure is 6.70×10^{-4} mmHg (=0.0893 Pa) (25°C) (extrapolated value). This substance is judged to be readily biodegradable (aerobic degradation). Furthermore, this substance does not have any hydrolyzable groups.

This substance 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 uses are as a photograph developing agent, as a raw material for dyestuffs and pigments, as a monomer polymerization suppressor, and as a rubber antioxidant. The production and import quantity in FY 2009 was 13,586 t. The production and import category under the PRTR Law is more than 100 t.

2. Exposure assessment

Total release to the environment in FY 2009 under the PRTR Law was 48 t, of which approximately 3.4 t or 7% of overall releases were reported. The major destination of reported releases was public freshwater bodies. In addition, 72 t was transferred to waste materials. The major source of reported releases was the chemical industry. The largest release among releases to the environment including unreported ones was to water bodies. A multi-media model used to predict the distribution into each medium in the environment indicated that in regions where the largest quantities were estimated to have been released to the environment, the proportion distributed to water bodies would be 91.7%.

Data for setting the predicted maximum exposure to humans via inhalation could not be obtained. Meanwhile, the mean value of atmospheric concentration estimated from reported releases to the atmosphere under the PRTR Law was a maximum of $0.011 \ \mu g/m^3$. The predicted maximum oral exposure was estimated to be around $0.0018 \ \mu g/kg/day$ based on calculations from data for public freshwater bodies. Meanwhile, the maximum river concentration was 4.3 $\mu g/L$ based on reported releases to public freshwater bodies under the PRTR Law. Using this estimated concentration for rivers to calculate oral exposure gives 0.17 $\mu g/kg/day$. 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 0.046 μ g/L for public freshwater bodies and around 0.058 μ g/L for seawater. The maximum river water concentration calculated from reported releases to public freshwater bodies under the PRTR Law was estimated to be 4.3 μ g/L.

3. Initial assessment of health risk

This substance is highly irritating to eyes, skin and respiratory tract. When orally taken, the substance causes dizziness, headache, nausea, shortness of breath, convulsion, vomiting, and tinnitus. Inhalation of the substance causes coughing and exertional dyspnea. Contact of eyes with the substance makes them red and causes pain and blurred

vision. Contact of skin with the substance makes it red. It has been reported that LDLo, TDLo and TCLo of the substance for humans are 29 mg/kg, 170 mg/kg (for coma, increased pulse rate, and cyanosis), and 1% (for allergic dermatitis), respectively.

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 NOAEL of 15 mg/kg/day (for suppressed body weight increase and tremor) obtained from mid- and long-term toxicity tests on rats was divided by 10 due to their rather short test periods. Its outcome of 1.5 mg/kg/day derived was deemed to be the lowest reliable dose without any effect, and this was identified as its 'non-toxic level*'. As for inhalation exposure to the substance, its 'non-toxic levels*' could not be identified.

As for its oral exposure, its mean exposure would be about 0.00076 µg/kg/day and its predicted maximum exposure would be around 0.0018 µg/kg/day, respectively, if its intakes through freshwater from public water bodies and through soil were assumed. The MOE would be 83,000 when calculated from the 'non-toxic level*' of 1.5 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. For reference, its concentrations in receiving river water around its major sources were estimated from its releases to public water bodies reported in FY 2009 under the PRTR Law, and it was suggest that its maximum exposure would be 0.17 µg/kg/day and associated MOE would be 880. 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*' and exposure concentrations did not allow its health risk assessment. For information, the half life of the substance in the ambient air would be 2.8 to 28 hours. Its discharge to water bodies would account for 99% of its total release to the environment, and most of the substance would be allocated in media other than the ambient air in the environment. For reference, if 100% absorption were assumed, its 'non-toxic level*' for oral exposure would be converted to its 'non-toxic level*' of 5 mg/m³ for inhalation exposure. The MOE would be 45,000 when calculated from the 'non-toxic level' of 5 mg/m³ and its maximum annual average concentration of $0.011 \,\mu\text{g/m}^3$ in the ambient air reported for FY2009 under Japanese PRTR. Therefore, collection of information would not be required at the moment to assess health risk from inhalation exposure to this substance in the ambient air.

			Toxicity			Exposu	re assessmen	t					
Exposure Path	 Criteria for risk assessment 		Animal	Criteria for diagnoses (endpoint)	Exposure medium	exposure dose and		Result of risk assessment			Judgment		
Quil	Non-toxic	1.5		Dette	Suppressed body weight	Drinking water	-	µg/kg/day	MOE	—	×	0	
Oral	level * '	mg/kg/day	Rats	increase, tremor	Freshwater	0.0018	µg/kg/day	MOE	83,000	0	0		
Tabalada a	Non-toxic				_	Ambient air	-	µg/m ³	MOE	—	×	(())	
Inhalation	level * '	— mg/m³	_	-	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 53 μ g/L for growth inhibition in the green algae *Pseudokirchneriella subcapitata*; a 48-h EC₅₀ of 61 μ g/L for immobilization in the crustacean *Daphnia magna*; and a 96-h LC₅₀ of 97 μ g/L for the fish *Oncorhynchus mykiss* (rainbow trout). Also obtained was a 24-h LC₅₀ of 240 μ g/L for the marine rotifer *Brachionus calyciflorus*. Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 0.53 µg/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 1.5 μ g/L for growth inhibition in the green algae *P. subcapitata*; and a 21-d NOEC of 2.9 μ g/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 0.015 μ g/L was obtained. This 0.015 μ g/L obtained from the algae chronic toxicity was used as the PNEC for this substance.

The PEC/PNEC ratio was 3 for freshwater bodies and 4 for seawater. For this reason, the substance is considered to be a candidate for detailed assessment.

Hazard Assessment (Basis for PNEC)				Predicted no	Exposure Assessment			Judgment based	
Species	Acute/ chronic	Endpoint	Assessment factor	effect concentration PNEC (µg/L)	Water body	Predicted environmental concentration PEC (µg/L)	PEC/PNEC ratio	on PEC/PNEC ratio	Assessment result
Green algae	Chronic	NOEC	100	0.015	Freshwater	0.046	3		
Sieten argue	Cimoline	growth inhibition	100	0.015	Seawater	0.058	4		

5. Conclusions

	Conclusions								
Health risk	Oral exposure	No need for further work.	0						
neatui fisk	Inhalation exposure	Though a risk characterization cannot be determined, there would be little necessity of collecting information.	(())						
Ecological risk	Candidates for	Candidates for further work.							
[Risk judgme	ents] (): No nee	d for further work A: Requiring information collection							
	: Candid	ates for further work ×: Impossibility of risk characterization							
	(\bigcirc) : The	ough a risk characterization cannot be determined, there would be li	ittle necessity						
	collecting	information.							
(\blacktriangle) : Further information collection would be required for risk characterization.									