14	CAS No.: 7440-62-2(Vanadium)	Substance: Vanadium and its compounds

Chemical Substances Control Law Reference No.:

PRTR Law Cabinet Order No.: 1-321 (Vanadium compounds)

Element Symbol: V

Atomic Weight: 50.94

1. General information

Vanadium, vanadium (IV) oxide, and vanadium (III) oxide are insoluble in water. The aqueous solubilities of vanadium (V) pentoxide, ammonium metavanadate (V), and sodium metavanadate (V) are 700 mg/1,000 g (25°C), 4.8×10^4 mg/1,000 g (20°C), and 2.1×10^5 mg/1,000 g (25°C), respectively. Sodium metavanadate (V) and vanadium oxysulfate (IV) are soluble in water. Vanadium oxytrichloride (V) is thought to hydrolyze in the presence of moisture to form vanadium oxide and hydrochloric acid. Potassium vanadate (V) is almost insoluble in cold water. At temperatures lower than 63°C, vanadium (IV) tetrachloride is thought to gradually break down into vanadium trichloride and chlorine, and vanadium (IV) oxydichloride is also believed to break down gradually. Vanadium pentoxide is difficult to break down and bioaccumulation is judged to be low.

Vanadium compounds are designated as 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). Uses of metallic vanadium and vanadium alloys include electronic materials, encapsulant materials, heat-resistant materials, superalloys, and aircraft components. Uses of vanadium steel include turbines for nuclear reactors and turbo engines, cutting tools such as drills, pipelines, tanks, and bridges. Vanadium pentoxide is primarily used as a raw material for metallic vanadium, vanadium alloys, and ferrous alloys such as vanadium steel. In addition, vanadium pentoxide is used as a catalyst for producing sulfuric acid, phthalic acid, and maleic acid; a battery material; a raw material for phosphors; and other materials. Vanadium compounds other than vanadium pentoxide are used as catalysts for synthetic rubber (EPDM) manufacture and surface treatment agents.

The production (shipments) and import quantity in fiscal 2007 for vanadium pentoxide was 100 to <1,000 t/y. The production and import category under the PRTR Law for vanadium pentoxide is 1 to <100 t.

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2. Exposure assessment

Total release of vanadium compounds to the environment in fiscal 2010 under the PRTR Law was 27 t, of which approximately 21 t or 79 % of overall releases were reported. The major destination of reported releases was public water bodies. In addition, 1,000 t was transferred to waste materials and 2.9 t was transferred to sewage. Industry types with large reported releases were the chemical industry and the steelmaking industry for both the atmosphere and public water bodies. The largest release among releases to the environment including those unreported was to water bodies. Predicting the proportions distributed to individual media was not considered appropriate because the chemical forms of vanadium in the environment are not fully understood. Accordingly, the proportions distributed to individual media for vanadium were not predicted.

The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was around 0.0096 μg/m^3. However, the mean annual value for atmospheric concentration in fiscal 2010 was calculated by using a plume-puff model on the basis of reported releases to the atmosphere according to the PRTR Law; this model predicted a maximum level of 0.31 μg/m^3.

The maximum expected oral exposure was reported to be around 0.04 μg/kg/day on the basis of calculations from data for public freshwater bodies. However, the maximum expected oral exposure calculated by using past groundwater and soil data for a limited survey area was 4.2 μg/kg/day. The oral exposure calculated by dividing the daily intake quantity—itself calculated by using the metal element content in foods listed in the National

Health and Nutrition Survey of Japan food group annex list (aggregate of 13 out of a total of 17 broad categories) and the intake quantities for each food group—by a body weight of 50 kg, was 0.94 μg/kg/day for a limited survey area. Adding oral exposure from past groundwater data and soil data as well gave 5.1 μg/kg/ day.

When reported releases to public freshwater bodies in fiscal 2010 according to the PRTR Law were divided by the ordinary water discharge of the national river channel structure database, estimating the concentration in rivers while taking into consideration only dilution gave a maximum value of 4.8 μg/L. Using this estimated concentration for rivers to calculate oral exposure gave 0.19 μg/kg/day.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was reported to be 1.0 μg/L for public freshwater bodies and 2.0 μg/L for seawater. However, in a study of a limited area, a value of 18 μg/L was reported for public freshwater bodies. The maximum river concentration was estimated to be 4.8 μg/L from reported releases to public freshwater bodies under the PRTR Law.

3.Initial assessment of health risk

Vanadium pentoxide may cause irritation to eyes, skin and respiratory tract. Exposure to it in high concentrations may possibly lead to pulmonary edema, bronchitis and bronchospasms. Its inhalation exposure may cause sore throat, coughing, burning sensation, shortness of breath, labored breath and wheezing, while its oral exposure may cause abdominal spasms, lethargy, nausea, loss of consciousness and vomiting. Contact of the substance with skin causes redness, burning sensations and pain to it, while its contact with eyes causes pain, redness and conjunctivitis to them. Vanadium trioxide and sodium ammonium vanadate may also cause irritation to eyes, skin and respiratory tract. LDLo of sodium tetravanadate for human has been reported to be 1 mg/kg (for intravenous administration).

Although there were evidences of carcinogenic effects by vanadium pentoxide onexperimental animals, an initial assessment was conducted only on the basis of its non-carcinogenic effects since they were not sufficient to assess carcinogenic potential of the substance for human.

With regard to oral exposure to the substance, a LOAEL of 5 mg/kg/day of sodium metavanadate (pentavalent vanadium compounds, or 2.1 mg/kg/day as vanadium, for suppressed body weight increase in rat pups and decreased relative liver and spleen weights;) obtained from its reproductive and developmental toxicity tests on rats was divided by a factor of 10 to convert animal data to human, and further divided by a factor of 10 for the use of a LOAEL. 0.21 mg/kg/day was identified to be the reliable lowest dose of the substance as its ‘non-toxic level*’. Even though information was available also for human orally administered with vanadium oxysulfate, or tetravalent vanadium compound, assessment of the health effects of vanadium on the basis of toxicity evaluations of pentavalent vanadium compounds would be most appropriate, since pentavalent vanadium compounds have higher toxicity than tetravalent vanadium compounds and the pentavalent vanadium compounds are the most common vanadium compound in the ambient air. With regard to inhalation exposure, a LOAEL of 0.5 mg/m^3 for vanadium pentoxide (for degenerated lungs, pharynx or nasal tissue) obtained from its mid-term and long-term toxicity tests on rats was adjusted for their durations to provide 0.089 mg/m^3 (0.05 mg/m^3 as vanadium) for its intermittent to continuous exposure, and divided by a factor of 10 for the use of a LOAEL. 0.005 mg/m^3 was identified to be the reliable lowest dose of the substance as its ‘non-toxic level*’.

Additionally as for oral exposure to the substance, its maximum exposure was predicted to be 0.04 μg/kg/day when intakes of freshwater from public water bodies were assumed. The MOE (Margin of Exposure) would be 110 when calculated from its ‘non-toxic level* of 0.21 mg/kg/day and the maximum exposure predicted from animal experiments, which was divided by a factor of 10 to convert animal data to human data and further divided by a factor of 5 to extrapolate animal data to human carcinogenic hazards. In addition, the MOE would be 1.0 when calculated from its maximum exposure of 4.2 μg/kg/day from the historical groundwater and soil

data for some area. Furthermore, the MOE would be 0.8 when calculated from a total exposure of 5.1 μg/kg/day, which includes an exposure of 0.94μg/kg/day of its metallic elements through food intakes. In addition, its maximum exposure was calculated to be 0.19 μg/kg/day from its concentrations in river water with effluents from operators discharging it in high concentrations, reported in FY 2010 under the PRTR Law, and this maximum exposure would provide the MOE of 22. Therefore, collection of further information would be required to assess its health risk.

With regard to its inhalation exposure, , its maximum exposure concentration in the ambient air was predicted to be approximately 0.0096 μg/m^3. The MOE would be 10 when calculated from its 'non-toxic level* of 0.005 mg/m^3 and its maximum exposure predicted from animal experiments, and divided by a factor of 10 to convert animal data to human and further divided by a factor of 5 to extrapolate animal data to human carcinogenic hazards. Its maximum (annual mean) concentration in the ambient air was calculated to be 0.31 μg/m^3 near operators with its emissions in high concentrations reported in FY 2010 under the PRTR Law, and this maximum concentration would provide the MOE of 0.3. Therefore, collection of further information would be required at this moment to assess health risk from its inhalation exposure in the ambient air.

Toxicity				Exposure assessment		Result of risk assessment			Judgment
Exposure Path	Criteria for risk assessment	Animal	Criteria for diagnoses (endpoint)	Exposure medium	Predicted maximum exposure dose and concentration				
Oral	'Non-toxic level*' 0.21 mg/kg/day	Rat	Suppressed body weight increase in pups and decreased liver and spleen weights relative to body weight	Drinking water	— μg/kg/day	MOE	—	×	(▲)
				Freshwater	0.04 μg/kg/day	MOE	110	○	
Inhalation	'Non-toxic level*' 0.005 mg/m^3	Rat	Degenerated lungs, pharynx or nasal tissue	Ambient water	26 μg/m^3	MOE	10	▲	▲
				Indoor air	— μg/m^3	MOE	—	×	×

Non-toxic level *

- When a LOAEL is available, it is divided by 10 to obtain a NOAEL-equivalent level.
- 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 17,000 μgV/L for growth inhibition in the green alga *Pseudokirchneriella subcapitata*, a 48-h LC_{50} of 1,600 μgV/L for the crustacean *Daphnia magna*, a 96-h LC_{50} of 2,200 μgV/L for the fish species *Gila elegans* (bonytail chub), and a 96-h LC_{50} of 211 μgV/L for the sludge worm *Tubifex tubifex*. Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 16 μgV/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 72-h NOEC of 3,100 μgV/L for growth inhibition in the green alga *P. subcapitata*, a 23-d NOEC of 1,900 μgV/L for reproductive inhibition in the crustacean *D. magna*, a 30-d NOEC of 41 μgV/L for post-hatching second-generation growth (dry weight) in the fish species *Jordanella floridae* (flagfish), and a 10-d NOEC of 1,000 μgV/L for population reproductive inhibition in the freshwater hydroid *Cordylophora caspia*. Accordingly, based on these chronic toxicity values and an assessment factor of 10, a predicted no effect concentration (PNEC) of 4.1 μgV/L was obtained.

The value of 4.1 μgV/L obtained from the chronic toxicity to the fish species was used as the PNEC for this substance.

The PEC/PNEC ratio was 0.2 for freshwater bodies and 0.5 for seawater.

A concentration in water of 18 μg/L was reported in a past survey of limited public water bodies (freshwater). In addition, the maximum river concentration is estimated to be 4.8 μg/L from reported releases under the PRTR

Law, which suggests that locations with higher concentrations than the PEC may exist. Accordingly, efforts to collect data on this substance are needed. Regarding this substance, efforts are needed to understand the production and import quantities as well as trends in PRTR data, and prevalent concentrations in public water bodies need to be understood in detail while taking locations with naturally derived high concentrations into consideration.

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)			
Fish (flagfish)	Chronic	NOEC growth inhibition	10	4.1	Freshwater	1.0	0.2	▲	▲
					Seawater	2.0	0.5		

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5. Conclusions

	Conclusions		Judgment
Health risk	Oral exposure	Collection of further information would be required.	(▲)
	Inhalation exposure	Requiring information collection.	▲
Ecological risk	Requiring information collection.		▲

［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.