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| 9 | CAS No.: 7440-48-4 (Cobalt) | Substance: Cobalt and its compounds |

Chemical Substances Control Law Reference No.:

PRTR Law Cabinet Order No.: 1-132 (Cobalt and its compounds)

Element Symbol: Co

Atomic Weight: 58.93

**1. General information**

Cobalt (II) oxide, tricobalt (II, III) tetroxide, and cobalt (III) lithium dioxide are insoluble in water. The aqueous solubility of cobalt (II) chloride is $5.62\times10^5$ mg/1,000 g (25°C); the aqueous solubility of cobalt (II) nitrate is $1.03\times10^6$ mg/1,000 g (25°C) (anhydride, hexahydrate); the aqueous solubility of cobalt (II) sulfate is $3.83\times10^5$ mg/1,000 g (25°C) (anhydride, 7-hydrate); and the aqueous solubility of cobalt (II) bromide is $1.132\times10^6$ mg/1,000 g (20°C). Bioaccumulation of cobalt sulfate is thought to be low.

Cobalt and its 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). The main use of cobalt is in lithium ion rechargeable batteries used in cellular telephones and laptop computers. The main uses of cobalt compounds are permanent magnets and video tape magnetic materials for cobalt carbonate; pigments for ceramics and glass as well as catalysts for cobalt oxide; dyestuffs, ceramic colorants, plating, and raw materials for ink drying agents for cobalt chloride (anhydride); catalysts, magnetic powders (magnetic tape raw materials), and surface treatment reagents for storage batteries, plating, or other surface for cobalt sulfate; and cobalt salt raw materials, paint or ink drying agents, and ceramic pigments for cobalt chloride. The production and import quantities in fiscal 2010 were 4,000 t for cobalt oxide, less than 1,000 t for cobalt nitrate, 3,000 t for cobalt hydroxide, and 1,000 t for cobalt sulfate.

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**2. Exposure assessment**

Total release of cobalt and its compounds to the environment in fiscal 2010 under the PRTR Law was 28 t, of which approximately 6.4 t or 22% of overall releases were reported. The major destination of reported releases was public freshwater bodies. In addition, 220 t was transferred to waste materials and 2.5 t was transferred to sewage. Industry types with large reported releases were the chemical industry, electrical machinery manufacturing industry, and steelmaking industry for the atmosphere; and the chemical industry, metal products manufacturing industry, and electrical machinery manufacturing industry for 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 cobalt in the environment vary. Accordingly, the proportions distributed to individual media for cobalt were not predicted.

The maximum expected concentration of exposure to humans via inhalation, based on general environmental atmospheric data, was around 0.00074 μg/m$^3$. 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 0.029 μg/m$^3$.

The maximum expected oral exposure was estimated to be around 0.36 μg/kg/day on the basis of calculations from data for public freshwater bodies. However, 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 70 μg/L. Using this estimated concentration for rivers to calculate oral exposure gave 2.8 μg/kg/day.

The predicted environmental concentration (PEC), which indicates exposure to aquatic organisms, was around 9.1 μg/L for public freshwater bodies and around 0.45 μg/L for seawater. The maximum river concentration was estimated to be 70 μg/L from reported releases to public freshwater bodies under the PRTR Law.

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**3.Initial assessment of health risk**

Cobalt metal dust and fume are slightly irritating to respiratory tract. Inhalation exposure to this substance may cause coughing, shortness of breath, sore throat and wheezing, while its oral exposure may cause abdominal pain and vomiting. Its contact with eyes may cause redness. Cobalt acetate, cobalt nitrate, cobalt sulfate and cobalt carbonyl may cause irritation to eyes, skin and respiratory tract, and pulmonary edema may occur by inhalation of these chemicals. Cobalt chloride and cobalt naphthenate may cause irritation to eyes and respiratory tract. Cobalt oxide and cobalt sulphide may cause mechanical irritation.

As for carcinogenic potential of the substance, an initial assessment was conducted only on the basis of its non-carcinogenic effects, since its carcinogenicity to human could not be confirmed although its carcinogenic effects on animals had been reported.

A LOAEL of 2.1 mg/kg/day (for increased red blood cells) obtained from the health effects of cobalt chloride on human (1 mg/kg/day as cobalt) was divided by a factor of 10 for the use of a NOAEL. 0.1 mg/kg/day was identified to be the reliable lowest dose of the substance as its 'non-toxic level*'. As the increase of red blood cells was attributed to the transient increase of erythropoietin production as a result of exposure to cobalt, short durations of the tests were not taken into account. With regard to inhalation exposure to the substance, a NOAEL of 0.0053 mg/m$^3$ (for reduced pulmonary function) obtained from its effects on workers exposed to it was adjusted for their durations to provide 0.001 mg/m$^3$ for its intermittent to continuous exposure. This value was identified to be the reliable lowest dose of the substance as its 'non-toxic level*'.

With regard to its oral exposure, its maximum exposure was predicted to be around 0.36 μg/kg/day, when its intakes through freshwater from public water bodies were assumed. The MOE would be 56 when calculated from its 'non-toxic level*' of 0.1 mg/kg/day and the predicted maximum exposure, and divided by a factor of 5 to extrapolate animal data to human carcinogenic hazards. Its maximum exposure was also calculated to be 2.8 μg/kg/day from its concentrations in river water with effluents from operators discharging high concentrations of the substance, reported in FY 2010 under the PRTR Law. The MOE would be 7 when calculated from this value. In addition, the MOE would be 22 when calculated from its oral exposure of approximately 0.91 μg/kg/day, estimated on the basis of intakes of metallic elements of 0.42 μg/kg/day through food, combined with its exposure of 0.13 μg/kg/day through soil ingestion calculated with its predicted maximum exposure of approximately 0.36 μg/kg/day for its intakes through freshwater from public water bodies. Therefore, collection of further information would be required to assess health risk from its oral exposure.

With regard to inhalation exposure to the substance, its maximum exposure concentration in the ambient air was estimated to be approximately 0.00074 μg/m$^3$. The MOE would be 270 when calculated from its 'non-toxic level*' of 0.001 mg/m$^3$ and the predicted maximum exposure concentration, and divided by a factor of 5 to extrapolate animal data to human carcinogenic hazards. Its maximum (annual mean) concentration in the ambient air near operators with its emissions in high concentrations was calculated to be 0.029 μg/m$^3$ from its emissions reported in FY 2010 under the PRTR Law. The MOE would be 7 when calculated from this value as its reference. Therefore, collection of further information would be required to assess health risk from its inhalation 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*' 0.1 mg/kg/day | Human | Increased red blood cells | Drinking water | — μg/kg/day | MOE | — | × | ▲ |
| | | | | Freshwater | 0.36 μg/kg/day | MOE | 56 | ▲ | |
| Inhalation | 'Non-toxic level*' 0.001 mg/m$^3$ | Human | Reduced lung function | Ambient air | 0.00074 μg/m$^3$ | MOE | 270 | ○ | (▲) |
| | | | | 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 96-h $EC_{50}$ of 520 μgCo/L for growth inhibition in the green alga *Chlorella pyrenoidosa*, a 48-h $LC_{50}$ of 1,110 μgCo/L for in the crustacean *Daphnia magna*, a 96-h $LC_{50}$ of 1,406 μgCo/L for the fish species *Oncorhynchus mykiss* (rainbow trout), and a 96-h $EC_{50}$ of 136 μgCo/L for growth inhibition in the duckweed *Spirodela polyrhiza*. Accordingly, based on these acute toxicity values and an assessment factor of 100, a predicted no effect concentration (PNEC) of 5.2 μgCo/L was obtained.

With regard to chronic toxicity, the following reliable data were obtained: a 96-h NOEC of 38 μgCo/L for growth inhibition in the green alga *C. pyrenoidosa*, and a 28-d NOEC of 26 μgCo/L for growth inhibition in the great pond snail *Lymnaea stagnalis*. Accordingly, based on these chronic toxicity values and an assessment factor of 100, a PNEC of 0.38 μgCo/L was obtained.

The value of 0.38 μgCo/L obtained from the chronic toxicity to the alga was used as the PNEC for this substance.

The PEC/PNEC ratio was 24 for freshwater bodies and 1.2 for seawater. For this reason, the substances are considered as candidates for further work.

| 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) | | | |
| Green algae | Chronic | NOEC growth inhibition | 100 | 0.38 | Freshwater | 9.1 | 24 | ■ | ■ |
| | | | | | Seawater | 0.45 | 1.2 | | |

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**5. Conclusions**

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

[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.