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Air Toxicological Summary for: Cobalt Metal and Compounds

C A S :

- 7440-48-4 (Cobalt (Co) metal, dust)
- 71-48-7 (Co (II) acetate)
- 917-69-1 (Co (III) acetate)
- 7789-43-7 (Co (II) bromide)
- 513-79-1 (Co (II) carbonate)
- 10210-68-1 (Co carbonyl)
- 7646-79-9 (Co (II) chloride)
- 13586-82-8 (Co (II) ethylhexanoate)
- 136-52-7 (Co (II) ethylhexanoate (known stoichiometry))
- 21041-93-0 (Co (II) hydroxide)
- 16842-03-8 (Co hydrocarbonyl)
- 61789-51-3 (Co naphthenate)
- 10141-05-6 (Co (II) nitrate)
- 10026-22-9 (Co (II) nitrate hexahydrate)
- 814-89-1 (Co (II) oxalate)
- 1307-96-6 (Co (II) oxide)
- 1308-04-9 (Co (III) oxide)
- 1308-06-1 (Co (II,III) oxide)
- 1560-69-6 (Co propionate)
- 1002-88-6 (Co stearate)
- 10124-43-3 (Co (II) sulfate)
- 10026-24-1 (Co (II) sulfate (heptahydrate))
- 1317-42-6 (Co sulfide)

Air exposure durations

- **Acute_{1hr}** = 1 hour
- **Acute_{24hr}** = 24 hours
- **Intermediate** = greater than 24 hours to 1 year
- **Chronic** = greater than 1 year to a lifetime
- **Cancer** = 0 to 70 years (lifetime)

NOTE: The Health Based Values (HBVs) developed in this summary apply to the $\mu\text{g}/\text{m}^3$ air concentration of elemental cobalt (Co), or the $\mu\text{g}/\text{m}^3$ air concentration of the compound, reported as Co. This can be determined as follows:

$$\begin{aligned} & \text{Compound air concentration, as elemental cobalt} \\ &= \text{compound air concentration (mg/m}^3\text{)} \times \left(\frac{\text{molecular weight of Co}}{\text{molecular weight of compound}} \right) \end{aligned}$$

Acute_{1hr} Non-Cancer Health Based Value (HBV_{Acute1hr}) = 1 $\mu\text{g}/\text{m}^3$

$$\begin{aligned} &= \frac{(\text{Point of Departure (POD}_{\text{HEC}}), \text{ mg/m}^3\text{)}}{(\text{Uncertainty Factors (UF)})} \\ &= \frac{(0.138 \text{ mg/m}^3)}{(100)} \end{aligned}$$

$$= 0.0014 \text{ mg/m}^3 \text{ (or } 1.4 \text{ } \mu\text{g/m}^3\text{, rounded to } 1 \text{ } \mu\text{g/m}^3\text{)}$$

Reference Concentration:	POD _{HEC} /Total UF = 0.14 mg/m ³ (animal study)
Source of toxicity value:	Determined by MDH 2026 based on Viegas et al. (2022a, 2022b, and 2024)
POD and Critical Effect:	NOAEL = 0.2 mg/m ³ ; increased bronchoalveolar lavage fluid (BALF) neutrophils and decreased BALF cell viability in rats
Human Equivalent Concentration (HEC):	NOAEL _{HEC} = 0.317 (NOAEL _{adj1hr}) x 0.4338 (DAF) = 0.138 mg Co/m³ . The study NOAEL of 0.2 mg/m ³ was adjusted from a 4-hour to a 1-hour exposure using Haber's rule as modified by tenBerge (1986). The DAF is the regional deposited dose ratio (RDDR) as described in EPA (1994) using Multiple-Path Particle Dosimetry Model (MPPD v 3.03).
Total uncertainty factor (UF):	100
Uncertainty factor allocation:	An uncertainty factor of 3 was used for interspecies extrapolation to account for extrapolation from animals to humans with dosimetric adjustments. An uncertainty factor of 10 was applied for human response variability. An additional database uncertainty factor of 3 was applied due to the lack of two generation reproductive/developmental studies.

Acute_{24hr} Non-Cancer Health Based Value (HBV_{Acute24hr}) = 0.1 $\mu\text{g}/\text{m}^3$

$$\begin{aligned} &= \frac{(\text{POD}_{\text{HEC}}), \text{ mg/m}^3}{(\text{UF})} \\ &= \frac{(0.0144 \text{ mg/m}^3)}{(100)} \end{aligned}$$

$$= 0.00014 \text{ mg/m}^3 \text{ (or } 0.14 \text{ } \mu\text{g/m}^3\text{, rounded to } 0.1 \text{ } \mu\text{g/m}^3\text{)}$$

Reference Concentration: POD_{HEC}/Total UF = 0.00014 mg/m³ (animal study)

Source of toxicity value:	Determined by MDH 2026 based on Viegas et al. (2022a, 2022b, and 2024)
POD and Critical Effect:	NOAEL = 0.2 mg/m ³ ; increased BALF neutrophils and decreased BALF cell viability in rats
Human Equivalent Concentration:	NOAEL _{HEC} = 0.0333 (NOAEL _{adj24hr}) x 0.4338 (DAF) = 0.0144 mg Co/m³ . The study NOAEL was adjusted from a 4-hour to a 24-hour exposure using Haber's rule as modified by tenBerge (1986). The DAF is the regional deposited dose ratio (RDDR) as described in EPA (1994) using Multiple-Path Particle Dosimetry Model (MPPD v 3.03).
Total uncertainty factor (UF):	100
Uncertainty factor allocation:	An uncertainty factor of 3 was used for interspecies extrapolation to account for extrapolation from animals to humans with dosimetric adjustments. An uncertainty factor of 10 was applied for human response variability. An additional database uncertainty factor of 3 was applied due to the lack of two generation reproductive/developmental studies.

Intermediate Non-Cancer Health Based Value (HBV_{inter}) = 0.02 µg/m³

$$= \frac{(\text{POD}_{\text{HEC}}), \text{ mg/m}^3}{(\text{UF})}$$

$$= \frac{(0.00465 \text{ mg/m}^3)}{(300)}$$

$$= 0.0000155 \text{ mg/m}^3 \text{ (or } 0.0155 \text{ µg/m}^3 \text{, rounded to } 0.02 \text{ µg/m}^3 \text{)}$$

Reference Concentration:	POD _{HEC} /Total UF = 0.0000155 mg/m ³ (animal study)
Source of toxicity value:	Determined by MDH 2026 based on Bucher et al. 1990; NTP 1991, 2023
POD and Critical Effect:	LOAEL _{adj} = 0.114 x 6h/24h x 5d/7d = 0.0204 mg/m ³ ; squamous metaplasia of the larynx in female rats.
Human Equivalent Concentration:	LOAEL _{HEC} = 0.0204 (LOAEL _{adj})x 0.2277 (DAF) = 0.00465 mg Co/m³ . The DAF is the regional deposited dose ratio (RDDR) as described in EPA (1994) using Multiple-Path Particle Dosimetry Model (MPPD v 3.03).
Total uncertainty factor:	300
Uncertainty factor allocation:	A factor of 3 was applied to account for animal to human extrapolation when DAF is applied, and an uncertainty factor of 10 was applied to account for human response variability among susceptible populations. An uncertainty factor of 3 was applied for use of a LOAEL for a less serious health endpoint when a NOAEL cannot be established. An additional database uncertainty factor of 3 was applied due

to the lack of two generation reproductive/developmental studies.

Chronic Non-Cancer Health Based Value (HBV_{chronic}) = $0.02 \mu\text{g}/\text{m}^3$

$$\begin{aligned} &= \frac{(POD_{\text{adj}}), \text{ mg}/\text{m}^3}{(\text{UF})} \\ &= \frac{(0.00189 \text{ mg}/\text{m}^3)}{(100)} \\ &= 0.0000189 \text{ mg}/\text{m}^3 \text{ (or } 0.0189 \mu\text{g}/\text{m}^3, \text{ rounded to } 0.02 \mu\text{g}/\text{m}^3) \end{aligned}$$

Reference Concentration:	$POD_{\text{adj}} / \text{Total UF} = 0.0000189 \text{ mg}/\text{m}^3$ (human study)
Source of toxicity value:	Determined by MDH 2026 based on Nemery et al. 1992.
POD and Critical Effect:	NOAEL = $0.0053 \text{ mg}/\text{m}^3$; Respiratory, decreased forced expiratory volume (FEV1) and forced vital capacity (FVC); increased cough, wheezing, and upper airway irritation in workers
Adjusted POD Concentration:	$POD_{\text{ADJ}} = POD (\text{mg}/\text{m}^3) * \frac{VE_{\text{ho}}}{VE_h} * \frac{5 \text{ days}}{7 \text{ days}} = 0.00189 \text{ mg}/\text{m}^3$ VE_{ho} = human occupational ventilation rate of $10 \text{ m}^3/\text{day}$; VE_h = human ventilation rate of $20 \text{ m}^3/\text{day}$.
Total uncertainty factor:	100
Uncertainty factor allocation:	An uncertainty factor of 3 was applied to account for extrapolating from an assumed subchronic exposure duration to a chronic exposure duration. An uncertainty factor of 10 was applied to account for human response variability among susceptible populations. An uncertainty factor of 3 was applied to account for the lack of reproductive and developmental studies that might inform additional infant/child susceptibility and the fact that occupational studies assess only healthy adult worker populations.

Cancer Health Based Value / Risk Assessment Advice = $0.001 \mu\text{g}/\text{m}^3$

Cancer classification: Cobalt metal (without tungsten carbide or other metal alloys) and soluble cobalt (II) salts (cobalt chloride, cobalt sulfate) are classified as probably carcinogenic to humans and cobalt (II) oxide as possibly carcinogenic to humans (IARC 2023). Metal mixtures containing cobalt, including cobalt metal with tungsten carbide and weapons-grade tungsten (with nickel and cobalt) are classified as probably and possibly carcinogenic to humans, respectively (IARC 2006, 2023). IARC (2023) determined that cobalt (II, III) oxide (cobalt tetraoxide), cobalt (II) sulfide, and other

cobalt (II) compounds cannot be classified as to their carcinogenicity to humans. NTP determined that cobalt and cobalt compounds that release cobalt ions in vivo are reasonably anticipated to be human carcinogens (NTP 2021). The EPA Integrated Risk Information System (IRIS) program was recently conducting an inhalation cancer risk assessment for cobalt and cobalt compounds and released the protocol for the assessment along with its dose response methodology (EPA 2022a). However, the IRIS Program was terminated prior to finalizing this assessment.

Inhalation Unit Risk (IUR): 4.038 (mg/m³)⁻¹; Note: per MDH policy (MDH 2020), age-dependent adjustment factors (ADAF) are applied to the IUR to protect against early-life sensitivity (see calculations below for the ADAF adjusted IUR, or IUR_{adj}).

Source of IUR: NTP 1998; 2014

Tumor site(s): bronchioalveolar adenomas and carcinomas

Cancer ADAF and HBV Calculations:

$$\text{IUR}_{\text{adj}} = \text{IUR } 4.038 \text{ (mg/m}^3\text{)}^{-1} \times [(2 \text{ yrs} \times 10) + (14 \text{ yrs} \times 3) + (54 \text{ yrs} \times 1)] / 70 \text{ yrs}$$

$$\text{IUR}_{\text{adj}} = 6.69 \text{ (mg/m}^3\text{)}^{-1}$$

$$\text{Cancer HBV} = [1 \text{ in } 100,000 \text{ additional lifetime cancer risk} / \text{IUR}_{\text{adj}}]$$

$$\text{Cancer HBV} = [0.00001 / 6.69 \text{ (mg/m}^3\text{)}^{-1}] = 1.495 \times 10^{-6} \text{ mg/m}^3, \text{ or } 0.001 \text{ }\mu\text{g/m}^3$$

Volatile: No

Summary of guidance value history:

MDH had no previously published air guidance values for cobalt and cobalt compounds.

Summary of toxicity testing for health effects identified in the Health Standards Statute (144.0751):

Even if testing for a specific health effect was not conducted for this chemical, information about that effect might be available from studies conducted for other purposes. MDH has considered the following information in developing health protective guidance.

	Endocrine	Immunotoxicity	Development	Reproductive	Neurotoxicity	Respiratory
Tested for specific effect?	Yes	Yes	Yes (oral)	Yes (oral)	Yes	Yes
Effects observed?	Yes ¹	Yes ²	No ³	Yes ⁴	Yes ⁵	Yes ⁶

Comments on extent of testing or effects:

¹From ATSDR, 2024 - Limited data from occupational studies do not provide consistent evidence of alterations in serum thyroid hormone levels following inhalation exposure to cobalt or cobalt compounds. A study of Danish porcelain workers found alterations in serum thyroid hormone levels in workers utilizing semi-soluble cobalt dye (cobalt-zinc silicate), but not in workers utilizing insoluble cobalt dye (cobalt aluminate), despite measured air cobalt levels of 0.05 mg Co/m³. Another study reported a significant reduction in serum triiodothyronine (T₃) in a cohort of 82 workers from Belgium occupationally exposed to cobalt oxides, cobalt salts, and cobalt metal at a mean air concentration of 0.125 mg/m³, compared to unexposed referents. These thyroid related effects were observed with air concentrations about 3 to 8 times higher than the lowest cobalt concentrations found to be associated with respiratory health effects. There is limited evidence for non-cancer effects in the adrenal gland following chronic-duration inhalation exposure to cobalt. An increased incidence of medullary hyperplasia in the adrenal gland was seen in female F344/N rats after exposure to cobalt metal at 1.25 mg Co/m³ for 105 weeks; this was not observed in similarly exposed male F344/N rats or B6C3F1 mice at concentrations up to 5 mg Co/m³ (Behl et al. 2015; NTP 2014).

²From ATSDR, 2024 - Occupational exposure to cobalt (**see Respiratory Section below for further details**) has been associated with increased risk of asthma in some cohorts (Kusaka et al. 1986b; Linna et al. 2003; Roto 1980; Walters et al. 2012). Shirakawa et al. (1988, 1989) showed an increase in immunoglobulin E (IgE) antibodies specific to cobalt in several cases of occupational asthma associated with hard metal exposure. Similarly, a case study in a diamond polisher diagnosed with occupational asthma suggests that cobalt-mediated asthma may be IgE-mediated, and that sensitization of lymphocytes could play a crucial role in disease development (Krakowiak et al. 2005). Conversely, studies of workers in cobalt production or hard metal industries showed no positive associations between estimates of current or cumulative exposure to cobalt and markers of inflammation. Cobalt is a sensitizer. A meta-analysis of contact allergy in the general population found that cobalt was the third most prevalent hapten (2.7%) in individuals with positive patch tests, behind nickel (11.4%) and fragrance mix (3.5%); however, in most cases, the source of cobalt sensitization exposure was unknown (Alinaghi et al. 2019). **Although the respiratory effects the non-cancer HBVs are based on for this assessment are considered protective for the general public, including sensitive sub-populations, MDH cannot ensure they will provide protection for chemically hypersensitive individuals.**

³No studies were identified that examined developmental toxicity in humans or animals following inhalation exposure to cobalt. From TCEQ, 2017 - Oral exposure of female rats to cobalt chloride at 5.4 or 21.8 mg Co/kg-day from gestation day 14 through lactation day 21 has been shown to result in stunted growth and decreased survival, respectively, of newborn pups. The effects on the offspring occurred at levels that also caused maternal toxicity (reduced body weight and food consumption and altered hematological measurements) and might therefore have been an indirect effect of maternal toxicity rather than a direct effect of cobalt on the fetus (Domingo et al. 1985). Szakmary et al. (2001) reported that exposure of pregnant rats to 0-38 mg Co/kg-day as cobalt sulfate did not result in changes in fetal death rates, maternal body weight gain, average litter size, or average fetal or placental weights; however, a dose-related trend was seen for the percent of rat fetuses with retarded body weights that was significant beginning at 19 mg Co/kg-day, although average fetal body weight was not significantly reduced at any dose (e.g., ≈ 2% at 38 mg Co/kg-day). **The maximum possible**

daily internal dose of inhaled cobalt via lung dissolution/transport along with incidental ingestion from the lung's mucociliary escalator system are expected to be much lower than the daily internal dose from the oral doses associated with reported effects from the animal developmental studies described above.

⁴From ATSDR, 2024 - No studies were identified that examined reproductive toxicity in humans following inhalation. Intermediate-duration animal inhalation exposures to cobalt produced reproductive effects in some studies; the male rodent appears to be more susceptible than the female. Testicular atrophy, along with a decrease in number of cells in the seminiferous tubules and atypical germinal epithelial cells in the epididymal ducts, were observed in male rats exposed to ≥ 19.0 mg Co/m³ for 16 days. In the longer intermediate-duration studies (13–14 weeks), effects noted included decreased percent motile sperm at cobalt metal concentrations ≥ 2.5 mg Co/m³ in male rats and mice and at cobalt sulfate concentrations ≥ 1.11 mg Co/m³ in male mice (NTP 1991, 2014). Additional effects observed in exposed male rodents included increased relative testes weights in male rats at ≥ 2.5 mg Co/m³ as cobalt metal, decreased absolute testes weight in male mice at ≥ 5 mg Co/m³ as cobalt metal, and a 3-fold increase in percent abnormal sperm with testicular atrophy in male mice at 11.4 mg Co/m³ as cobalt sulfate. **In general, animal reproductive effects were observed at air concentrations that are similar to and up to 10 times the concentration associated with animal respiratory effects.**

⁵From ATSDR, 2024 - Studies examining neurotoxicity in humans following inhalation exposure to cobalt are limited to a couple of occupational studies evaluating neurotransmitter levels in workers exposed to hard metals. In a year-long prospective cohort from Taiwan of 69 shipyard workers exposed to particulate matter and metal fumes, urinary cobalt levels were not associated with urinary serotonin levels (Lai et al. 2021). In a cross-sectional study of 186 Chinese welders, urinary cobalt levels were not associated with serum dopamine levels (Wu et al. 2023b). Available intermediate-duration inhalation studies in rodents indicate that exposure to high levels of cobalt (10 to 20 mg Co/m³, which is **approximately 1000x concentrations associated with respiratory effects in humans**) caused central nervous system depression, while histopathological damage was only observed at concentrations associated with lethality.

⁶From ATSDR, 2024 - Respiratory effects are a known health effect for humans following inhalation exposure to cobalt based on a high level of evidence in humans and animals. In humans, occupational exposure ranging from 0.015 to 0.14 mg Co/m³ has been associated with increased respiratory symptoms, impaired lung function, and asthma in multiple studies. In animals, inhalation exposure greater than 2.2 mg Co/m³ is consistently associated with inflammatory changes throughout the upper and lower respiratory tract progressing to widespread lesions with increasing concentration and duration in multiple species. At high exposure levels, severe pulmonary damage was the cause of death in rodents. **All of the non-cancer air guidance values in this review are based upon these respiratory effects as the most sensitive health endpoint. As a result, these cobalt HBVs are expected to be protective of all of the other effects reported in this table with the exception of hypersensitized individuals (see Immunologic section above for more details).**

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