

# Cobalt Compounds

## Hazard Summary

Cobalt is a natural element found throughout the environment. Acute (short-term) exposure to high levels of cobalt by inhalation in humans and animals results in respiratory effects, such as a significant decrease in ventilatory function, congestion, edema, and hemorrhage of the lung. Respiratory effects are also the major effects noted from chronic (long-term) exposure to cobalt by inhalation, with respiratory irritation, wheezing, asthma, pneumonia, and fibrosis noted. Cardiac effects, congestion of the liver, kidneys, and conjunctiva, and immunological effects have also been noted in chronically-exposed humans. Cobalt is an essential element in humans, as a constituent of vitamin B<sub>12</sub>. Human studies are inconclusive regarding inhalation exposure to cobalt and cancer, and the one available<sup>12</sup> oral study did not report a correlation between cobalt in the drinking water and cancer deaths. EPA has not classified cobalt for carcinogenicity.

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Please Note: The main sources of information for this fact sheet are the Agency for Toxic Substances and Disease Registry's (ATSDR's) Toxicological Profile for Cobalt (1) and California Environmental Protection Agency's Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels. (5)

## Uses

- Cobalt is used to make superalloys (alloys that maintain their strength at high temperatures approaching their melting points) and in pigment manufacture. (1,5)

## Sources and Potential Exposure

- Cobalt is a natural element found throughout the environment; the general population may be exposed to cobalt in the air, drinking water, and food. (1,5)
- The average concentration of cobalt in ambient air in the United States is approximately 0.0004 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ). However, higher levels have been detected; in one industrial area, levels of 0.61  $\mu\text{g}/\text{m}^3$  were measured. (1)
- A study found average cobalt levels in drinking water of 2 micrograms per liter ( $\mu\text{g}/\text{L}$ ), but values up to 107  $\mu\text{g}/\text{L}$  have been reported. (1)
- The average daily intake of cobalt from food is estimated to be 5 to 40  $\mu\text{g}/\text{d}$ . (1)
- Occupational exposure to cobalt may occur, particularly in workers in the hard metal industry. (1)

## Assessing Personal Exposure

- Cobalt can be measured in the urine and the blood, for periods up to a few days after the exposure. (1)

## Health Hazard Information

### Acute Effects:

- Acute exposure to high levels of cobalt by inhalation in humans and animals results in respiratory effects, such as a significant decrease in ventilatory function, congestion, edema, and hemorrhage of the lung. (1)
- Acute animal tests in rats have shown cobalt to have **extreme** toxicity from inhalation exposure, and **moderate** to **high** toxicity from oral exposure. (1,2)

#### Chronic Effects (Noncancer):

- Cobalt is an essential element in humans and animals as a constituent of vitamin B<sub>12</sub>. Cobalt has also been used as a treatment for anemia, because it stimulates red blood cell production. (1)<sup>2</sup>
- Chronic exposure to cobalt by inhalation in humans results in effects on the respiratory system, such as respiratory irritation, wheezing, asthma, decreased lung function, pneumonia, and fibrosis. (1,5)
- Other effects noted in humans from inhalation exposure to cobalt include cardiac effects, such as functional effects on the ventricles and enlargement of the heart, congestion of the liver, kidneys, and conjunctiva, and immunological effects that include cobalt sensitization, which can precipitate an asthmatic attack in sensitized individuals. (1,3)
- Cardiovascular effects (cardiomyopathy) were observed in people who consumed large amounts of beer over several years time containing cobalt sulfate as a foam stabilizer. The effects were characterized by cardiogenic shock, sinus tachycardia, left ventricular failure, and enlarged hearts. The beer drinkers ingested cobalt at an average concentration of 0.04 milligrams per kilogram per day (mg/kg/d) to 0.14 mg/kg/d. (1,3)
- Gastrointestinal effects (nausea, vomiting, and diarrhea), effects on the blood, liver injury, and allergic dermatitis have also been reported in humans from oral exposure to cobalt. (1)
- Animal studies have reported respiratory, cardiovascular, and central nervous system (CNS) effects, decreased body weight, necrosis of the thymus, and effects on the blood, liver, and kidneys from inhalation exposure to cobalt. (1,3)
- EPA has not established a Reference Concentration (RfC) or a Reference Dose (RfD) for cobalt.
- The California Environmental Protection Agency (CalEPA) has established a chronic reference exposure level of 0.000005 milligrams per cubic meter (mg/m<sup>3</sup>) for cobalt based on respiratory effects in rats and mice. The CalEPA reference exposure level is a concentration at or below which adverse health effects are not likely to occur. It is not a direct estimator of risk, but rather a reference point to gauge the potential effects. At lifetime exposures increasingly greater than the reference exposure level, the potential for adverse health effects increases. (5)
- ATSDR has established an intermediate inhalation minimal risk level (MRL) of 0.00003 mg/m<sup>3</sup> based on respiratory effects in rats. The MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. (1)

#### Reproductive/Developmental Effects:

- No information is available on the reproductive or developmental effects of cobalt in humans via inhalation exposure. In one oral study, no developmental effects on human fetuses were observed following treatment of pregnant women with cobalt chloride. (1)
- Animal studies, via inhalation exposure, have reported testicular atrophy, a decrease in sperm motility, and a significant increase in the length of the estrus cycle, while oral studies have reported stunted growth and decreased survival of newborn pups. These effects on the offspring occurred at levels that also caused maternal toxicity. (1,5)

#### Cancer Risk:

- Limited data are available on the carcinogenic effects of cobalt. In one study on workers that refined and processed cobalt and sodium, an increase in deaths due to lung cancer was found for workers exposed only to cobalt. However, when this study was controlled for date of birth, age at death, and smoking habits, the difference in deaths due to lung cancer was found to not be statistically significant. In another study assessing the correlation between cancer deaths and trace metals in water supplies in the United States, no correlation was found between cancer mortality and the level of cobalt in the water. (1)
- In a study by the National Toxicology Program (NTP), cobalt sulfate heptahydrate exposure via inhalation resulted in increased incidences of alveolar/bronchiolar tumors in rats and mice. (9)
- In an animal study, inhalation of cobalt over a lifetime did not increase the incidence of tumors in hamsters. (1,4)

- Cobalt, via direct injection under the muscles or skin, has been reported to cause tumors at the injection site in animals. (1,4)
- EPA has not classified cobalt for carcinogenicity.

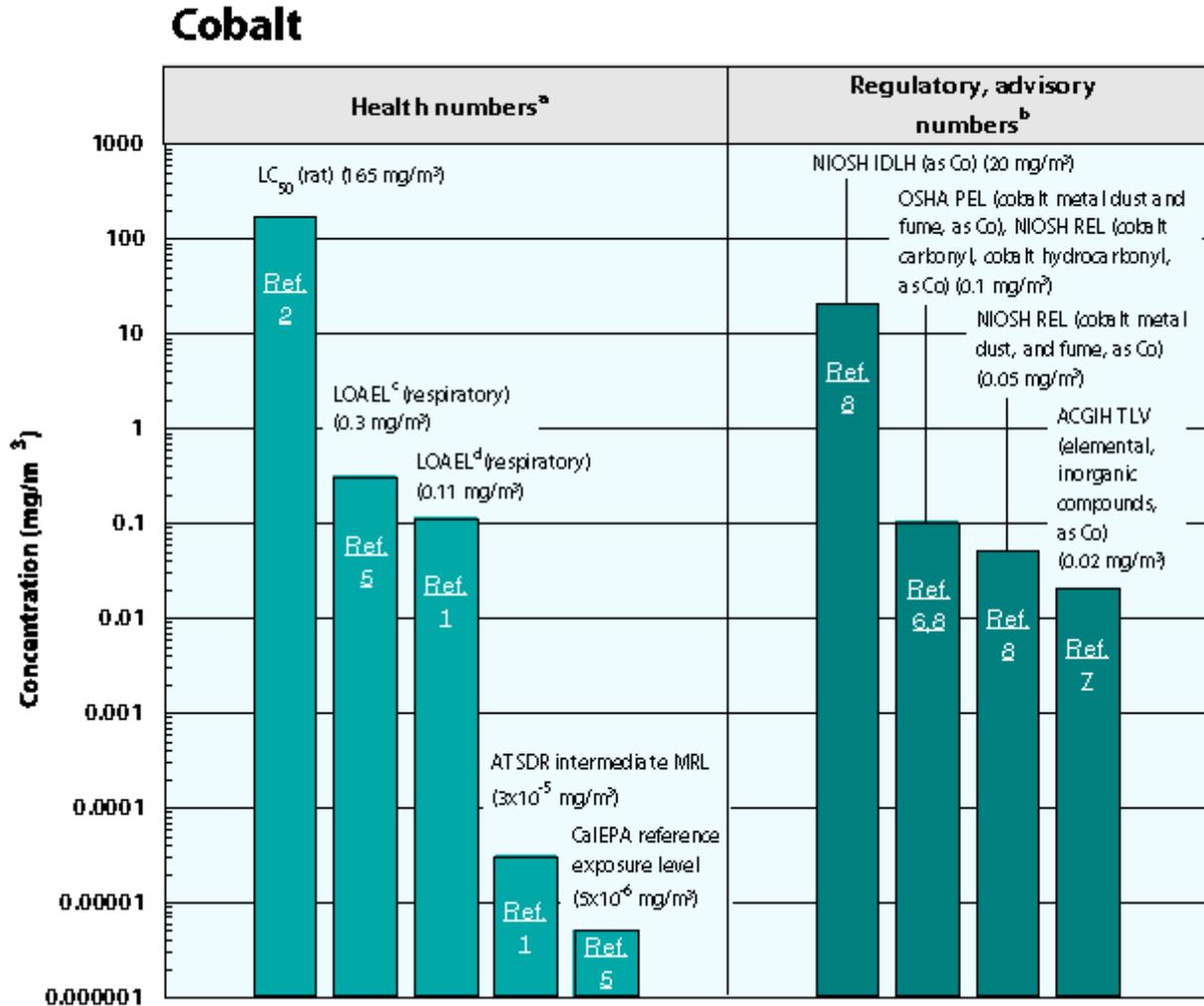
## Physical Properties

- Cobalt usually occurs in the environment in association with other metals such as copper, nickel, manganese, and arsenic. (1)
- Pure cobalt is a steel-gray, shiny, hard metal that is insoluble in water. (1)
- The chemical symbol for cobalt is Co, and the atomic weight is 58.93 g/mol. (1,5)

Conversion Factors:

To convert concentrations in air (at 25°C) from ppm to  $\text{mg}/\text{m}^3$ :  $\text{mg}/\text{m}^3 = (\text{ppm}) \times (\text{molecular weight of the compound}) / (24.45)$ . For cobalt:  $1 \text{ ppm} = 2.4 \text{ mg}/\text{m}^3$ .

### Health Data from Inhalation Exposure



ACGIH TLV--American Conference of Governmental and Industrial Hygienists' threshold limit value expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effects.

LC<sub>50</sub> (Lethal Concentration<sub>50</sub>)--A calculated concentration of a chemical in air to which exposure for a specific

length of time is expected to cause death in 50% of a defined experimental animal population.

LOAEL--Lowest-observed-adverse-effect level.

NIOSH IDLH --National Institute of Occupational Safety and Health's immediately dangerous to life or health limit; NIOSH recommended exposure limit to ensure that a worker can escape from an exposure condition that is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from the environment.

NIOSH REL --NIOSH's recommended exposure limit; NIOSH-recommended exposure limit for an 8- or 10-h time-weighted-average exposure and/or ceiling.

OSHA PEL --Occupational Safety and Health Administration's permissible exposure limit expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effect averaged over a normal 8-h workday or a 40-h workweek.

The health and regulatory values cited in this factsheet were obtained in December 1999.

<sup>a</sup> Health numbers are toxicological numbers from animal testing or risk assessment values developed by EPA.

<sup>b</sup> Regulatory numbers are values that have been incorporated in Government regulations, while advisory numbers are nonregulatory values provided by the Government or other groups as advice. OSHA numbers are regulatory, whereas NIOSH and ACGIH numbers are advisory.

<sup>c</sup> The LOAEL is from the critical study used as the basis for the CalEPA reference exposure level.

<sup>d</sup> The LOAEL is from the critical study used as the for the ATSDR intermediate MRL.

Summary created in April 1992, updated in January 2000

## References

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