9 Cobalt in the Environment

The pages that follow give an overview of the role of cobalt in health and the environment.

For further information, please refer to the HS&E section on this website.

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Cobalt Exposure and Heart Disease
(April 2006)

Cobalt in the form of Vitamin B_{12} (hydroxocyanocobalamin) is essential for humans. Vitamin B_{12} supports important synthetic reactions in metabolic processes and is essential for the production of red blood cells. The metabolism of Vitamin B_{12} and the daily ingestion of cobalt–containing foodstuffs provide the most significant source of cobalt (e.g. background level) in the human body. Background levels of cobalt are not known to be associated with adverse health effects in humans.

Humans may ingest up to several milligrams of cobalt per day in their diet and based on case reports, appear to tolerate even higher daily doses of cobalt during clinical treatment for anaemia without adverse effects to the heart. However, the ingestion of relatively high levels of cobalt (when compared to dietary intake) from inorganic cobalt salts with large amounts of alcohol has been reported to pose health risks to some humans. In the mid 1960's, small amounts of cobalt (1-2 parts per million) in the form of cobalt chloride were added to a brand of beer as a foam stabiliser. A number of fatalities related to cardiomyopathy were reported in men who consumed large amounts (more than 8 pints per day) of the cobalt-laden beer. Daily oral doses of cobalt in this group were in the range of 0.1 milligram per kilogram body weight.

Cardiomyopathy is a type of heart disease characterised by damage to the muscle and structure of the heart. The resultant effect is muscle cell death and a decrease in the volume of blood pumped. Studies of animal and human exposure to cobalt indicate that oral cobalt exposure, poor diet, and alcohol consumption are jointly associated with heart damage similar to that reported in beer drinkers.

A recent (2004) cross-sectional occupational exposure study of about 200 cobalt refinery workers found no clinically significant heart disease. However, among the most highly exposed workers, there was a relationship between cumulative cobalt exposure and alterations in left ventricular filling and relaxation times. The clinical significance of these changes is currently being investigated.

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Cobalt Exposure and Red Blood Cells
(April 2006)

Cobalt is known to stimulate the production of red blood cells. A cobalt-iron medication was once used to treat specific types of anaemia. In clinical situations where decreased numbers of red blood cells were associated with anaemia, ingestion of inorganic cobalt (cobalt +2 ion in the form of cobalt chloride) stimulated an increase in the production of red blood cells (polycythemia or erythrocytosis). Typical adult doses were administered daily in the range of 50-100 milligrams of cobalt (0.7-2.0 milligrams cobalt per kilogram body weight).

Clinical treatments for sickle cell anaemia in children, utilizing higher cobalt doses (in the range of 3 to 4 milligrams per kilogram body weight), were associated with thyroid effects (decreased iodine uptake, e.g. goiter) in addition to the desired polycythemia. The thyroid and polycythemic effects were reversible upon cessation of oral cobalt treatment. Doses lower than 3 milligrams per kilogram body weight were not reported to induce thyroid effects in children. Currently, the clinical use of cobalt for the treatment of anaemia has been replaced by the use of synthetic erythropoetin, a hormone that induces the production of red blood cells.

Environmental cobalt exposures have been associated with altitude-induced polycythemia and Mountain Sickness* in some residents of a Peruvian mining village located 4300 meters above sea-level. High altitudes may also induce polycythemia in humans due to decreased oxygen levels. One study group of villagers, none of whom currently worked in the mine, with polycythemia (induced by high altitude) and significantly increased levels of cobalt measured in their blood (>1 microgram per litre), had greater frequency of Mountain Sickness when compared to villagers with polycythemia and normal cobalt blood levels.

Two occupational exposure studies have assessed the presence of polycythemia in cobalt-exposed workers. One study was conducted in a cobalt refinery and the other was conducted in a factory where cobalt dyes were used to paint porcelain plates. Both studies reported slight but non-significant decreases in red blood cells of workers. The prevalence and clinical significance of polycythemia in occupational cobalt exposures is not known.

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*Mountain Sickness: Polycythemia (erythrocytosis) usually develops at high altitudes due to lowered ambient oxygen levels. If the polycythemia is severe enough headache, dizziness, weakness, mental confusion, shortness of breath, decreased oxygen saturation, and death may occur. For review see:
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The Effect of Inhalation of Cobalt Substances on the Lungs.
(April 2006)

Occupational inhalation exposures to elemental cobalt powders, salts, oxides and mixed-metal cobalt compounds such as hardmetal (cobalt-tungsten carbide) powders have been associated with asthma in workers. In some instances, antibodies to cobalt were detected in their blood. Workers exposed to cobalt-containing diamond dust in diamond polishing operations (which use cobalt powders as adhesives on the polishing disk) have been reported to undergo pathological changes in their lung tissue that range from intense inflammation to cellular infiltration and fibrosis. Some hardmetal workers have also been reported to contract this condition which has been named "hardmetal lung disease" or "hardmetal pneumoconiosis". Outside of diamond polishing and hardmetal operations, hardmetal disease has not been reported in workers exposed to cobalt substances alone. The prevalence of hardmetal lung disease in hardmetal operations is not known.

With respect to cobalt exposure (in the absence of mixed metals) in humans and lung cancer, there is one epidemiology study and a follow-up study investigating deaths in cobalt salt workers. The initial study reported four deaths due to lung cancer which would have been significantly higher than the expected deaths. However, the study authors later reclassified one lung cancer death after review of death certificates. Based on three deaths, the initial study results were not statistically significant, and the follow-up study did not report any increased deaths due to lung cancer.

There are four epidemiological evaluations of occupational hard metal exposure and lung cancer. While all four studies reported low but significantly increased deaths due to lung cancer, each study has confounding issues. These issues include: the effects of tobacco smoking and other life-style factors, incomplete current exposure data on workers, and lack of information on previous exposure history of the workers.

Animal studies include a lifetime (2-year) inhalation study of cobalt sulphate heptahydrate aerosol in rodents where lifetime inhalation exposure was associated with lung cancer (significant increases in bronchio-alveolar tumours) in rats and mice. Severe inflammation of the entire respiratory tract at all doses was reported as well. Studies with rodents and miniature swine have reported inflammation, lung fibrosis and emphysema after long-term inhalation or intra-tracheal instillation of cobalt, cobalt oxide and cobalt-tungsten carbide powders. Studies investigating the long-term inhalation of hardmetal powder and lung cancer in experimental animals have not been found in the scientific literature.

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The Effects of Cobalt Exposure on the Mammalian Reproductive System
(November 2006)

Exposure to cobalt can occur through inhalation, oral or dermal (skin) routes. Mammals, including humans, are exposed to natural sources of cobalt in their food, water and air. In addition to naturally occurring forms in the environment, cobalt substances may also be present in certain occupational settings and in some consumer products.

Cobalt in the chemically distinct form of Vitamin B_{12} is essential for humans. While humans require Vitamin B_{12}, mammals such as deer, moose and elk, as well as domestic farm ruminants such as cattle and sheep, directly require the bioavailable cobalt (II) ion for reproductive health. It is common veterinary and agricultural practice to provide cobalt salt supplements to ensure a sufficient source of bioavailable cobalt (II) ion for animal health. Agricultural and veterinary experiences with bioavailable cobalt indicate there are "safe" doses which can maintain beneficial levels of cobalt in the animal. Doses of cobalt in diets that are too low (deficient) or too high (over-exposure) have been reported to have harmful effects. Cobalt-deficient diets are associated with a "wasting disease" in farm ruminants, deer, elk and moose where the animals fail to thrive and their reproductive output is significantly decreased. Over-exposures are associated with decreased reproductive output in farm ruminants.

Over-exposure to water-soluble cobalt salts (and thus the cobalt II ion) has been shown to cause damage to testicular tissue in male rats and mice. The effects are reported by oral exposure to cobalt chloride and by inhalation exposure to cobalt sulphate and they appear to be dose-dependent. These studies also reported sperm number and motility were affected. The oral and inhalation studies reported a threshold level (exposure dose) below which no adverse effects were observed for testicular tissue, sperm motility and sperm number. At the highest oral doses studied, fertility in male mice was significantly reduced (measured as the percentage of fertilised ova). A threshold level was also found for the adverse effects on fertility in male mice. It is unclear whether fertility in male mice is restored after the cessation of oral exposure.

Similar reproductive studies with female mice as well as other rodents and animals have not been found in the literature. In addition, no studies have been found with respect to the impact on the human reproductive system of exposure to elemental cobalt or cobalt substances

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Cobalt in Food
(February 2006)

This is a short article on the occurrence of cobalt in food. It aims to present the variety of cobalt concentrations in different food groups.

Why is Cobalt found in food?

Cobalt (Co) is a natural element that is essential for the healthy functioning of many plants and animals and it is often found in the food that we eat.

What foods in Cobalt found in?

Research suggests that the top three food groups for Co in the human diet are: milk and dairy products, which account for approximately 32% of the total Co intake; fish and crustaceans, which account for approximately 20%, and condiments, sugar and oils, which account for about 16%. One investigation of specific foods (Leblanc *et al*., 2004) found that chocolate contains the highest level of Co, with molluscs and crustaceans, and dried fruit and nuts also containing the high levels in comparison to other foods. Other studies show different Co levels for the same products in neighbouring countries (e.g. coffee levels as reported by Ostapczuk *et al*. 1987), and even within the same food type in a single country (Nigerian tea as reported by Onianwa et al. 1999; Chinese tea as reported by Qiu-e et al. 1999). For example, an American study found less than 0.05 mg/L of Co in white bread, pancakes, cheddar cheese and spaghetti with meatballs (Dolan and Capar, 1999). The Agency for Toxic Substances and Disease Registry (ATSDR) website contains a profile for Co, including its presence in a number of different foodstuffs, and references to various studies (e.g., 20 brands of beer as reported by Camean et al. 1998).

How much Cobalt is in an average diet?

One study used the figures in the accompanying Table and calculated Co levels in a typical diet (Leblanc *et al.* 2004). It was found that adults (over 15 years old) ingested around 7.5µg of Co per day (µg = microgram = 1 x 10^{-6} grams) and children (aged 3 to 14 years) ingested approximately 7.3µg of Co per day. Another study found that the average Canadian male (40-65 years) had a 'mean daily dietary intake' of 12µg of Co per day, and that women of the same age group averaged 9µg of Co per day, with the differing levels mainly due to differences in diet between the sexes. (Dabeka and McKenzie 1995)

Food Group	Concentration (mg/kg)
Bread	0.006
Breakfast Cereals	0.008
Rice	0.010

Other Cereals	0.001
Milk	0.001
Cheese	0.018
Eggs and derivatives	0.005
Butter	0.018
Oils	0.018
Meats	0.008
Poultry and game	0.002
Offal	0.033
Fish	0.007
Crustaceans and molluscs	0.046
Vegetables (excluding potatoes)	0.006
Pulses	0.008
Fruits	0.009
Dried fruits and nuts	0.041
Chocolate	0.050
Sugar and derivatives	0.021
Soups	0.006
Ready meals	0.008

Cobalt contents in various foodstuffs (Le Blanc *et al,* 2004)

What is Vitamin B12?

Cobalt in the form of vitamin B12 (cyanocobalamin) is essential for human health. The molecular formula for Vitamin B12 is $C_{63}H_{88}CoN_{14}O_{14}P$ which means that only 4.34% of the molecule by weight is cobalt. It is a coenzyme in a number of cellular processes including the oxidation of fatty acids and the synthesis of DNA. It also works with folic acid in the synthesis of certain amino acids, and is required for the normal production of red blood cells. Vitamin B12 is also essential for the nervous system.

International organizations responsible for setting standard levels of vitamin intake, the FAO (Food and Agricultural Organisation of the United Nations) and WHO (World Health Organisation) recommend a 2.4µg/day of vitamin B12 (equivalent to 0.1 µg/day of cobalt) in the adult diet.
(ftp://ftp.fao.org/es/esn/nutrition/Vitrni/pdf/TOTAL.pdf)

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DISCLAIMER

This summary is intended to provide general information about the topic under consideration. It does not constitute a complete or comprehensive analysis, and reflects the state of knowledge and information at the time of its preparation. This summary should not be relied upon to treat or address health, environmental, or other conditions.

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Cobalt in the Atmosphere
(October 2006)

This short article aims to bring together information on Cobalt in the atmosphere.

What are the sources for Cobalt in the Air?

As Cobalt is a non-volatile material, it is rarely found in the atmosphere alone. Typically it is found in the air attached to anthropogenic pollutant particles, with a fraction of the cobalt found in natural dust. There are a number of anthropogenic sources for such particles, and natural sources of dust include the weathering of local geology, volcanic eruptions, forest fires and seawater spray. Coarse particles with diameters greater than 2µm may be deposited within 10km of the point of emission, whilst smaller particles may travel further. The mass median diameter of atmospheric cobalt has been found to be 2.6 µm (Milford and Davidson, 1985)

The main sources of atmospheric pollution for cobalt are industrial plants such as incinerators and chemical plants. In many countries, there is a limit on the amount of cobalt which such plants can release. Smaller sources for cobalt entering the atmosphere are exhaust fumes, the burning of fossil fuels (Vouk and Piver, 1983) and agriculture.

How much Cobalt is in the Atmosphere?

Friberg et al (1986) reported average natural background levels of cobalt in the atmosphere at around $1.0x10^{-9}mg/m^3$ and stated that the atmospheric concentrations of cobalt in remote areas is very low (less than $1.0x10^{-10}mg/m^3$ in the Antarctic) while in urban areas the ambient air concentration is usually higher (in the order of $1.0x10^{-6}$ mg/m^3 and exceeding $1.0x10^{-5}mg/m^3$ in heavily industrialised cities). Seiler et al. (1988) reported the cobalt concentrations in ambient air in several places in North and South America and in the United Kingdom and found that the levels were in the range $7.0x10^{-8}$ to $5.0x10^{-6}$ mg/m^3. Over the open ocean, cobalt concentrations ranged from 0.0004 to 0.08 ng/m^3 (Chester et al. 1991)

Khan et al (2003) investigating dustfall in Peshawar (Pakistan) found levels of cobalt at around 68µg per g of dustfall. In the paper these levels were ascribed to industrial uses such as the manufacture of alloys and its use as a catalyst in industry, although no direct evidence was given for this relationship.

Where does the Cobalt end up?

The length of time that cobalt stays in the atmosphere depends upon factors such as meteorological conditions, particle size, density and form. When the dust settles out of the atmosphere it can either land on soil, where it will eventually add to the soil concentration of cobalt, or into water, both of which are discussed in other documents available on this website.

Rainwater washes out any soluble cobalt species which are in the atmosphere. Studies have identified mean cobalt concentrations in rainwater to be between 0.3 µg/l in rural areas and 1.7µg/l in highly industrial areas (Arimoto, 1985; Hansson et al, 1988). Recent data from the Swedish Environmental

Research Institute (ECOLAS, pers comm) indicate that cobalt levels in precipitation over rural areas were below 0.039 μg/l between 2001 and 2003. Equally low values were seen in the Netherlands for 1999 and 2000 with the annual concentration of cobalt in rainwater being 0.001μmol/l, translating to a mean annual cobalt wet deposition rate of 4.1μg/m^2 in the Netherlands for 1999 and 2000. In comparison a wet deposition level of 12μg/m^2 was found in Massachusetts Bay as part of a total (wet and dry) of 47μg/m^2 between September 1992 and September 1993 (Golomb *et al*. 1997).

Studies in the UK have shown that between 33 and 44% of the cobalt occurred as stable organic complexes (Nimmo and Fones, 1997), meaning it was not bio-available to soil dwelling or aquatic organisms. Total Cobalt deposition flux at a site in the Rhone delta in southern France in 1988-1989 was 0.42±0.23kg/km^2 year with 0.15 kg//km^2 year in the form of wet deposition (Guieu et al. 1991)

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Cobalt in Soils
(February 2006)

This short article aims to explain the importance of cobalt in soils, concentrating on European Soils for which more information is currently available.

How much Cobalt is in European Soils?

The average Cobalt (Co) concentration in European soils is between 1- 20 mg/kg of Co (i.e. 1 – 20 mg of Co per kg of soil) dry weight, although this can become much higher in areas which are geologically rich in Co such as North Wales. For example, Paveley (1998) found natural levels of Co at over 2,500 mg/kg dry weight in soil. The study noted that the area had a totally healthy eco-system which had adapted to these naturally high concentrations. Maps of total Co and other metals in European Soils can be found on the FOREGS website (http://www.gsf.fi/publ/foregsatlas/index.php).

The majority of Co in the soil is not bioavailable. Co forms stable carbonate and hydroxide minerals which cannot be absorbed by the animal or plant life (Perez-Espinosa et al, 2004). Consequently, a very large amount of Co would have to be introduced into a volume of soil before local wildlife could be adversely affected.

How does Cobalt get into the Soil?

Cobalt occurs naturally in soils through two major pathways: the breakdown of organic matter which contains Vitamin B12, and the weathering of the local geology into soil particles. Mankind also adds Co to the soil, primarily through three mechanisms. The major mechanism is use of Co salts, e.g. Cobalt sulphate, as a feed additive to keep cattle and crops healthy in areas where there is not enough natural bioavailable Co. Smaller amounts of Co also enter the soil from the air transport of particulate emissions and application of sewage sludge onto fields.

Why is Cobalt added to some soils?

Due to the problems associated with Cobalt deficiency in agricultural soils, the behaviour of Co entering, and within, soils has been studied for a number of years. A lack of Co in a form which plants or earth dwelling organisms are able to absorb can have major effects on the health of the wildlife in an area. A classic example of this is the “Nova Scotia Moose Mystery” (Frank et al, 2004) where moose in Eastern North America were observed to have a wasting debilitating disease. It was found to be related to inadequate levels of bioavailable Co in their diet. The authors concluded

that Co salt licks should be introduced in limited areas of Nova Scotia to balance the moose's diet and restore them to health.

Bioavailable Co in soil is also necessary for healthy functioning of some plants. This is especially true for leguminous plants, Co being an essential nutrient for the micro-organisms which fix atmospheric nitrogen in the plants root nodules (Gad, 2002).

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Cobalt in the Aquatic Environment
(February 2006)

For the purposes of this short article, we have taken the aquatic environment to include freshwater systems such as lakes, streams and rivers and salt water or marine systems including estuaries and oceans.

What level of Cobalt is found in Natural Waters?

As Cobalt (Co) is naturally occurring and widely dispersed element, all natural waters contain a trace level of this element; sometimes called "background", i.e. the level that is present without any anthropogenic influence. This "background" level varies widely around the world and can even vary within the same water body. This can be due to water quality characteristics such as acidity or alkalinity (pH), temperature, and salinity, as well as, the plants and animals present and the composition of the sediments. For example, if Manganese is present in the sediment, then it will attract and adsorb almost all of the Co out of the water column and onto the mineral grains within the sediment. As a result, "nodules" form naturally in some areas of the ocean. One day Co may be recovered or extracted from these sediments as an alternative to terrestrial mining (i.e. mining on land).

The levels of Co found in surface waters of the Pacific and Atlantic Oceans are very low, averaging below 30 micrograms of Co per litre of sea water (i.e. 0.00003 g per litre). It should also be remembered that Co in the form of vitamin B12 is regarded as essential to the microbial ecology of the ocean.

In European freshwaters, the amount of dissolved Co is typically between 0.18 – 0.21 micrograms. Total concentrations vary between 0.22 – 0.5 micrograms, with the difference between total and dissolved being the proportion associated with organic matter in the water, e.g. humic acids. At these concentrations no toxicity to local aquatic organisms is expected to occur.

How does Co enter the aquatic environment?

Co can enter the aquatic environment from a number of sources, both natural and anthropogenic. The natural sources include volcanic emissions, the weathering of rocks by the action of water and decomposition of plant waste. One of the main human related releases into the aquatic environment is from sewage which, due to advances in modern sewage treatment, may only release around one part per billion (i.e. one unit Co for every billion units of sewage released) into the environment.

Where does Co go after the aquatic environment?

Natural and anthropogenic Co have the same transport pathways within the aquatic environment. The principle pathway involves binding to sediment at the bottom of the water body. Depending on the depositional environment, Co may remain on the sea/lake floor, become buried under further deposition or be transported into ocean where it will be deposited as sediment. The Co can be bound to sediments permanently unless there is a major change in the chemistry of the overlying waters. This is a natural geological process which, millions of years ago, created some of the ore bodies that are mined today.

Some Co, however, will remain in the water column (i.e. the water above the sediment). Co, if it is in the form of vitamin B12, is utilised by fauna in the environment. If this low level of Co in water was unavailable, a number of the aquatic organisms in these waters would show signs of vitamin B12 deficiency such as not achieving optimal growth or development.

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summary should not be relied upon to treat or address health, environmental, or other conditions.

May 29th 2006	**Cobalt**	**Cobalt Oxide**	**Cobalt Sulphide**	**Cobalt Chloride**	**Cobalt Sulphate**	**Cobalt Carbonate**	**Cobalt Nitrate**	**Cobalt Acetate**
Endpoint	CAS: 7440-48-4	CAS : 1307-96-6	CAS: 1317-42-6	CAS: 7646-79-9 AND **Cobalt Chloride $6H_20$** CAS : 7791-13-1 (NO EINECS)	CAS : 10124-43-3 AND **Cobalt Sulphate $7H_20$** CAS : 10026-24-1 (NO EINECS)	CAS: 513-79-1	CAS: 10141-05-6 AND **Cobalt Nitrate $6H_20$** CAS: 10026-22-9 (NO EINECS)	CAS: 71-48-7 AND **Cobalt Acetate $4H_20$** CAS: 6147-53-1 (NO EINECS)
	EINECS: 231-158-0	EINECS: 215-154-6	EINECS: 215-273-3	EINECS: 231-589-4	EINECS: 233-334-2	EINECS: 208-169-4	EINECS: 233-402-1	EINECS: 200-755-8
Physical Properties	None	None	None	None	None	None	None	None
Acute Oral	None	Xn; R22	None	Xn; R22	Xn; R22	None	None	None
Acute Inhalation	None	None	None	None	None	None	None	None
Dermal Irritation	Xi, R42/43	R43	R43	R42/43	R42/43	R42/43	R42/43	R42/43
Eye irritation	None	None	None	None	None	None	None	None
Dermal Sensitization	Xi, R42/43	R43	R43	R42/43	R42/43	R42/43	R42/43	R42/43
Respiratory Sensitisation	Xi, R42/43	None	None	R42/43	R42/43	R42/43	R42/43	R42/43
Chronic Toxicity	None	None	None	None	None	None	None	None
Reproductive Toxicity	None	None	None	Cat 2; R60	Cat 2; R60	Cat 2; R60	Cat 2; R60	Cat 2; R60
Mutagenicity	None	None	None	Cat 3; R68	Cat 3; R68	Cat 3; R68	Cat 3; R68	Cat 3; R68
Carcinogenicity	None	None	None	Cat 2; R49	Cat 2; R49	Cat 2; R49	Cat 2; R49	Cat 2; R49
Aquatic Environment	R53	N; R50/53	N; R50/53	N; R50/53	N; R50/53	N; R50/53	N; R50/53	N; R50/53
Indications of Danger	Xi	Xn, N	Xi, N	T, N	T, N,	T, N,	T, N,	T, N,
S-Phrases	2, 22, 24, 37, 61	2, 24, 37, 60, 61	2, 24, 37, 60, 61	53, 45, 60, 61	53, 45, 60, 61	53, 45, 60, 61	53, 45, 60, 61	53, 45, 60, 61

NOTE: Those classifications shaded grey do not enter force until the 30th ATP.

Specific Concentration Limits:

Cobalt Sulphide	
Concentration	Classification
C ≥ 2,5 %	Xi, N; R43-50/53
1 % ≤ C < 2,5 %	Xi, N; R43-51/53
0,25 % ≤ C < 1 %	N; R51/53
0,025 % ≤ C < 0,25 %	N; R52/53

Cobalt Chloride and Sulphate	
Concentration	Classification
C ≥ 25 %	T, N; R49-60-22-42/43-68-50/53
2,5 ≤ C < 25 %	T, N; R49-60-42/43-68-50/53
1 % ≤ C < 2,5 %	T, N; R49-60-42/43-68-51/53
0,5 % ≤ C < 1 %	T, N; R49-60-51/53
0,25 % ≤ C < 0,5 %	T, N; R49-51/53
0,025 % ≤ C < 0,25 %	T; R49-52/53
0,01 % ≤ C < 0,025 %	T; R49

Cobalt Nitrates, Carbonate and Acetates	
Concentration	Classification
C ≥ 2.5%	T, N; R49-60-42/43-68-50/53
1 % ≤ C < 2,5 %	T, N; R49-60-42/43-68-51/53
0,5 % ≤ C < 1 %	T, N; R49-60-51/53
0,25 % ≤ C < 0,5 %	T, N; R49-51/53
0,025 % ≤ C < 0,25 %	T; R49-52/53
0,01 % ≤ C < 0,025 %	T; R49

R22 = Harmful if swallowed
R43 = May cause sensitization by skin contact
R42/43 = May cause sensitisation by inhalation and skin contact
R49 = May cause cancer by inhalation
R50/53 = Very toxic to aquatic organisms, may cause long term adverse effects in aquatic environment
R53 = May cause long term adverse effects in the aquatic environment
R60 = May impair fertility
R68 = Possible risk of irreversible effects

S2 = Keep out of the reach of children
S22 = Do not breathe dust
S24 = Avoid Contact with skin
S37 = Wear Suitable Gloves
S45 = In case of accident or if you feel unwell, seek medical advice immediately (show the label where possible).
S53 = Avoid exposure – obtain special instructions before use.
S60 = This material and its container must be disposed of as hazardous waste.
S61 = Avoid release to the environment. Refer to special instructions/safety data sheets.

Note E: Substances with specific effects on human health (see chapter 4 of Annex VI of Directive 6715481EEC) that are classified as carcinogenic, mutagenic, and/or toxic for reproduction in categories 1 or 2 are ascribed Note E if they are also classified as very toxic (T+), toxic (T) or harmful (Xn). For these substances, the risk phrases R2O, R21, R22, R23, R24, R25. R26. R27, R28, R39, R68 (harmful), R48 and R65 and all combinations of these risk phrases shall be preceded by the word 'Also'.

Xi = Irritating
Xn = Harmful
T = Toxic
N = Dangerous for the environment