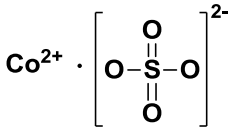
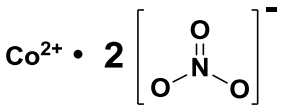
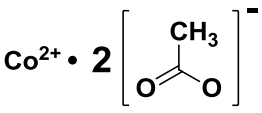
INITIAL TARGETED ASSESSMENT PROFILE (Human Health and Environment)

CAS RN and Chemical Name	**Sponsored Substances** Cobalt [Elemental cobalt]: CAS RN 7440-48-4 Cobalt chloride: CAS RN 7646-79-9 Sulphuric acid, cobalt (2+) salt (1:1) [Cobalt sulphate]: CAS RN 10124-43-3, CAS RN 10393-49-4 **Supporting Substances** Nitric acid, cobalt salt [Cobalt nitrate]: CAS RN 14216-74-1 Acetic acid, cobalt salt [Cobalt acetate]: CAS RN 5931-89-5
Structural Formula	**Sponsored Substances** **Co** CAS RN 7440-48-4 $Co^{2+}\,[Cl^-]_2$ CAS RN 7646-79-9  CAS RN 10124-43-3 and CAS RN 10393-49-4 **Supporting Substances**  CAS RN 14216-74-1  CAS RN 5931-89-5

SUMMARY CONCLUSIONS OF THE TARGETED ASSESSMENT

NOTE: The present assessment is targeted to address the following human health endpoints: toxicokinetics, respiratory irritation, repeated dose toxicity, carcinogenicity and genotoxicity; and the following environment endpoints: environmental fate and bioavailability in water, bioaccumulation potential, and acute and chronic toxicity to aquatic organisms. It cannot be considered as a full SIDS Initial Assessment. Summary information on use and exposure is also reported here. Other endpoints for human health and the environment are included in the Canadian screening assessment but have not been agreed upon by OECD member countries, and thus are not included in this profile.

The final screening assessment has been published under the responsibility of the Government of Canada. [http://www.ec.gc.ca/ese-ees/default.asp?lang=En&n=8E18277B-1]

Rationale for Targeting the Assessment

The Government of Canada "categorized" or prioritized all 23,000 chemical substances on its Domestic Substances List (DSL) from 1999 to September 2006, as required by its *Canadian Environmental Protection Act, 1999* (CEPA 1999). Using information from Canadian industry, academic research and other countries, Government of Canada scientists applied a set of rigorous tools to the 23,000 chemical substances on the DSL. They were categorized to identify those that were: **inherently toxic** to humans or to the environment and that might be **persistent** and/or **bioaccumulative**; and substances to which people might have **greatest potential for exposure**. During this priority-setting exercise, distinct approaches were taken for identifying substances of likely concern for human health and the environment, and subsequent assessment activities may have focused on either human health or ecological endpoints. Through categorization, the Government of Canada has identified approximately 4,000 of the 23,000 chemical substances on the DSL as priorities for further assessment, research and/or measures to control their use or release.

These cobalt substances were identified as high priorities for human health risk because elemental cobalt and cobalt sulphate were considered to pose greatest potential for exposure whereas cobalt chloride was considered to pose intermediate potential for exposure and all three substances had been classified by other agencies on the basis of carcinogenicity. They also met the ecological categorization criteria for persistence, and cobalt chloride and cobalt sulphate met the categorization criteria for inherent toxicity to aquatic organisms. Therefore, they were also identified as priorities for environmental risk. The Primary RN for cobalt sulphate is CAS RN 10124-43-3 while CAS RN 10393-49-4 is an Alternate Registry Number for this substance. In this document, cobalt sulphate refers to both CAS RN 10124-43-3 and CAS RN 10393-49-4: therefore three substances rather than four are mentioned.

Analogue/Category rationale

As shown above, these substances were identified as Canadian priorities for assessment by Canada's categorization process. Other cobalt substances were not considered at this time, but it is recognized that they may also contribute to physiological and environmental loadings via the common moiety of concern discussed below.

In solution, cobalt (II) salts including cobalt chloride and cobalt sulphate are considered to be toxicologically equivalent, as they generate a common moiety of concern, Co^{2+}. Elemental cobalt may also be oxidized under physiological and environmental conditions to produce Co^{2+} cations. Therefore, these substances were considered together in this assessment. Exposure of humans to cobalt from environmental sources was based on total cobalt.

All exposure concentrations are expressed in terms of total cobalt; measurement methods of cobalt in environmental media and foods do not distinguish between forms of cobalt. Much of the available data on cobalt toxicity in laboratory animals is for soluble cobalt (II) salts including the chloride and sulphate, with studies on the nitrate and acetate salts included as supporting information only (see below). These salts are expected to dissociate in physiological media to generate Co^{2+} cations and are therefore considered to be toxicologically equivalent. Studies with both the anhydrous and hydrated forms of the soluble Co(II) salts are considered relevant, as they are expected to be indistinguishable in solution. In biological media, elemental cobalt particles can bind to proteins, and may be oxidized to generate Co^{2+} ions. Blood and urine levels of cobalt (in solution) are well correlated with recent occupational exposure to soluble cobalt salts and cobalt metal. The systemic effects of elemental cobalt are likely primarily due to cations which are released from the particles and absorbed, whereas local effects may be due to both the ions released and the particles themselves at

the point of contact (i.e., lungs or skin) (reviewed in ATSDR 2004, IPCS 2006, IARC 2006).

Although there are data on almost all of the targeted endpoints for elemental cobalt, cobalt chloride and cobalt sulphate, measured data from cobalt's nitrate and acetate salts are also included as supporting information solely for additional information on some of the genotoxicity endpoints (mutagenicity, chromosomal aberration, DNA damage).

Physical-chemical properties

Elemental cobalt, cobalt chloride and cobalt sulphate are solid at room temperature. The melting points of cobalt and cobalt chloride are 1495°C and 724-737°C, respectively. Cobalt sulphate decomposes at 735°C. Even though no experimental data were available, their vapour pressure and Henry's Law constant are likely negligible at ambient temperature. Cobalt chloride and cobalt sulphate have high water solubility ranging from 450 to 562 g/L, and from 362 to 383 g/L, respectively. Elemental cobalt, in the form of powders, has a limited capacity for dissolution in water based on results from a 7-day Transformation Dissolution (T/D) Protocol test (OECD Guidance Document No. 29) indicating dissolved concentrations as high as 0.3 and 12.78 mg/L at loading rates of 1 to 100 mg/L, respectively. Ranges for the log K_{sw} (partition coefficient soil-water), log K_{sdw} (partition coefficient sediment-water) and log K_{spw} (partition coefficient suspended particles-water) for dissolved forms of cobalt are 0.41-3.49, 2.92-3.48 and 4.18-5.83 L/kg, respectively. The dissociation constants (pK_a, pK_b) and the partition coefficients between octanol and water (Log K_{ow}) and between organic carbon and water (Log K_{oc}) are not relevant to its environmental fate and so were not considered.

Human Health Targeted Endpoints

Oral absorption of cobalt in humans depends on the form of cobalt, the dose, and the nutritional status of the individual. Estimates of the absorption of cobalt chloride in humans range from 5 to 44% of the administered dose. There is no human data on the distribution of cobalt following oral absorption, but studies in laboratory animals indicate increased cobalt levels primarily in liver, as well as in other organs. In humans, orally administered cobalt for therapeutic purposes is eliminated primarily in faeces (reviewed in IPCS 2006).

Following inhalation exposure, large cobalt particles are deposited in the upper respiratory tract where they are subject to mechanical ciliary clearance, including transfer to the gastrointestinal (GI) tract. Smaller cobalt particles are, however, deposited in the lower respiratory tract where they may be solubilised and absorbed, or phagocytosed and absorbed. Cobalt solubility of these particles affects clearance from the lung, with faster absorption into blood and subsequent elimination for more soluble cobalt compounds. Data from experimental animal studies suggest that urinary excretion rates correlate with the rate of translocation into blood, and faecal excretion rates correlate with the clearance rate to the gastrointestinal tract (reviewed in IPCS 2006).

In humans, cobalt has been shown to stimulate red blood cell (RBC) production and this property has been used therapeutically. A transient increase in red blood cell numbers and haemoglobin levels was observed in a study of 6 adult male volunteers dosed orally with cobalt chloride at approximately 1mg Co/kg/day for 3 weeks. Similar effects were observed in anephric patients (without functioning kidneys) given cobalt chloride as treatment for anaemia at approximately 0.16 to 0.32 mg Co/kg-bw per day for several months. In contrast, women given lower doses of cobalt chloride (0.45 to 0.62 mg Co/kg-bw per day) during the third trimester of gestation did not have increased haemoglobin and red blood cells.

In short term and subchronic studies on cobalt chloride in rats, polycythemia (high RBC count) and increased haemoglobin were induced at doses equal to or greater than 0.5 mg Co/kg bw/day (lowest LOEL in experimental animals).

In the mid-1960s, a series of clinical case reports were published describing cardiomyopathy in subjects in North America and Europe who consumed large quantities of beer from specific producers containing cobalt sulphate, which was added by these breweries as a foam stabilizer. The exposure to cobalt from beer in these subjects was estimated to be 0.04 to 0.14 mg Co /kg-bw per day, based on a cobalt concentration in beer of 1 to 1.5 mg/L and consumption ranging from 8 to 30 pints per day (lowest LOAEL in humans of 0.04 mg Co/kg-bw per day). Potential influences on the susceptibility to the deleterious health effects included a protein-poor diet and the possibility of existing cardiac damage from prior alcohol abuse.

In rats given cobalt sulphate in the diet (24 weeks at 8.4 mg Co/kg-bw per day), heart enzyme activity and heart mitrochondrial ATP production were significantly reduced. The hearts of treated animals were found to have left ventricular hypertrophy and impaired ventricular function. Rats treated with higher doses of cobalt sulphate for shorter periods (26 mg Co/kg-bw per day for 8 weeks or cobalt chloride at 12.4 mg Co/kg-bw per day for 3 weeks) had similar cardiac degeneration. Guinea pigs similarly dosed with cobalt sulphate (20 mg Co/kg-bw per day for 5 weeks) had abnormal EKGs, increased heart weight, and cardiac lesions.

In some anaemic patients, cobalt therapy for longer periods (doses of 2.8 to 3.9 mg Co/kg bw per day for 3 to 8 months) resulted in thyroid hyperplasia and enlargement. Thyroid hyperplasia was also seen in some of the "beer drinkers cardiomyopathic" individuals at autopsy. Direct thyroid necrosis was observed in mice orally dosed with cobalt chloride (at 26 mg Co/kg bw/day) for 15 to 45 days.

The US National Toxicology Program (NTP) reported that in 13-week and 2-year cobalt sulphate inhalation studies (rat and mouse), all tested concentrations (0 to 11.38 mg Co/m^3) resulted in a 'spectrum of inflammatory, fibrotic and proliferative lesions', in the nose, larynx and lung with increasing severity at higher exposures, consistent with observations in humans. The LOAEC in these studies was 0.1 mg Co/m^3; no NOAEC was determined.

In a cross-sectional study[1] of humans (166 men and 28 women in the diamond polishing industry) exposed to cobalt dust, a LOAEC of 0.0151 mg Co/m^3 was determined based on a significant increase in the reported prevalence of eye, nose, and throat irritation and cough; and reduced lung function compared to 59 unexposed control workers (46 men and 13 women). A NOAEC of 0.0053 mg Co/m^3 was also determined. Cobalt exposure groups were defined based on air measurements at the time of the study, and exposure was confirmed by measurement of cobalt in urine. In another cross-sectional study, workers in a cobalt refinery who were exposed to cobalt metal, salts and oxides (average concentration of 0.125 mg Co/m^3) for up to 39 years had increased dyspnoea and wheezing, and decreased lung function compared to unexposed controls. Similarly, asthma symptoms were more prevalent in workers in a cobalt plant who were exposed to cobalt compounds. In a study examining the effects of similar cobalt exposures on the heart, no significant differences were reported in the electrocardiograms, blood pressure, heart rate, or clinical chemistry between 203 cobalt-exposed workers and 94 unexposed controls. It has been proposed that a mixture of cobalt and tungsten carbide may behave as a unique toxicological entity; however, studies on exposure to hard metal were not reviewed for this assessment.

Available studies on the carcinogenicity of cobalt in humans are based on exposures to cobalt in occupational settings, often in the presence of carbides such as tungsten carbide. Due to co-exposure with other substances, the human workplace data were considered insufficient by the International Agency for Research in Cancer (IARC) to conclude on the carcinogenic potential of elemental cobalt alone.

Although the available short-term and subchronic data do not provide any indication of carcinogenic potential following oral exposure to cobalt or soluble cobalt (II) salts, no studies of cancer from oral exposure in humans or experimental animals were identified. Data on the carcinogenicity of elemental cobalt in experimental animals consist of studies done by injection or implantation (intra-muscular, subcutaneous, intra-osseous, intra-peritoneal, intra-thoracic, and intra-renal); and the only study on carcinogenicity of cobalt chloride in experimental animals was carried out by subcutaneous injection. Injection or implantation site tumours were observed in some of these studies; however, the relevance of data from these routes of administration to carcinogenicity in humans resulting from oral or inhalation routes of exposure is unclear.

A US NTP 2 year cobalt sulphate inhalation study conducted in mice and rats, showed that aerosols of 0.11, 0.38, or 1.14 mg Co/m^3 induced a concentration-related increase in benign and malignant alveolar/bronchiolar tumours in both species and sexes (significant at high-concentration for male mice and rats; significant at mid- and high-concentrations for female mice and rats). There was also a concentration-related increase in incidence of benign and malignant adrenal tumours (pheochromocytomas) in the exposed female rats (significant at the high dose). Pheochromocytomas, a common age-related tumour in males, are less commonly seen in untreated female rats. The investigators considered the increased incidence of this tumour type to be an "uncertain" finding because it was seen only in the top dose group and was not supported by increased incidence or severity of hyperplasia. In contrast, IARC has classified elemental cobalt and soluble cobalt (II) salts as possibly carcinogenic to humans based on inadequate evidence in humans and sufficient evidence in experimental animals.

In vitro mutagenicity assays in bacteria with soluble cobalt (II) salts were primarily negative both with and without activation. Mixed results were obtained in a bacterial indicator assay for DNA damage (Rec assay in *B. subtilis*). In yeast, mainly positive results were obtained in mutagenicity and gene conversion assays in *S. cerevisiae* strains without activation. In mammalian cells *in vitro*, mutagenicity and cell transformation assays gave mixed results. However, cobalt chloride at high concentrations induced DNA damage (strand breaks and DNA-protein cross links), chromosome damage (micronuclei and sister chromatid exchanges) and aneuploidy in rodent and human cells in culture. In contrast, negative results were obtained for cobalt acetate and cobalt nitrate in chromosomal aberration assays in human cells in culture. Elemental cobalt, though insoluble, as particles

[1] Cross-sectional studies involve data collected at a defined time. They are often used to assess the prevalence of acute or chronic conditions.

induced DNA damage (strand breaks) and chromosome damage (micronuclei) *in vitro*.

In vivo, a single intraperitoneal injection of cobalt chloride induced micronuclei in mouse bone marrow. Cobalt chloride also induced aneuploidy, pseudoploidy and hyperploidy in the bone marrow and testes of hamsters when dosed intraperitoneally over 9 days; and chromosome aberrations in the bone marrow of mice given a single oral dose. In *Drosophila melanogaster*, cobalt chloride was positive in the wing spot test, and cobalt nitrate was positive for gene mutations, chromosomal deletion, non disjunction or mitotic recombination. Mice exposed to cobalt dust by inhalation for 13 weeks did not have an increase in micronuclei in the peripheral blood. There was no indication of increased DNA strand breaks or micronuclei in blood lymphocytes of 35 workers in a cobalt refinery exposed to cobalt dust compared to 27 unexposed workers.

The overall weight of the available evidence shows that cobalt metal particles and soluble cobalt (II) salts have the capacity to cause DNA damage and chromosomal damage *in vitro.* Although *in vivo* data for cobalt particles are limited, the *in vivo* data for cobalt chloride are consistent with the *in vitro* data for soluble cobalt (II) salts.

It is generally accepted that cobalt likely induces DNA damage through the generation of reactive oxygen species (ROS) and increased intracellular oxidative stress. Some of the supporting evidence is described below. Both elemental cobalt particles and Co^{2+} ions have been shown to generate ROS under biologically relevant conditions. An aqueous suspension of elemental cobalt particles (0.1 to 1.5 µm) was found to react with dissolved oxygen, forming a strong oxidant, likely Co-O-O$^{\bullet}$, and in the presence of either superoxide dismutase or Fe^{2+} ions the oxidant was found to release hydroxyl radicals. Hydroxyl radicals are extremely reactive and can damage virtually all types of key cellular macromolecules (carbohydrates, nucleic acids, lipids and amino acids). Free Co^{2+} ions (pH 7.4 phosphate buffer), promoted the conversion of hydrogen peroxide to the superoxide anion; however in the presence of chelating peptides such as glutathione, conversion of hydrogen peroxide to hydroxyl radicals was observed. This Fenton-type mechanism generated ROS in both *in vitro* and *in vivo* studies.

In vitro and *in vivo*, exposure to soluble cobalt resulted in increased evidence (indices) of oxidative stress. Cobalt (II) ions in the presence of hydrogen peroxide stimulated *in vitro* formation of 8-hydroxy-2'-deoxyguanosine (8-OH-dG), and cobalt sulphate induced DNA strand cross-links. *In vivo*, cobalt acetate, administered as a single intraperitoneal dose, induced oxidative DNA damage in liver, kidneys, and lungs of rats. Additional evidence supportive of an oxidative stress mechanism for causing DNA damage resulting in tumour induction comes from examination of tumours in mice treated with cobalt sulphate. In the tumours from this study, the guanine to thymine base pair transversion frequency in codon 12 of the K-ras oncogene was 55% compared to the zero percent base pair transversions detected in untreated control mice.

A second potential mechanism contributing to the indirect genotoxicity of cobalt is the inhibition of intracellular DNA repair processes, possibly through competition with other essential ions and binding to zinc finger domains in DNA repair proteins. *In vitro*, cobalt (II) inhibits the mammalian repair protein Xeroderma pigmentosum group A (XPA), which contains zinc finger domains. Cobalt chloride and cobalt acetate inhibited DNA repair following UV-induced DNA damage in human cells in culture, by inhibiting the incision and polymerization steps, but not the ligation step. In a small epidemiological study in which workers were exposed to cobalt dust, individuals with variations in several DNA repair genes had higher incidences of genotoxicity markers in the lymphocytes.

As the tumours observed in experimental animals are unlikely to have resulted from direct interaction with genetic material, these substances are considered to be non-genotoxic carcinogens.

Elemental cobalt, cobalt chloride, and cobalt sulphate possess properties indicating a hazard for the following human health endpoints: respiratory irritation, repeated dose toxicity, cardiomyopathy, carcinogenicity and genotoxicity.

Environment Targeted Endpoints

Fate

A fate analysis based on log K_{ow}, K_{oc} and typical mass-balance fugacity modelling is not applicable to elemental cobalt, cobalt chloride and cobalt sulphate, nor to the metal ions they release upon dissolution, because, as for other non-volatile chemicals, these substances exert negligible partial pressure and fugacity in air. Cobalt chloride and cobalt sulphate are highly soluble and, upon introduction in water, will dissociate and release cobalt ions (Co^{2+}). Elemental cobalt powders may also release cobalt ions in solution if discharged to surface waters. In addition to the cobalt ions, the substances will yield a variety of dissolved cobalt species of varying proportions depending on the environmental conditions. Under conditions commonly found in oxic freshwaters (i.e., pH between 5 and 9; E_h between 0.5 and 1 V), Co^{2+}, $CoCO_3^0$, and $CoHCO_3^+$ will be the dominant species in solution. Because of the tendency of cobalt to sorb to solid particles in aquatic media (log K_{spw} of 4.18-5.83), a

proportion of dissolved forms of this metal will end up in sediments (log K_{sdw} of 2.92-3.48), through adsorption to settling suspended particles. When released to dry soils, elemental cobalt will mainly remain there with some of the substance dissolving and leaching locally into ground and/or surface water ecosystems (via runoff) when the soil gets soaked by rain or melting snow/ice. Elemental cobalt is not expected to be found in significant amounts in the water column, considering that its density is greater than that of water. Considering the high solubility of cobalt chloride and sulphate, they are not likely to be found in solid forms in the environment unless they are released in a very dry environment. Being a non-gaseous element with a negligible vapour pressure, cobalt is emitted to air principally in the form of fine particulate matter (PM).

Elemental cobalt, cobalt chloride and cobalt sulphate all release cobalt ions in solution, that cannot be degraded. Therefore, biodegration, photodegradation and hydrolysis as a function of pH are not applicable to these inorganic metal-containing substances.

Bioavailability

For most metal-containing compounds, it is the potentially bioavailable metal ion that is liberated upon contact with water that is the moiety of toxicological concern. The bioavailability of metals controls their potential to cause adverse effects. Factors such as pH and ligands (e.g., major cations, dissolved organic matter) can in turn influence the bioavailability of dissolved metal ions. Experimental evidence shows that high water hardness and high dissolved organic carbon levels (DOC) decrease uptake of dissolved cobalt ions by aquatic organisms. Other evidence suggests that cobalt complexes with humic and fulvic acids may also be available for uptake.

Bioaccumulation and Biomagnification

Cobalt is an essential micro-nutrient element for bacteria, plants and animals. As such, its uptake is expected to be regulated to some extent by many organisms through mechanisms of homeostasis and detoxification. Thirty-one bioaccumulation and bioconcentration factors (BAF and BCF) values were found to be acceptable from the literature for various species of algae, invertebrates and fish (laboratory and field studies), and range from 7.4 to 3110 L/kg (average is 878 L/kg, with only 2 values > 2000 L/kg). Four biota-to-sediment accumulation factors (BSAF-sediment) values obtained for freshwater invertebrates range from 0.091 to 0.645. Four biomagnification factors (BMF) were found in the literature for cobalt in marine and freshwater environments, with values ranging from 0.004 to 0.087. Overall, considering these values as well as the metal regulation mechanisms that most organisms possess, the bioaccumulation and biomagnification potentials of cobalt in aquatic ecosystems are expected to be low.

Aquatic toxicity

Being soluble in water, elemental cobalt powders, cobalt chloride and cobalt sulphate, like other soluble cobalt compounds, will release cobalt species upon dissolution, in particular the free ion Co^{2+}. Reliable short term (acute) studies for soluble cobalt compounds were identified for 12 invertebrate species and 3 fish species; toxicity values range from 89 to 585 800 µg/L as total dissolved cobalt. Reliable long-term (chronic) studies were identified for 5 plant/algae species, 7 invertebrate species and 3 fish species; toxicity values range from 2.9 to 59 000 µg/L as total dissolved cobalt. A species sensitivity distribution (SSD) was developed using the chronic toxicity data. The Weibull model provided the best fit for the data and the 5^{th} percentile (HC_5), i.e., hazardous concentration to 5% of species, of the SSD plot derived in Canada is 2.5 µg/L. Overall, there is experimental evidence that dissolved cobalt has a relatively high potential to cause harm to aquatic organisms following short-term and longer-term exposure at very low concentrations.

Elemental cobalt, cobalt chloride, and cobalt sulphate possess properties indicating a hazard for the environment. They have acute and chronic aquatic toxicity below 1 mg/L (total dissolved cobalt),

Uses / Exposure

Uses

There are a few applications of elemental cobalt; it is predominantly used as a component in alloys and carbides for applications requiring high strength and temperature resistance. Anhydrous cobalt chloride is commonly used as an indicator in desiccants. Cobalt sulphate is the most inexpensive form of ionic cobalt and is used in the electroplating industry and agriculturally as a feed supplement and fertilizer. Cobalt chloride or sulphate may also be used as the cobalt source in storage batteries, porcelain pigments, glazes and ink driers. While the chloride and sulphate salts may be the source of cobalt for applications such as pigments, glazes, and batteries, generally the salt is thermally decomposed or calcined; therefore cobalt sulphate and chloride will not be present in the final product.

Elemental cobalt uses reported under a survey in Canada include pigment manufacturing, chemical and alloy production, cement production, metallurgy, manufactured automotive parts, formulation component,

water/waste treatment chemical, catalyst/accelerator/initiator/activator, and copper refining.

Table 1 shows an estimate of the uses of the three cobalt substances of interest by industrial sector as reported by the Cobalt Development Institute for 2008.

Table 1: World-wide cobalt uses estimated by the Cobalt Development Institute for 2008 for elemental cobalt, cobalt chloride and cobalt sulphate.

Use	Estimated % of the world market	Cobalt substances used for manufacture			
		Elemental	Chloride	Sulphate	Other cobalt compounds
Batteries	27	x	x	-	x
Super alloys	19	x		-	x
Hard Material Tools	14	x		-	x
Colours – Glass, enamels, plastics, ceramics, artists colours, fabrics	10	-		x	x
Catalysts	9	-	x	x	x
Magnets	7	x		-	x
Tire Adhesives, Soaps, Driers for paint and dyes	6	-		-	x
Other Alloys	4	x		-	x
Feedstuffs and others uses	4			x	-

Natural Sources

Cobalt is a naturally occurring element in the terrestrial crust. Cobalt concentrations in the upper continental crust have been determined to average about 25 ppm and to range between 0.1 and 110 ppm. Cobalt is not known to naturally exist in its elemental (metallic) form; naturally occurring cobalt is comprised of various mineral, oxide and salt forms; sources include windblown continental dusts, weathering of rocks, seawater spray, forest fires and volcanoes.

Natural emissions to the atmosphere have been estimated to range between 690 and 11 000 tonnes of cobalt per year globally. Atmospheric fall-out and introduction of cobalt into surface water and soil as a result of natural weathering and erosion processes are reflected in the geochemical background levels in these media.

Anthropogenic Sources

Anthropogenic sources of cobalt include burning of fossil fuels (primarily oxides), sewage sludge, phosphate fertilizers, mining and smelting of cobalt containing ores and industrial processes that use cobalt compounds.

The quantities of elemental cobalt, cobalt chloride and cobalt sulphate that are used, imported or manufactured in Canada, based on a 2006 survey in Canada, range from 100 000 to 10 million kg, 10 000 to 1 million kg, and 64,000 to 10 million kg, respectively. It should be noted that the term “manufacture” as defined in the survey includes the incidental production of a substance at any level of concentration as a result of the manufacturing, processing or use of other substances, mixtures, or products. Also, it should be noted that products containing cobalt, cobalt chloride or cobalt sulphate may enter Canada even if they are not identified as such in the Canadian survey because they may be imported unknowingly in manufactured items, or in quantities below the 100 kg reporting threshold for the survey.

Canada’s National Pollutant Release Inventory (NPRI) data for cobalt are not form-specific and therefore,

represent all forms of cobalt. Between 1995 and 2008, the on-site release over the total release reported decreased from approximately 10% to 2%, while the proportion sent to disposal increased from approximately 15% to 27%. Off-site recycling was the most significant removal pathway corresponding to approximately 70% of the annual total reported between 1995 and 2008. Most of the cobalt produced in Canada is exported, so that in Canada a relatively small proportion of total cobalt releases are associated with the manufacture of consumer products.

Human Exposure Estimates

The principal route of exposure to cobalt by the general population is diet. Cobalt occurs naturally in soil at concentrations ranging between 0.1 ppm and 110 ppm and may enter the food chain via uptake by plants and livestock, consequently cobalt is found in the majority of foods at varying levels. In Canada, the estimated daily dietary intake of cobalt by adults is 17 μg/day, based on the 2002 Canadian Total Diet Study conducted in one major city (Vancouver). Two separate analyses of food samples from the U.S. Food and Drug Administration Total Diet Studies for 1982-1984 reported an average dietary cobalt intake of 11 μg/day and 14 μg/day, respectively, for males in the 25 – 30 years age group. The WHO has estimated daily cobalt intakes via food of 5-45 μg/day. Consumer exposure to cobalt may occur through use of personal care products; however, the actual concentrations and forms of cobalt are generally not known with precision. Occupational exposure was not considered.