Sparingly soluble cobalt salts: Human health tier II assessment

27 November 2014

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Cobaltate (CoO21-), lithium	12190-79-3
Phosphoric acid, cobalt(2+) salt (2:3), hydrate	10101-56-1
Phosphoric acid, cobalt(2+) salt (2:3)	13455-36-2
Cobalt molybdenum oxide (CoMoO4)	13762-14-6
Phosphoric acid, ammonium cobalt(2+) salt (1:1:1)	14590-13-7

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:**www.nicnas.gov.au**

Disclaimer

ACRONYMS & ABBREVIATIONS

Grouping Rationale

The chemicals in this group are sparingly soluble cobalt salts. Although sparingly soluble in water, these compounds are expected to have moderate dissociation in artificial gastric fluid and low dissociation in lysosomal fluid based on bioaccessibility tests undertaken on cobalt lithium dioxide (CAS No. 12190-79-3) (CoRC, 2014). While cobalt lithium dioxide contains cobalt in the trivalent state, on dissolution, the Co^{3+} is expected to undergo protonation forming an unstable hydrated species giving rise to the divalent Co^{2+} ion (Cotton & Wilkinson, 1988). Therefore, cobalt lithium dioxide may be grouped with these chemicals for assessment purposes.

No solubility data are available for the phosphate or molybdate cobalt compounds in this group. The solubility of these phosphate and molybdate compounds is expected to be similar to erythrite, a hydrous cobalt arsenate which has low solubility at neutral pH, but is appreciably soluble in acidic pH (Zhu et al., 2013).

Considering the limited data on chemicals in this group, data for oral and systemic toxicity can be read-across from studies on soluble cobalt compounds (NICNASa) according to the principles of OECD (2014). This is possible as the chemicals in this group are expected to have moderate bioaccessibility and bioavailability in artificial gastric fluid (CoRC, 2014). Regarding other routes of exposure the chemicals in this group are expected to have a hazard profile ranging between non-toxic to that of cobalt oxide (NICNASb) based on the available bioaccessibility data. Hence, data have been read across from cobalt oxide (NICNASb) where relevant.

Import, Manufacture and Use

Australian

A search of local websites and safety data sheets have identified the following uses:

Cobalt lithium oxide (CAS No. 12190-79-3) has reported commercial use as a electrochemical ingredient in lithium ion batteries (rechargeable batteries).

Cobalt phosphate (CAS No. 13455-36-2) has reported domestic use in artists' paint.

International

The following international uses have been identified through European Union Registration, Evaluation, Authorisation and Restriction of Chemicals (EU REACH) dossiers; Galleria Chemica; Substances and Preparations in the Nordic countries (SPIN) database.

Cobalt phosphate (CAS No. 13455-36-2) has reported domestic use including in artists' paint (up to 70%);

Cobalt phosphate (CAS No. 13455-36-2), cobalt phosphate hydrate (CAS No. 10101-56-1) and cobalt ammonium phosphate (CAS No. 14590-13-7) have reported commercial uses as colourants and dyes in glass, ceramic glazes, enamels and plastic resins.

Cobalt molybdenum oxide (CAS No. 13762-14-6) has reported site limited use as a chemical intermediate.

The following site limited uses have been identified for cobalt lithium oxide (CAS No. 12190-79-3):

- as chemical intermediate;
- electrochemical ingredient in lithium ion batteries; and
- as a intermediate for computer, electronic and optical products and electrical equipment.

Cobalt ammonium phosphate (CAS No. 14590-13-7) has reported non-industrial use as a plant nutrient.

Restrictions

Australian

Cobalt and its compounds are listed in Schedule 10 (prohibited carcinogens, restricted carcinogens and restricted hazardous chemicals) of the Work Health and Safety Regulations (WHS, 2014) for restricted use in abrasive blasting at a concentration of greater than 0.1 % cobalt.

International

No known restrictions have been identified.

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals in this group are not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified (Galleria Chemica):

Chemicals in this group have exposure limits (time weighted average–TWA) of 0.02–0.1 mg/m^3 in different countries such as Spain, USA (Washington), Canada (Yukon), Ireland and Greece.

Health Hazard Information

Toxicokinetics

This group of chemicals consists of sparingly soluble cobalt compounds. Bioaccessibility data on one of the chemicals in this group demonstrated moderate dissociation in artificial gastric fluid and low dissociation in lysosomal fluid (Stopford et al., 2003; CoRC, 2014).

The oral cobalt absorption in humans varies from 3-97 % depending on the type and dose of the cobalt compounds administered (Leggett, 2008). A study by Firrolo et al. (1999) demonstrated that upon oral administration; absorption, disposition and elimination of various cobalt salts were the same, and that the original identity of the salts do not affect cobalt ion absorption in vivo once the compounds have dissociated (Firrolo et. al., 1999).

Dermal absorption of cobalt has been demonstrated to be relatively low (NICNASa). In an in vitro study, percutaneous absorption of soluble cobalt through human skin was 1.08 % from a 100 mg/mL cobalt chloride hexahydrate (CAS No. 7791-13-1) (NICNASa; CDI, 2014). Absorption through intact skin of guinea pigs was very low (<1 %) while absorption through abraded skin was almost 80 % after a three-hour exposure (ATSDR, 2004).

Absorption following inhalation exposure to cobalt depends on the particle size. Particles larger than 2 µm are deposited in the upper respiratory tract, whereas smaller particles are deposited into the lower respiratory tract. Larger particles can be absorbed into the blood after dissolution or can be moved into the gastrointestinal tract by mucociliary action. Smaller particles are either dissolved or phagocytised by macrophages (ATSDR, 2004).

Once absorbed, the cobalt ion is widely distributed in the body, including the skeleton, with the highest concentration found in the liver and kidney. After inhalation exposure, although there is a high initial excretion in faeces, the primary route of elimination is via urine (IPCS, 2006). Urinary excretion of cobalt from workers under experimental conditions was multiphasic: an initial rapid elimination ($T_{1/2}$ = 44 hours), a second slower elimination ($T_{1/2}$ = 10 days) and a third long-term retention ($T_{1/2}$ in the order of years) (Leggett, 2008). The estimated long-term half life of cobalt after oral intake by a female volunteer was 625 days (Leggett, 2008). Faecal elimination is the primary route of elimination after oral exposure. Faecal elimination varies from 3–99 % in individuals, depending on the dose, form and nutritional status of the individual (IPCS, 2006). Although the liver and kidneys had the highest initial cobalt concentrations after exposure, concentrations were considered low at 100 days. One study demonstrated that cobalt sulfate (CAS No. 10124-43-3) administered orally results in a dose-dependent increase in cobalt levels in foetal blood and amniotic fluid (ATSDR, 2004).

Acute Toxicity

Oral

Chemicals in this group have low acute toxicity in animal tests following oral exposure.

In a study carried out on cobalt molybdenum oxide (CAS No. 13762-14-6) according to OECD Test Guideline (TG) 423, the median lethal dose (LD50) in female Sprague Dawley (SD) rats is > 2000 mg/kg bw. Observed sub-lethal effects included a decrease in spontaneous activity and piloerection in one out of six animals (REACHa).

In a study carried out according to OECD TG 425, the LD50 in female SD rats is > 5000 mg/kg bw cobalt lithium dioxide (CAS No. 12190-79-3). There were no adverse clinical signs or abnormal behaviour (REACHb).

Dermal

No data are available.

Given that the toxicokinetics data show low dermal absorption (ATSDR, 2004; Leggett, 2008; CDI, 2014), acute toxicity through the dermal route is not expected.

Inhalation

No data are available on chemicals in this group. Considering that the chemicals in this group have similar bioaccessibility to cobalt oxide in artificial alveolar fluid, the chemicals in this group should be classified for acute inhalation toxicity, similar to cobalt oxide (NICNASb) (refer to **Regulatory Control–Occupational Health and Safety**).

In a study conducted according to OECD TG 436, male and female Crj: CD(SD) rats were exposed to a single inhalation concentrations of 0.11 and 0.53 mg/L of cobalt oxide (CAS NO. 1307-96-6) (mass median aerodynamic diameter (MMAD) ranging from 3.3–3.7 μm) in the main study group for four hours, and then observed over 14 days and 0.05, 0.11, 0.53, 1.07 or 5.06 mg/L cobalt oxide in a satellite group, for four hours and observed for 24 hours. In the main study group, all animals exposed to concentrations of 0.11 and 0.53 mg/L died prematurely on days four and three of the observation period, respectively. In the satellite group observation only occurred for 24 hours and during this time mortality was reported at all concentrations above 0.05 mg/L (0.11 mg/L - 2/3 males and 0 females, 0.53mg/L - 3/3 males and 1/3 females, 1.07 mg/L - 2/3 males and 3/3 females, 5.06 mg/L - 3/3 males and 1/3 females). Sub-lethal effects reported in the main study included ataxia, tremor and dyspnoea across all exposure groups. In the satellite group, sub-lethal effects of ataxia, tremor, reduced muscle tone and dyspnoea were reported in animals exposed to the two highest concentrations (1.07 and 5.06 mg/L). Based on this study the LC50 was calculated to be 0.06 mg/L (NICNASb).

Corrosion / Irritation

Skin Irritation

Chemicals in this group are not considered to be skin irritants. Three in vitro studies performed in accordance or similar to OECD TG 439 and 431, using cobalt lithium dioxide (CAS No. 12190-79-3) and cobalt molybdenum oxide (CAS No. 13762-14-6) suggest that the chemicals are not skin irritants.

In an in vitro study carried out according to OECD TG 439, cobalt lithium dioxide (CAS No. 12190-79-3) was introduced for 15 minutes at a volume of 10 mg to a cell culture of human-derived epidermal keratinocytes, cultured to form a multilayered, highly differentiated model of the human epidermis. Each sample was performed in triplicate. After exposure, a 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay was performed. The score obtained after the 15-minute treatment was 76.9 % for cell viability. As this value is above 50 % viability threshold, the substance was not considered to be an irritant (REACHb).

In an in vitro study carried out according to OECD TG 431, cobalt lithium dioxide (CAS No. 12190-79-3) was introduced for three or 60 minutes, at a volume of 25 mg, to a cell culture of human-derived epidermal keratinocytes, cultured to form a multilayered, highly differentiated model of the human epidermis. Each sample was performed in duplicate. After exposure, a MTT assay was performed. Scores obtained after the three and 60 minute treatments were 100.1 % and 93.5 % for cell viability. As values did not go below the threshold for corrosivity of less than 50 % cell viability for the three minute exposure, or less than 15 % cell viability for the 60 minute exposure, cobalt lithium dioxide (CAS No. 12190-79-3) was not considered to be corrosive (REACHb).

In an in vitro study carried out according to Commission Regulation (EC) No. 761/2009, cobalt molybdenum oxide (CAS No. 13762-14-6) was introduced at a volume of 25 mg to a cell culture of human-derived epidermal keratinocytes, cultured to form a multilayered, highly differentiated model of the human epidermis. Each sample was performed in triplicate. The score obtained for formazan production averaged 110.6 %. As this value is above 50 % threshold for irritancy, the substance was not considered to be an irritant (REACHa).

Eye Irritation

Based on the available data chemicals in this group are not eye irritants.

In an in vivo study carried out according to OECD TG 405, cobalt lithium dioxide (CAS No. 12190-79-3) was applied to the eyes of three male New Zealand White rabbits. Marked reddening of the conjunctivae were observed in two female animals one hour after treatment and slight reddening was observed in the male animal 24 hours after treatment. Overall irritation results 24, 48 and 72 hours after application per animal are as follows: corneal opacity - 0.0, 1.0, 1.0; iritis - 0.0, 0.0, 0.0; conjunctival redness, 0.67, 0.33, 0.33 and conjunctival oedema - 0.0, 0.0 and 0.0. Slight reddening of the conjunctivae was noted in all animals and slight ocular discharge was present in two animals; these effects were transient and resolved during the 14 day study (REACHb).

In an in vitro study carried out according to OECD TG 437, cobalt lithium dioxide (CAS No. 12190-79-3) was administered to bovine corneas for 240 minutes at a volume of 0.75 mL (20 % w/v). Each chemical test was performed in triplicate. After exposure, a mean in vitro score of 0.29 was determined. The mean in vitro score for the negative control, with no increase in opacity or permeability of the corneas, was approximately 0.89, whereas the mean in vitro score for the positive control was approximately 208.67, with clear observations of opacity and distinctive permeability of the cornea, corresponding to a corrosive effect. Under the experimental conditions reported, cobalt lithium dioxide (CAS No. 12190-79-3) is not corrosive to the eye (REACHb).

In an in vitro study carried out similar to OECD TG 437, cobalt molybdenum oxide (CAS No. 13762-14-6) was administered to bovine corneas at a volume of 0.75 mL (1039.0 mg, 1091.6 mg and 1068.1 mg). Each chemical test was performed in triplicate. After exposure, a mean in vitro score of -0.37 was determined. The mean in vitro score for the negative control, with no increase in opacity or permeability of the corneas, was approximately 1.32, whereas the mean in vitro score for the positive control was approximately 93.67, with clear observations of opacity and distinctive permeability of the cornea, corresponding to a corrosive effect. Under the experimental conditions reported, cobalt molybdenum oxide (CAS No. 13762-14-6) is not corrosive to the eye (REACHa).

Sensitisation

Skin Sensitisation

Chemicals in this group are not considered to be skin sensitisers based on a mouse local lymph node assay (LLNA) carried out in accordance with OECD TG 429, using cobalt lithium dioxide (CAS No. 12190-79-3).

In the LLNA assay, five female CBA mice were treated by topical application of 25 μL of either 50 %, 25 % or 12.5 % cobalt lithium dioxide (CAS No. 12190-79-3) suspended in acetone. Application to the dorsal surface of each ear was performed once daily over three consecutive days. All mice were then dosed with radioactive methyl thymidine via an intravenous injection. The proliferative response of lymph nodes was determined by the number of radioactive disintegrations per minute per lymph node (DPM/NODE) and as the ratio of radioactive incorporation into lymph nodes (stimulation index). The reported results for 12.5 %, 25 % and 50 % concentrations were 1026.5, 1187.0 and 1150.3 DPM and 1.5, 1.8 and 1.7 for stimulation index, respectively. There were no adverse clinical signs during the observation period and the chemical was not considered sensitising (REACHb).

Observation in humans

Respiratory sensitisation

Based on the available epidemiological evidence, the chemicals in this group are recommended for classification for sensitisation via the inhalation route of exposure (refer to **Observation in Humans**).

Several epidemiological studies conducted in cobalt-producing facilities support the findings that occupational exposure to inorganic cobalt compounds is associated with occupational asthma (ATSDR, 2004; WHO, 2006; CoRC 2014). Specifically, studies show that there was a significant correlation between decreasing lung function tests (FEV1/VC ratio) and increasing concentrations of cobalt in the air and urine of occupationally exposed workers (CoRC, 2014).

Repeated Dose Toxicity

Oral

No data are available for the chemicals in this group. As data on cobalt lithium dioxide (CAS No. 12190-79-3) show moderate bioaccessibility and bioavailability in artificial gastric fluid, data from soluble cobalt compounds are read-across (NICNASa; OECD, 2014). Data available from the NICNAS assessment of soluble cobalt compounds (NICNASa), particularly data available for cobalt sulfate heptahydrate (CAS No. 10026-24-1) and cobalt chloride hexahydrate (CAS No. 7791-13-1) show that the main effect after repeated oral exposure to soluble compounds is polycythaemia (increased erythrocytes). However, this effect is reversible after cessation of exposure (NICNASa). The severity and/or reversibility of effects seen in these studies do not meet the criteria for hazard classification.

Dermal

No data are available for chemicals in this group.

Given that the toxicokinetics data shows low dermal absorption (ATSDR, 2004; Leggett, 2008; CDI, 2014), repeated dose toxicity through the dermal route is not expected.

Inhalation

No data are available for the chemicals in this group. Considering that the chemicals in this group have similar bioaccessibility to cobalt oxide in artificial alveolar fluid, data on systemic toxicity will be read across from cobalt oxide (NICNASb; OECD 2014) for repeated dose toxicity via the inhalation route (refer to **Recommendation** section).

Observation in humans

A read-across approach was taken from data available on analogue chemicals, cobalt sulfate heptahydrate (CAS No. 10026-24-1) and cobalt chloride (CAS No. 7646-79-9).

Cobalt-induced cardiac failure was attributed to 50 patients who had ingested, over a period of years, an average of 0.2 to 0.7 mg/kg bw/day of cobalt sulfate heptahydrate (CAS No. 10026-24-1) that was added to stabilise foam in beer. The first signs of beer–cobalt cardiomyopathy were gastrointestinal effects including nausea, vomiting and diarrhoea. Beer–cobalt cardiomyopathy and alcoholic cardiomyopathy have similar symptoms, although the onset of beer–cobalt cardiomyopathy was found to be very abrupt. These patients had protein-poor diets and consumed significant quantities of alcohol, which might affect the

symptoms of cardiomyopathy, pulmonary rales and pulmonary oedema that were observed (ATSDR, 2004). In an occupational study of 237 workers from a cobalt refinery, no dose-effect relationships were observed between cobalt exposure and incipient signs of cardiomyopathy (Lantin et al., 2013).

Polycythaemia (increase in erythrocytes) and an increase in haemoglobin levels have been observed in all subjects in another study where six volunteers were exposed to a daily dose of 150 mg/day of cobalt chloride (CAS No. 7646-79-9) for up to 22 days. Erythrocyte counts returned to normal in all subjects 15 days after treatment (ATSDR, 2004).

As a result of cobalt's effects which have potential to increase haemoglobin levels, it has been previously used therapeutically to treat anaemia. In a series of studies, anephric patients were treated with 0.65–4.0 mg/kg/day of cobalt chloride (CAS No. 7646-79-9), daily for 3–32 weeks. The increase in erythrocytes resulted in a decreased need for blood transfusions (ATSDR, 2004). In another series of studies, sickle-cell anaemia patients receiving cobalt therapy showed enlargement and hyperplasia of the thyroid gland, which were reversible upon cessation of cobalt therapy (ATSDR, 2004). When pregnant women were treated for 90 days with 2 to 2.4 mg/kg/day of cobalt chloride (CAS No. 7646-79-9), it did not prevent the common occurrence of decreasing levels of haemoglobin and haematocrit levels observed during pregnancy. There were also no effects observed on the heart, in liver function or obvious birth defects (ATSDR, 2004).

Genotoxicity

Limited data are available on one of the chemicals in this group. As the chemicals in this group have moderate bioaccessibility and bioavailability in artificial gastric fluid, data are read-across from the NICNAS assessment of soluble cobalt compounds (NICNASa). It was concluded that effective protective processes exist in vivo to prevent genotoxicity in human (OECD, 2014b) and hence, no classification is required (NICNASa).

In vitro

In a mammalian cell gene mutation assay carried out according to OECD TG 476, cobalt lithium dioxide (CAS No. 12190-79-3) did not induce mutation at the hypoxanthine-guanine phosphoribosyl transferase (HPRT) locus of L5178Y mouse lymphoma cells. Concentrations of 10 to 60 μg/mL (up to toxic concentrations determined in previous experiments) were tested for three hours in the absence and presence of a rat liver metabolic activation system (S9) (REACHb).

Carcinogenicity

No data are available on the chemicals in this group. Considering that the chemicals in this group have similar bioaccessibility to cobalt oxide (NICNASb; OECD, 2014) in artificial alveolar fluids, data on carcinogenicity could be read across from cobalt oxide. However, the available carcinogenicity data for cobalt oxide are inappropriate for interpreting the hazard classification due to the non-physiologically relevant exposure routes used, absence of guideline laboratory studies and confounding in epidemiological studies arising from mixed exposure to cobalt oxide with other cobalt compounds and metals (nickel, arsenic, tungsten carbide). The mechanisms of carcinogenicity for cobalt compounds found to be carcinogenic by IARC (IARC, 2006) have not been clearly identified, making read across for compounds of different solubility difficult. Further studies are required to investigate the specific mode of action prior to hazard classification.

Reproductive and Developmental Toxicity

No data are available on the chemicals in this group. Based on data that indicate moderate bioaccessibility and bioavailability in artificial gastric fluid, data are read-across from the NICNAS assessment of soluble cobalt compounds (NICNASa; OECD, 2014). Data available for cobalt sulfate heptahydrate (CAS No. 10026-24-1) and cobalt chloride (CAS No. 7646-79-9) indicate that there is a concern for reproductive toxicity. Cobalt sulfate heptahydrate is classified as hazardous, a Category 2 substance toxic to reproduction, with the risk phrase ‘May impair fertility’ (T; R60) in HSIS (Safe Work Australia). Based on read-across principles (OECD, 2014), the data for cobalt sulfate heptahydrate support a recommendation to classify this group of chemicals.

Reproductive toxicity

In a 12-week oral fertility study, adult male Swiss mice were exposed to cobalt chloride (CAS No. 7646-79-9) in drinking water (average of 25, 47 or 93 mg/kg bw/day) and then mated with unexposed females. The number of pregnant females and implantation sites were significantly reduced in females mated with exposed males at 47 and 93 mg/kg bw/day. At all doses, the incidence of resorption was significantly higher, whereas the number of viable foetuses decreased. Decreased relative testes weight, decreased sperm concentration, and testis necrosis and degeneration were observed (Elbetieha et al., 2008).

In the 13-week NTP study, rats and mice (10 animals/sex/species) were exposed to aerosols containing 0, 0.3, 1.0, 3.0, 10 or 30 mg/m^3 cobalt sulfate heptahydrate (CAS No. 10026-24-1) for six hours a day, five days a week for the duration of the study. Absolute and relative testis weights and epididymis weight were significantly decreased, together with increased number of abnormal sperm in male mice at 30 mg/m^3 cobalt sulfate heptahydrate (CAS No. 10026-24-1). Data were not collected on mice exposed to lower concentrations. Sperm motility was significantly reduced in mice exposed to $\geq$3 mg/m^3, but data were not collected on mice exposed at lower concentrations (NTP, 1991).

Developmental toxicity

While there are several non-guideline studies on developmental toxicity for soluble cobalt compounds, it is difficult to draw firm conclusions about the developmental toxicity of these chemicals due to various methodological deficiencies. Testing is currently underway for this endpoint.

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include a systemic long-term effect (reproductive toxicity), systemic acute effect (acute toxicity from inhalation exposure) and local effect (respiratory sensitisation). The chemicals may also cause toxic effects following repeated exposure through inhalation. There are

insufficient data to enable classification for carcinogenicity.

Public Risk Characterisation

Some chemicals in this group, cobalt phosphate (CAS No. 13455-36-2), have reported use as in specialty artists' paint. The artists' paint, cobalt violet, can be purchased locally and be used in a domestic setting. Considering that this paint is a specialised pigment that is costly, and is used in instances requiring colour-fade protection for the painting over many decades, this product is likely to be used by speciality artists. In this form, the main concern with the use of the product is reproductive toxicity through significant ingestion. In a professional setting, significant ingestion is unlikely, therefore the risk is not considered to be unreasonable. Furthermore, the safety data sheet warns against ingestion of the product.

Although the public could come into contact with articles/coated surfaces containing chemicals in this group, it is expected that the chemicals will be bound within the article/coated surface and hence will have low bioavailability. Therefore, the risk to the public is not considered to be unreasonable.

Occupational Risk Characterisation

During use of chemicals in this group dermal, ocular and inhalation exposure of workers to these chemicals may occur, particularly where manual or open processes are used. These may include transfer and blending activities, quality control analysis, and cleaning and maintenance of equipment. Worker exposure to the chemicals at lower concentrations may also occur while using formulated products containing the chemicals. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical systemic long-term effect, systemic acute effect and local effect, these chemicals may pose an unreasonable risk to workers unless adequate control measures to minimise inhalation exposure to the chemicals are implemented. The chemicals should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine appropriate controls.

The data available support an amendment to the hazard classification in HSIS (refer to **Recommendation** section).

NICNAS Recommendation

Assessment of these chemical are considered to be sufficient, provided that the recommended amendment to the classification is adopted, and labelling and all other requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Regulatory Control

Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical hazards and environmental hazards.

The classification proposed below is based on read across principles (refer to section on **Grouping Rationale**). It should be used as a default for all members of the group. If empirical data become available for any member of the group indicating that a lower (or higher) classification is appropriate for the specific chemical, these may be used to amend the default classification for that chemical.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Very toxic by inhalation (T+; R26)	Fatal if inhaled - Cat. 2 (H330)
Sensitisation	May cause sensitisation by inhalation (Xn, R42)	May cause allergy or asthma symptoms or breathing difficulties if inhaled - Cat. 1 (H334)
Repeat Dose Toxicity	Toxic: danger of serious damage to health by prolonged exposure through inhalation (T; R48/23)	Causes damage to organs through prolonged or repeated exposure through inhalation - Cat. 1 (H372)
Reproductive and Developmental Toxicity	Repro. Cat 2 - May impair fertility (T; R60)	May damage fertility - Cat. 1B (H360F)

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for industry

Control measures

Control measures to minimise the risk from inhalation exposure to the chemicals should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- health monitoring for any worker who is at risk of exposure to the chemical if valid techniques are available to monitor the effect on the worker's health;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to assist with meeting obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((m)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (m)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals— Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemicals has not been undertaken as part of this assessment.

References

Agency for Toxic Substances and Disease Registry (ATSDR) 2004. Toxicological Profile for Cobalt. Accessed June 2014 at **http://www.atsdr.cdc.gov/toxprofiles/tp33.pdf**

Approved Criteria for Classifying Hazardous Substances [NOHSC: 1008(2004)] Third edition. Accessed at **http://www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents/258/ApprovedCriteria_Classifying_Hazardous_Substances_NOHSC1008-2004_PDF.pdf**

Clyne N, Hofman-Bang C, Haga Y, Hatori N, Marklund SL, Pehrsson SK and Wibom R. 2001. Chronic cobalt exposure affects antioxidants and ATP production in rat myocardium. Scandinavian Journal of Clinical and Laboratory Investigation 61 (8) pp 609-614.

Cobalt Development Institute 2014. In Vitro Bioaccessibility studies for Lithium Cobalt Dioxide (CAS# 12190-79-3). Unpublished report submitted to National Industrial Chemicals Notification and Assessment Scheme under the Inventory Multi-tiered Assessment and Prioritisation framework.

Cobalt Development Institute 2014. The In Vitro Percutaneous Absorption of Cobalt Through Human Skin. Testing facility: Charles River, Tranent Edinburgh. Testing Facility Study No. 785464, Report No. 30161. An unpublished report to the Cobalt Development Institute, Guildford, Surrey, United Kingdom. CDI/37.

Cotton F A& Wilkinson G 1988. The chemistry of the transition elements 18(F): Cobalt: Group VIII(9) in Advanced Inorganic Chemistry, 5th edition, Chapter 18, pp 725 - 741.

Elbetieha A, Al-Thani AS, Al-Thani RK, Darmani H& Owais W 2008. Effects of Chronic Exposure to Cobalt Chloride on the Fertility of Testes in Mice. Journal of Applied Biological Sciences 2(1) pp. 1-6.

Galleria Chemica. Accessed June 2014 at **https://jr.chemwatch.net/galleria/**

Globally Harmonised System of Classification and Labelling of Chemicals (GHS) United Nations, 2013. Fifth edition. Accesed at **http://www.unece.org/fileadmin/DAM/trans/danger/publi/ghs/ghs_rev05/English/ST-SG-AC10-30-Rev5e.pdf**

Government of Canada 2011. Cobalt (7440-48-4), Cobalt chloride (7646-79-9), Sulfuric acid, cobalt (2+) salt (1:1) (10124-43-3)& Sulfuric acid, cobalt salt (10393-49-4). Batch 10 Challenge Substances. Accessed June 2014 at **http://www.ec.gc.ca/ese-ees/8E18277B-457E-4073-8F27-**

EF5878648820/batch10_4substances%281%29_en.pdf

Haga Y, Clyne N, Hatori N, Hoffman-Bang C, Pehrsson SK and Ryden L 1996. Impaired myocardial function following chronic cobalt exposure in an isolated rate heart model. Trace Elements and Electrolytes 13 (2) pp 69-74.

International Agency for Research on Cancer (IARC) 2006. IARC monographs on the evaluation of carcinogenic risks to humans. Volume 86. Cobalt in Hard Metals and cobalt sulfate, gallium arsenide, idium phosphide and vanadium pentoxide. Accessed in March 2014 at **http://monographs.iarc.fr/ENG/Monographs/vol86/mono86.pdf**

Leggett RW 2008. The biokinetics of inorganic cobalt in the human body. Science of the Total Environment 389 pp. 259-269.

National Industrial Chemicals Notification and Assessment Scheme (NICNASa). Tier II Human health assessment for Soluble cobalt (II) and salts. Australian Government Department of Health. Accessed April 2014 at **http://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=952**

National Industrial Chemicals Notification and Assessment Scheme (NICNASb). Inventory Multi-Tiered and Prioritisation (IMAP): Human Health Tier II Assessment for Cobalt oxide. Available at **http://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments**

OECD 2014a. Guidance on Grouping of Chemicals, Second Edition. Environment Directorate. Joint meeting of the Chemicals Committee and the Working party on Chemicals, Pesticides and Biotechnology. Series on Testing& Assessment No. 194. Accessed June 2014 at **http://search.oecd.org/officialdocuments/displaydocumentpdf/?cote=env/jm/mono(2014)4&doclanguage=en**

OECD 2014b. SIDS Initial Assessment Profile (SIAP) on soluble cobalt salts. Unpublished.

REACH Dossier (REACHa). Cobalt molybdate (CAS No. 13762-14-6). Accessed June 2014 at **http://apps.echa.europa.eu/registered/data/dossiers/DISS-d6b2e148-690a-025d-e044-00144f67d031/AGGR-3e7b2f5f-0bdc-49fa-89e3-486dada2d87f_DISS-d6b2e148-690a-025d-e044-00144f67d031.html#AGGR-3e7b2f5f-0bdc-49fa-89e3-486dada2d87f**

REACH Dossier (REACHb). Cobalt lithium oxide (CAS No. 12190-79-3). Accessed June 2014 at **http://apps.echa.europa.eu/registered/data/dossiers/DISS-df93e076-ebd3-4f98-e044-00144f67d031/AGGR-a6218132-d1c9-45dc-b801-7cc9ee12720a_DISS-df93e076-ebd3-4f98-e044-00144f67d031.html#AGGR-a6218132-d1c9-45dc-b801-7cc9ee12720a**

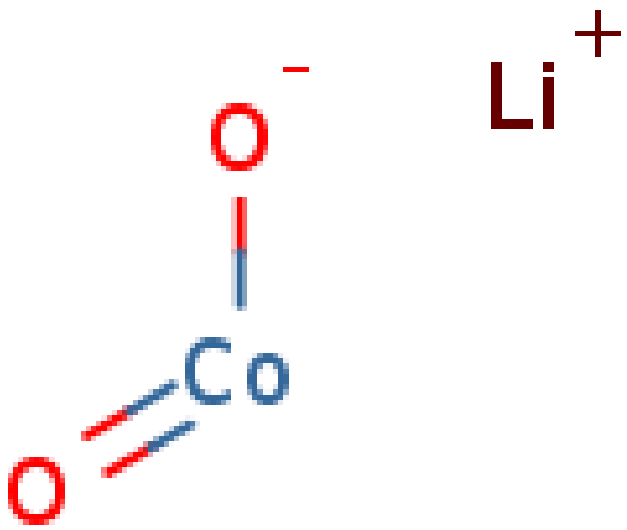
The Cobalt REACH Consortium Ltd. 2014. Chemical Safety Report for Lithium cobalt dioxide, CAS RN 12190-79-3 EC Number 235-362-0. March 2014. Unpublished report submitted to National Industrial Chemicals Notification and Assessment Scheme under the Inventory Multi-tiered Assessment and Prioritisation framework.

Zhu Y N, Zhang X H, Chen Y D, Zheng H H, Liu J& Wang X M (2013). Characterisation, dissolution and solubility of synthetic erythrite [Co3(AsO4)2.8H2O] and annabergite [Ni3(AsO4)2.8H2O] at 25°C. Canadian Metallurgical Quarterly, 52 (1): pp 7 -17.

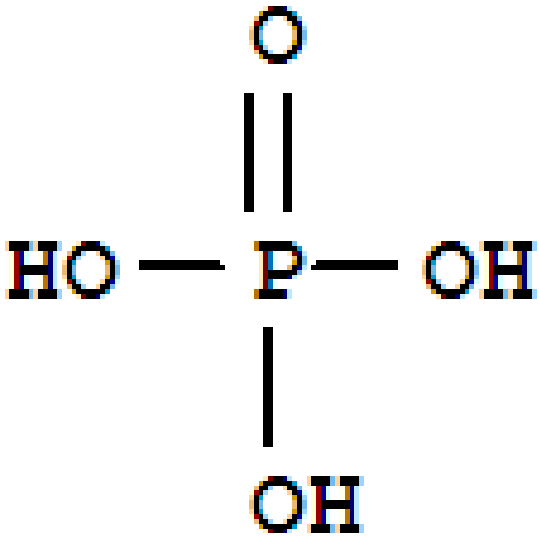
Last Update 27 November 2014

Chemical Identities

Chemical Name in the Inventory and Synonyms	**Cobaltate (CoO21-), lithium** Lithium colbaltite Cobalt lithium oxide Cobalt lithium dioxide Lithium cobalt III oxide
CAS Number	12190-79-3
Structural Formula	

	
Molecular Formula	CoO2.Li
Molecular Weight	97.872

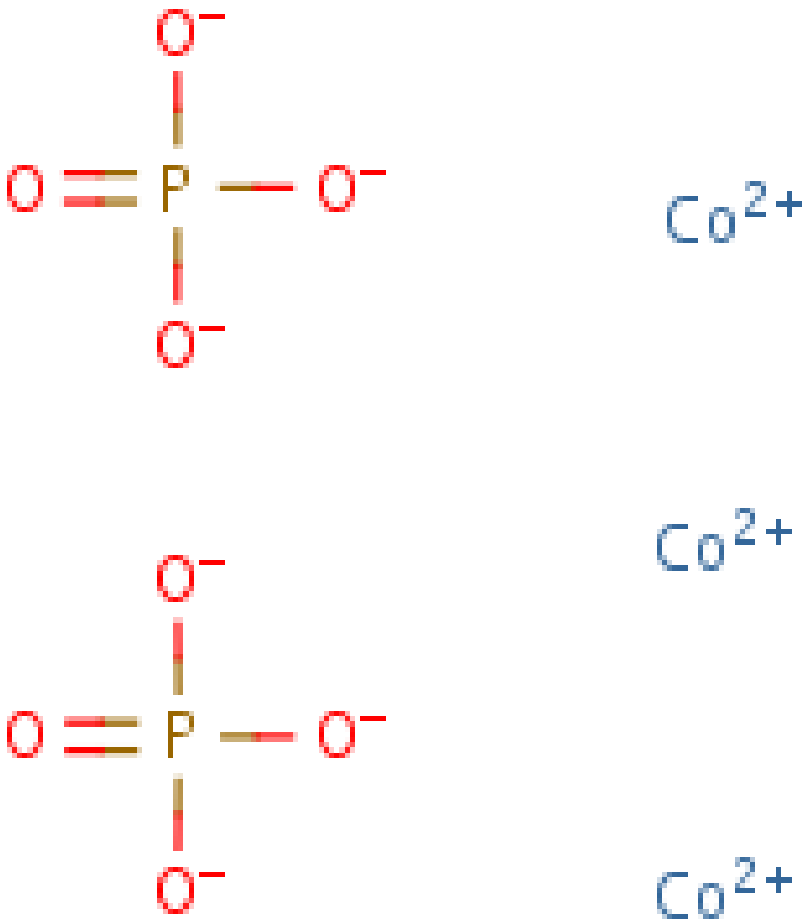
Chemical Name in the Inventory and Synonyms	**Phosphoric acid, cobalt(2+) salt (2:3), hydrate** C.I. 77360 Pigment violet 14
CAS Number	10101-56-1
Structural Formula	



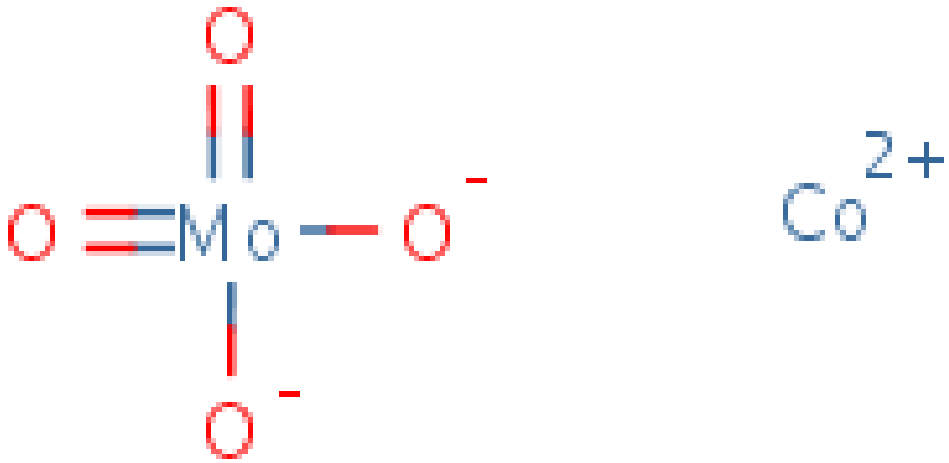
- 3/2 Co(II)

- x H_2O

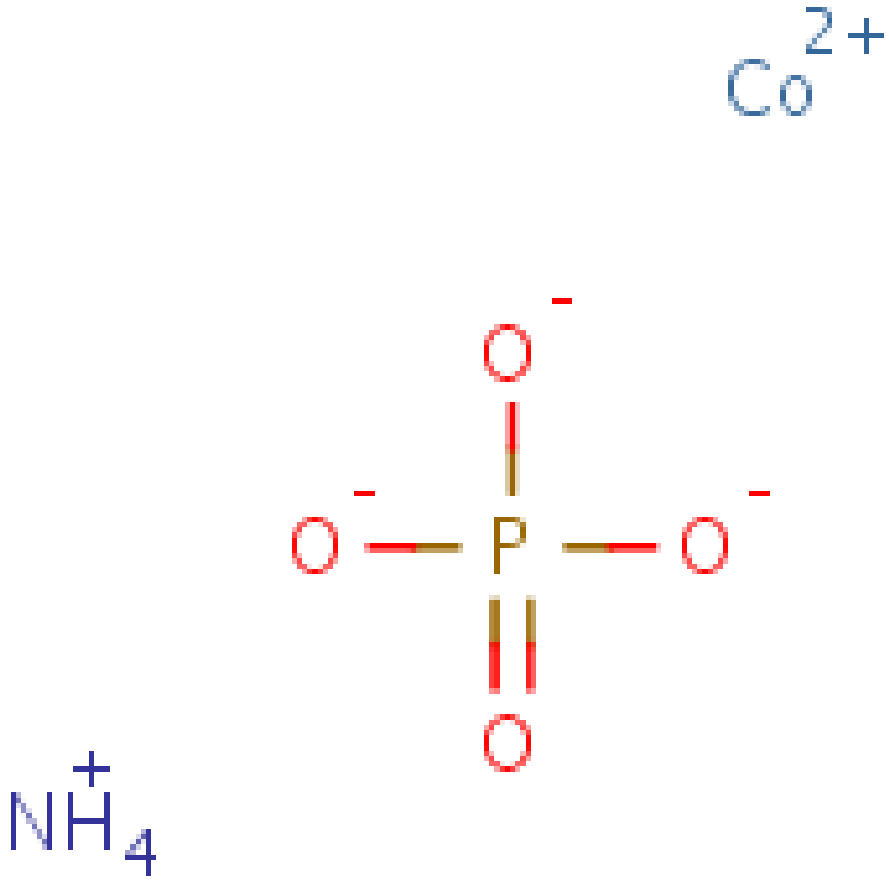
Molecular Formula	Co.2/3H3O4P.xH2O
Molecular Weight	

Chemical Name in the Inventory and Synonyms	**Phosphoric acid, cobalt(2+) salt (2:3)** Cobalt phosphate (Co3(PO4)2) Tricobalt diphosphate Cobalt(II) phosphate Tricobalt bis(orthophosphate) Cobaltous phosphate
CAS Number	13455-36-2
Structural Formula	
Molecular Formula	Co.2/3H3O4P
Molecular Weight	366.74

Chemical Name in the Inventory and Synonyms	**Cobalt molybdenum oxide (CoMoO4)** Molybdic acid (H2MoO4), cobalt(2+) salt (1:1) Cobalt (II) molybdenum (IV) oxide cobalt molybdate Cobaltous molybdate
CAS Number	13762-14-6
Structural Formula	

	
Molecular Formula	Co.Mo.O
Molecular Weight	218.87

Chemical Name in the Inventory and Synonyms	**Phosphoric acid, ammonium cobalt(2+) salt (1:1:1)** Cobalt ammonium phosphate Ammonium cobaltous phosphate Ammonium cobalt phosphate Cobalt Violet Brilliant Light
CAS Number	14590-13-7
Structural Formula	

	
Molecular Formula	Co.H3N.H3O4P
Molecular Weight	171.942

Share this page