IARC MONOGRAPHS

COBALT, ANTIMONY COMPOUNDS, AND WEAPONS-GRADE TUNGSTEN ALLOY

VOLUME 131

This publication represents the views and expert opinions of an IARC Working Group on the Identification of Carcinogenic Hazards to Humans, which met remotely, 2–18 March 2022

LYON, FRANCE - 2023

IARC MONOGRAPHS ON THE IDENTIFICATION OF CARCINOGENIC HAZARDS TO HUMANS

International Agency for Research on Cancer

World Health Organization

Table S1.19 Exposure assessment review and critique for mechanistic studies in humans exposed to cobalt

Reference and mechanistic end-point	What was the study design?	Relevant form(s) of cobalt in exposed population[a]	What methods were used for the exposure assessment (including data source, environmental and biological measurements etc.)?	What was the exposure definition?	Was exposure assessment qualitative, semiquantitative, or quantitative?	Concerns noted on sampling and collection protocols for metals measurement	What routes of exposure were assessed?	What exposure metrics were derived for use in analyses (e.g. average exposure, exposure duration, cumulative exposure etc.)?	What was the timing of exposure relative to the outcome?	Was there potential for co-exposures to other metals/carcinogens? If yes, were these accounted for in analyses?	Was there potential for differential or non-differential exposure misclassification?
GENERAL POPULATION STUDIES											
Arslan et al. (2011)	Case–control	Not intended to be specified (general population study)	Biological samples	Exposure to a series of metals in blood, including cobalt, was assessed among patients with malignant glial tumours	Quantitative	The use of a single biological sample may not have captured an appropriate exposure window for the outcome under study	All routes	Average cobalt concentrations in plasma (µg/dL)	The exposure was assessed after the outcome	Several other metals were assessed and considered individually in analyses	Differential misclassification: unlikely Non-differential misclassification: potentially
Bibi et al. (2016) Blood antioxidant response	Cross-sectional	Not intended to be specified (general population study)	Geographical location Biological samples	Cobalt exposure was assessed among 48 individuals from four areas of Pakistan based on measurements in urine, blood, nails and hair; geographical location, categorized as low/medium/high risk, the latter categorization was not specific to cobalt	Semiquantitative (low/medium/high) Quantitative (biological measurements)	No	All routes	Risk of exposure was assigned as low, medium, or high risk (geographical location) Average concentrations of cobalt in urine and blood (units not reported)	Exposure information was collected at the same time as the outcome was measured	Exposure to a variety of metals was assessed, but these were considered separately in the analysis	Non-differential misclassification: potentially, in the geographical location of the study participants and due to the reliance on a single biological sample
Calderón-Garcidueñas et al. (2013) DNA repair and inflammatory markers in brain tissue	Cross-sectional	Not intended to be specified (general population study)	Biological samples	Metals, including cobalt, were assessed in the frontal cortex and lungs of 59 decedents who had died in high- or low-air pollution cities	Quantitative	None	All routes	Average metal concentrations in tissue (µg/g dry tissue)	Exposure information was collected at autopsy, the same time as the outcome was measured; it is unclear whether the exposure timing would have captured the relevant window of exposure for the outcomes assessed	Exposure to a variety of metals was assessed, but metals were considered individually	Differential misclassification: unlikely
Howe et al. (2021)	Cross-sectional analysis within a prospective cohort study	Not intended to be specified (general population study)	Biological samples	9 metals, including cobalt, were quantified in the urine of pregnant women (at first trimester of pregnancy) using ICP-MS; only the 3-month samples appear to be included in the reported analyses	Quantitative	The use of a spot urine sample may not have captured an appropriate exposure window for the outcome under study; urine is not an ideal sample type for all metals	All routes	Urinary cobalt (cobalt, µg/L)	Exposure and outcomes were assessed at the same time, but in additional analysis exposure was assessed before the outcome	9 metals were assessed and considered separately in analyses; multiple metal associations were tested using Bayesian Kernel Machine Regression	Differential misclassification: unlikely Non-differential misclassification: potentially
Johnstone et al. (2014)	Nested case–control	Not intended to be specified (general population study)	Biological samples	Urinary cobalt was assessed (along with several other metals and trace elements) among 473 women participating in the Endometriosis Natural	Quantitative	No	All routes	Mean cobalt concentrations in urine (µg/g creatinine)	Exposure was assessed before the outcome	Yes, there was potential for exposure to other metals and trace elements, many of which were measured in this study but not accounted for in the statistical analysis (adjusted models)	Differential misclassification: unlikely Non-differential misclassification: likely, due to the use of

Table S1.19 Exposure assessment review and critique for mechanistic studies in humans exposed to cobalt

Reference and mechanistic end-point	What was the study design?	Relevant form(s) of cobalt in exposed population[a]	What methods were used for the exposure assessment (including data source, environmental and biological measurements etc.)?	What was the exposure definition?	Was exposure assessment qualitative, semiquantitative, or quantitative?	Concerns noted on sampling and collection protocols for metals measurement	What routes of exposure were assessed?	What exposure metrics were derived for use in analyses (e.g. average exposure, exposure duration, cumulative exposure etc.)?	What was the timing of exposure relative to the outcome?	Was there potential for co-exposures to other metals/carcinogens? If yes, were these accounted for in analyses?	Was there potential for differential or non-differential exposure misclassification?
				History, Diagnosis and Outcomes (ENDO) Study							spot urine samples collected at baseline
Li et al. (2021b) Oxidative stress markers	Cross-sectional	Not intended to be specified (general population study)	Biological samples (blood)	Cobalt (and other metals) in blood were assessed among people living near a former electronic waste recycling site (*n* = 69) and a reference area (*n* = 53)	Quantitative	No	All routes	Cobalt concentration in whole blood of exposed and reference group (ng/mL)	Exposure information was collected at the same time as the outcome was measured	Other heavy metals (nickel, mercury, tin, lead, cadmium, copper, zinc) were also quantified and were examined individually in analyses	Differential misclassification: unlikely Non-differential misclassification: potentially
Scharf et al. (2014)	Cross-sectional	Cobalt metal	Biological samples	Surgical tissues from 18 patients undergoing revision surgery of medical implants (9 metal-on-metal implants, cobalt and chromium; 9 comparison implants) were examined	Qualitative	No	Implantation (medical device)	Cobalt ion concentrations in periprosthetic tissues surrounding control and metal-on-metal implants	Exposure occurred before the outcome measurement	It is possible that the implants contained metals other than cobalt; chromium was considered extensively in the study	N/A due to study design
Xue et al. (2021) Blood oxidative stress and inflammation markers	Cross-sectional	Not specified	Biological measures at a single point in time	Cobalt (and other metals) in plasma were assessed among people living near a former electronic waste recycling area (*n* = 62) and a reference location (*n* = 47)	Quantitative	No	All routes	Mean/median cobalt concentration in plasma (ng/mL)	Biological measures were assessed at the same time as the outcome was measured	Exposure to other metals is likely in this setting; other metals in biological samples were quantified	Differential misclassification: unlikely Non-differential misclassification: potentially; as the biological samples were collected only at one point in time and this may not reflect exposure in a time window relevant to the outcome
EXPERIMENTAL STUDIES											
Amirtharaj et al. (2008) Cobalt binding to serum albumin	Experimental	Cobalt salts	Laboratory controlled exposure	0.232 mM cobalt chloride was applied to serum samples to assess cobalt binding in this experimental study	Quantitative	No	N/A as cobalt was applied directly, and in a controlled manner, to blood (serum) samples	N/A due to study design	The exposure was applied to the experimental samples before the measurement of the outcome	No	N/A due to study design

Table S1.19 Exposure assessment review and critique for mechanistic studies in humans exposed to cobalt

Reference and mechanistic end-point	**What was the study design?**	**Relevant form(s) of cobalt in exposed population[a]**	**What methods were used for the exposure assessment (including data source, environmental and biological measurements etc.)?**	**What was the exposure definition?**	**Was exposure assessment qualitative, semiquantitative, or quantitative?**	**Concerns noted on sampling and collection protocols for metals measurement**	**What routes of exposure were assessed?**	**What exposure metrics were derived for use in analyses (e.g. average exposure, exposure duration, cumulative exposure etc.)?**	**What was the timing of exposure relative to the outcome?**	**Was there potential for co-exposures to other metals/carcinogens? If yes, were these accounted for in analyses?**	**Was there potential for differential or non-differential exposure misclassification?**
Katsarou et al. (1997)	Experimental	Cobalt salts	Laboratory controlled exposure	From 180 men who were cement workers with positive patch tests for chromium or cobalt salts who returned for a subsequent patch test, T-cells from 20patients were studied; 10 patients with consistently positive patch test results and 10 patients who had previously had a positive patch test and now had an negative patch test	Insufficient detail to assess	The amount of cobalt applied (concentration, volume) to the T-cells is not reported	N/A due to study design	The concentration and amount of cobalt the cells were exposed to is not reported	Exposure occurred before the outcome was measured	No	N/A due to study design
L'vova et al. (1990)	Experimental	Cobalt salts	Laboratory controlled exposure	An experimental study was carried out with human cell cultures; cobalt chloride was applied to cell culture as a mutagen	Quantitative	The concentration of cobalt chloride administered is reported, but the volume is not	N/A as cobalt chloride was applied directly, and in a controlled manner	N/A due to study design	The exposure was applied to the experimental samples before the measurement of the outcome	No	N/A due to study design
INDUSTRY-BASED STUDIES											
Andersson et al. (2021)	Cross-sectional	Cobalt metal	Air samples Biological samples Historical data	72 workers from 2 Swedish hard-metal companies participated in a study of cobalt exposure	Quantitative	None for cobalt Results for tungsten air concentrations are reported, but the source of these values is unclear	Inhalation (air samples) All routes (biological samples)	Average cobalt concentrations in air (mg/m^3) Personal samples: inhalable fraction Stationary samples: inhalable, total and respirable fractions Cumulative cobalt concentration in air (mg/m^3-years) Average cobalt concentration in blood (nmol/L) Average cobalt concentration in urine (nmol/L) Exposure variables were split into tertiles for analysis	Exposure was assessed on the same day that biological samples were collected	All job groups included had detectable tungsten exposure; the methods are not detailed nor are these results considered in analysis	Differential misclassification: unlikely
Bencko et al. (1983)	Cross-sectional	Not specified	Occupational history	35 workers "occupationally exposed to cobalt" were studied in comparison to 38 nickel-exposed workers and 42 controls	Qualitative (yes/no)	Insufficient detail to assess Metal was not measured	All routes (indirectly)	Occupational exposure to cobalt (yes/no)	Exposure information reported suggested workers had a mean duration of	Insufficient detail to assess	Insufficient detail to assess

Table S1.19 Exposure assessment review and critique for mechanistic studies in humans exposed to cobalt

Reference and mechanistic end-point	What was the study design?	Relevant form(s) of cobalt in exposed population[a]	What methods were used for the exposure assessment (including data source, environmental and biological measurements etc.)?	What was the exposure definition?	Was exposure assessment qualitative, semiquantitative, or quantitative?	Concerns noted on sampling and collection protocols for metals measurement	What routes of exposure were assessed?	What exposure metrics were derived for use in analyses (e.g. average exposure, exposure duration, cumulative exposure etc.)?	What was the timing of exposure relative to the outcome?	Was there potential for co-exposures to other metals/carcinogens? If yes, were these accounted for in analyses?	Was there potential for differential or non-differential exposure misclassification?
Serum immunoglobulin concentrations									employment ~10 years and thus exposure did occur before the assessment of outcomes		
Bencko et al. (1986a) Serum immunoglobulin concentrations	Cross-sectional	Not specified	Occupational history and biological samples	35 workers "occupationally exposed to cobalt" were studied in comparison to 38 nickel-exposed workers and 42 controls	Quantitative	Insufficient detail to assess	All routes (indirectly)	Average cobalt concentrations in hair (µg/g[1])	Exposure information reported in Bencko et al. (1983) suggests workers had a mean duration of employment ~10 years and thus exposure did occur before the assessment of outcomes	Exposure to nickel was measured in biological samples among in the group with occupational exposure to cobalt, but it was not accounted for in the analysis	Insufficient detail to assess
De Boeck et al. (2000)	Cross-sectional	Not specified [inferred by the Working Group to possibly include cobalt metal and cobalt-bearing tungsten carbide, as well as particles containing other metals]	Biological samples	35 male workers with exposure to cobalt dust were compared with 29 workers with tungsten carbide exposure and 35 matched controls with neither exposure	Quantitative	No	All routes	Cobalt in urine (µg/g creatinine)	Exposure was assessed at the end of the work week; the outcomes were assessed 3 days later, following a weekend off work	Yes, it seems likely that exposure to other metals is possible; however, a specific group of workers only exposed to cobalt dust was considered; no other metals were reported in the analysis; smoking and alcohol consumption were considered in the analyses	Differential misclassification: unlikely Non-differential misclassification: potentially
Gennart et al. (1993)	Cross-sectional	Cobalt-bearing metal [Authors note that exposure also to metal oxides cannot be excluded]	Biological samples Questionnaires Air samples	Cobalt in urine was assessed among 24 male workers in a metal powder production factory and compared with 23 clerical workers; mechanistic end-points were assessed between the two groups (exposed/unexposed) and by duration of exposure	Qualitative Quantitative	Methods for determining metals in spot urine samples are not described; the timing of spot urine samples is also not described	All routes	Exposed/unexposed Years of exposure (0 years; < 5 years, ≥ 5 years) Mean cobalt concentrations in urine (µg/g creatinine)	Exposure in biological samples was assessed at the same time as the outcome	Yes, the exposed workers had exposure to chromium, iron, and nickel in additional to cobalt, as demonstrated by the air sampling results; air levels of cobalt, chromium, iron, and nickel were reported but not analysed in relation to mechanistic end-points	Differential misclassification: unlikely Non-differential misclassification: likely; because of the use of spot urine samples
Hengstler et al. (2003) DNA strand breaks and repair	Cross-sectional	Not specified	Air samples (from breathing zone) Biological samples (urine)	Cobalt in air and urine was assessed among 78 workers from 10 facilities engaged in either the production of cadmium-containing pigment or batteries, or the recycling of electric tools	Quantitative	The exposure windows captured by the air and biological samples were not aligned; urine was collected at the end of the work shift, during which the air levels were measured and likely reflect earlier time periods of exposure	Inhalation (air samples) All routes (biological samples)	Cobalt concentration in air (µg/m^3) Cobalt concentration in urine (µg/L) Cobalt concentration in urine normalized to creatinine (µg/g creatinine)	Exposure information was collected at the same time as the outcome was measured	Cadmium and lead were also assessed quantitatively (cadmium: air, blood, urine; lead: air, blood); these metals were accounted for in the analysis Smoking, alcohol, ionizing radiation, and other carcinogens were also considered	Differential misclassification: unlikely

Table S1.19 Exposure assessment review and critique for mechanistic studies in humans exposed to cobalt

Reference and mechanistic end-point	What was the study design?	Relevant form(s) of cobalt in exposed population[a]	What methods were used for the exposure assessment (including data source, environmental and biological measurements etc.)?	What was the exposure definition?	Was exposure assessment qualitative, semiquantitative, or quantitative?	Concerns noted on sampling and collection protocols for metals measurement	What routes of exposure were assessed?	What exposure metrics were derived for use in analyses (e.g. average exposure, exposure duration, cumulative exposure etc.)?	What was the timing of exposure relative to the outcome?	Was there potential for co-exposures to other metals/carcinogens? If yes, were these accounted for in analyses?	Was there potential for differential or non-differential exposure misclassification?
Mateuca et al. (2005)	Cross-sectional	Cobalt metal	Occupational history	Participants drawn from the population of De Boeck et al. (2000) 21 men who were refinery workers with exposure to cobalt dust were compared with 26 refinery workers with tungsten carbide (hard-metal) exposure and 26- matched controls with neither exposure, but employed at the same plants	Qualitative	No	All routes (indirectly)	Exposed/unexposed (“exposure”) Three exposure groups: cobalt-exposed; tungsten carbide-exposed; unexposed (also referred to as “type of plant”)	Exposure was determined before the outcome	Yes, there is potential for exposure to other metals; however, a specific group of workers only exposed to cobalt dust was considered; the study participants were drawn from workers at several European refineries; no other exposures were considered in the analysis	Differential misclassification: unlikely Non-differential misclassification: potentially
Princivalle et al. (2017) Haemoglobin adducts	Repeated cross-sectional (exposure only)	Cobalt metal Cobalt oxides	Biological measures (urine and blood) Air samples	Cobalt was prospectively assessed in urine and blood among 34 workers at a hard-metal manufacturing plant	Quantitative	No	All routes	Average cobalt concentrations in urine (μg/L and μg/g creatinine) Cobalt in plasma (μg/L) Cobalt levels in whole blood (μg/L) Cobalt in air (mg/m^3)	Exposure and outcome were assessed at the same time	Yes, there was potential for other exposures including tungsten carbide and these were not accounted for in analyses, though the outcomes in this study are specific to cobalt	N/A due to study design
Shirakawa & Morimoto (1997) Immunoglobulin E antibodies against cobalt-conjugated serum albumin	Cross-sectional	Cobalt metal	Occupational groups (exposed/unexposed)	Hard-metal exposure was assessed qualitatively (yes/no) among hard-metal plant workers; this exposure definition is not specific to cobalt; included workers were engaged in the production of hard metals, a process that included other metals (e.g. tungsten, nickel, and molybdenum, as described by Kusaka et al.; 1986)	Qualitative	It is unclear how the exposure groups were constructed and the temporality of these decisions in related to the outcome measures	All routes (indirectly)	Exposed and unexposed groups There are multiple exposure variables listed in the multiple regression models, some reported with quantitative units (mg/m^3 – exposure concentrations; exposure doses) but neither is described in the methods	The timing of the construction of the exposure groups in relation to the assessment of the outcome measures is unclear, though it seems to be a prospective study	Yes, exposure to tungsten carbide is likely; smoking status was investigated explicitly in the analyses; earlier publications seemingly from the same hard-metal plant report tungsten, titanium, cobalt, and nickel in lung biopsy specimens of workers (Kusaka et al., 1986), and 96% of total dust was assumed to be tungsten carbide (Kusaka et al., 1992)	Differential misclassification: unlikely Non-differential misclassification: potentially
Swennen et al. (1993) Thyroid metabolism markers, white blood cell count, erythropoietic markers	Cross-sectional	Cobalt metal Cobalt and oxides possible	Several data sources were reported including: Location of employment Biological measures (blood and urine) Air samples (breathing zone)	Employment in a cobalt refinery (yes/no) was the method used in examination of the mechanistic end-points among 164 workers (82 exposed, 82 not exposed); the exposure definition is not specific to cobalt, but there is no mention of other metals; little information on the comparison group is provided	Associations with mechanistic end-points are limited to qualitative exposure assessment methods (exposed/unexposed based on location of employment)	No	Employment location: all routes (indirectly) for all participants	Exposed (yes/no)	Exposure (employment) occurred before the measurement of the mechanistic end-points	The study population was focused on a cobalt refinery reducing the potential for other metal exposures among the exposed group Smoking was considered and did not differ between the exposed and unexposed groups	Differential misclassification: unlikely Non-differential misclassification: low potential in the qualitative location of employment approach

Table S1.19 Exposure assessment review and critique for mechanistic studies in humans exposed to cobalt

Reference and mechanistic end-point	What was the study design?	Relevant form(s) of cobalt in exposed population[a]	What methods were used for the exposure assessment (including data source, environmental and biological measurements etc.)?	What was the exposure definition?	Was exposure assessment qualitative, semiquantitative, or quantitative?	Concerns noted on sampling and collection protocols for metals measurement	What routes of exposure were assessed?	What exposure metrics were derived for use in analyses (e.g. average exposure, exposure duration, cumulative exposure etc.)?	What was the timing of exposure relative to the outcome?	Was there potential for co-exposures to other metals/carcinogens? If yes, were these accounted for in analyses?	Was there potential for differential or non-differential exposure misclassification?
Walters et al. (2012)	Cross-sectional	Cobalt metal	Occupational history Biological samples	Following 5 cases of occupational asthma in the workplace, 62 workers from an aerospace manufacturing company were studied in a cross-sectional study; biological samples were specific to cobalt, however qualitative exposure groups were not specific to cobalt	Qualitative and quantitative	The use of spot urine samples may not have captured an appropriate exposure window for the outcome under study	All routes	Qualitative: working in areas with medium or higher exposure to metalworking fluid; working in areas with low or no exposure to metalworking fluid Quantitative: average urinary cobalt concentrations (μg/L)	Exposure was measured at the same time as the outcomes were measured	Yes, the workers were employed in aerospace manufacturing where alloys including nickel, chromium, light steel, and titanium were in use; there was additionally potential for exposure to tungsten as tungsten carbide-tipped tools were in use; urinary chromium was assessed but considered separately	Differential misclassification: unlikely; the use of spot urine samples is likely to introduce non-differential exposure misclassification
Wultsch et al. (2017)	Cross-sectional, with comparison group	Not specified, likely cobalt salts	Biological samples, air samples Questionnaires	42 workers from a bright electroplating factory were compared with 43 participants recruited from jail wardens	Quantitative	The use of a single biological sample may not have captured an appropriate exposure window for the outcome under study	All routes	Cobalt concentrations in blood were reported as μg/L plasma Cobalt concentrations in ambient air (mg/m^3) Duration of exposure (years) was constructed (None; < 5 years; 5 to < 10 years; > 10 years)	The exposure and outcome were assessed at the same time	Yes, the workers in the electroplating plant had potential exposure to other metals including chromium and nickel; chromium was assessed in blood (and air) but considered separately	Differential misclassification: unlikely Non-differential misclassification: potentially
CASE STUDIES/SERIES											
Krakowiak et al. (2005)	Case study (n = 1) with specific challenge testing	Cobalt salts in challenge testing	Occupational history Nasal provocation Patch testing	A case of occupational asthma is described in a 35-year-old man with approximately 10 years of work experience in the hard-metal industry as a diamond polishing disc former; as part of the clinical workup, controlled exposure to cobalt was administered to the patient during patch testing and nasal provocation tests	Quantitative	No	Patch testing: skin Nasal provocation testing: inhalation	Cobalt chloride was applied to the skin in varying concentrations (0.01%, 0.1% and 1%) diluted in phosphate buffer solution Cobalt chloride was delivered at 0.05% solution in phosphate-buffered saline through the nasal route	The exposure was quantified before the outcome	The case described was a hard-metal worker and likely had other exposures in the workplace (e.g. tungsten carbide), but the controlled exposures described in the case report were specific to cobalt	No, exposure was controlled as part of the patch testing and nasal provocation testing
Nemery et al. (1990) Inflammatory cells: single case study	Case study (n = 1)	Cobalt metal	Occupational history Biological measures	The exposure history is described for a diamond polisher who had a history of using polishing discs that contained cobalt Cobalt was assessed in lung tissue (mass) and through use of transmission electron microscopy	Qualitative and quantitative measures of exposure are reported for the case	No	Inhalation	Cobalt concentration in lung tissue (μg/g wet lung)	≥ 10 years of cobalt exposure preceded the diagnostic process	Yes, iron, nickel, and chromium were also measured and reported; 5 cigarettes/week	N/A due to study design

Table S1.19 Exposure assessment review and critique for mechanistic studies in humans exposed to cobalt

Reference and mechanistic end-point	What was the study design?	Relevant form(s) of cobalt in exposed population[a]	What methods were used for the exposure assessment (including data source, environmental and biological measurements etc.)?	What was the exposure definition?	Was exposure assessment qualitative, semiquantitative, or quantitative?	Concerns noted on sampling and collection protocols for metals measurement	What routes of exposure were assessed?	What exposure metrics were derived for use in analyses (e.g. average exposure, exposure duration, cumulative exposure etc.)?	What was the timing of exposure relative to the outcome?	Was there potential for co-exposures to other metals/carcinogens? If yes, were these accounted for in analyses?	Was there potential for differential or non-differential exposure misclassification?
Rizzato et al. (1994)	Case series (*n* = 3)	Not specified	Biological samples	Cobalt levels in various biological specimens (e.g. blood, urine, pubic hair, nails, sperm) were measured using neutron activation analysis for 4 patients with an occupational history of exposure to cobalt and chest X-rays suggestive for sarcoidosis	Quantitative	No	All routes	Concentrations in biological samples were reported as the ratio between the concentration of elements in tissue in each patient sample relative to a group of controls; results reported in Rizzato et al. (1992)	The exposure was assessed after the outcome	The 3 cases all had potential exposure to other metals at work as they were employed in the hard-metal industry; all of cobalt, tungsten, and tantalum were quantified in the samples and reported separately	N/A due to study design
Tilakaratne & Sidhu (2015)	Case study (*n* = 2)	Cobalt salts in challenge testing (as per Australian patch testing baseline series)	Occupational history	The work history is described for 2 workers with a history of work in home renovations; 1 case had a doubtful (±) patch test reaction to cobalt and the other with a strong positive (+++) reaction to cobalt	Qualitative description of work is reported for the cases	No	All routes (indirectly)	None, descriptive	Information on exposure was collected at the same time as the outcomes were measured	Yes, exposure to chromate and nickel is likely in both cases; this exposure was discussed but the nature of the case study meant that there was no exposure–response relationship reported	N/A due to study design

ICP-MS, inductively coupled plasma mass spectrometry; N/A, not applicable.

[a] Includes forms of cobalt explicitly described within the study; may not comprehensively describe all cobalt forms present

References

ACGIH (2019). Cobalt and inorganic compounds. TLVs and BEIs based on the documentation of the threshold limit values for chemical substances and physical agents & biological exposure indices. Cincinnati (OH), USA: American Conference of Governmental Industrial Hygienists. Available from: https://www.acgih.org.

Amirtharaj GJ, Natarajan SK, Mukhopadhya A, Zachariah UG, Hegde SK, Kurian G, et al. (2008). Fatty acids influence binding of cobalt to serum albumin in patients with fatty liver. Biochim Biophys Acta. 1782(5):349–54. https://doi.org/10.1016/j.bbadis.2008.02.006 PMID:18346470

Andersson L, Hedbrant A, Persson A, Bryngelsson IL, Sjögren B, Stockfelt L, et al. (2021). Inflammatory and coagulatory markers and exposure to different size fractions of particle mass, number and surface area air concentrations in the Swedish hard metal industry, in particular to cobalt. Biomarkers. 26(6):557–69. https://doi.org/10.1080/1354750X.2021.1941260 PMID:34128444

Arslan M, Demir H, Arslan H, Gokalp AS, Demir C (2011). Trace elements, heavy metals and other biochemical parameters in malignant glioma patients. Asian Pac J Cancer Prev. 12(2):447–51. PMID:21545211

Bai Y, Wang G, Fu W, Lu Y, Wei W, Chen W, et al. (2019). Circulating essential metals and lung cancer: risk assessment and potential molecular effects. Environ Int. 127:685–93. https://doi.org/10.1016/j.envint.2019.04.021 PMID:30991224

Basketter DA, Angelini G, Ingber A, Kern PS, Menné T (2003). Nickel, chromium and cobalt in consumer products: revisiting safe levels in the new millennium. Contact Dermatitis. 49(1):1–7. https://doi.org/10.1111/j.0105-1873.2003.00149.x PMID:14641113

Bencko V, Wagner V, Wagnerová M, Reichrtová E (1983). Immuno-biochemical findings in groups of individuals occupationally and non-occupationally exposed to emissions containing nickel and cobalt. J Hyg Epidemiol Microbiol Immunol. 27(4):387–94. PMID:6663071

Bencko V, Wagner V, Wagnerová M, Zavázal V (1986a). Human exposure to nickel and cobalt: biological monitoring and immunobiochemical response. Environ Res. 40(2):399–410. https://doi.org/10.1016/S0013-9351(86)80115-3 PMID:3732211

Bibi M, Hashmi MZ, Malik RN (2016). The level and distribution of heavy metals and changes in oxidative stress indices in humans from Lahore district, Pakistan. Hum Exp Toxicol. 35(1):78–90. https://doi.org/10.1177/0960327115578063 PMID:25791319

Calderón-Garcidueñas L, Serrano-Sierra A, Torres-Jardón R, Zhu H, Yuan Y, Smith D, et al. (2013). The impact of environmental metals in young urbanites' brains. Exp Toxicol Pathol. 65(5):503–11. https://doi.org/10.1016/j.etp.2012.02.006 PMID:22436577

Cuckle H, Doll R, Morgan LG (1980). Mortality study of men working with soluble nickel compounds. In: Brown SS, Sunderman FW Jr, editors. Nickel toxicology. London, UK: Academic Press; pp. 11–4.

De Boeck M, Lardau S, Buchet JP, Kirsch-Volders M, Lison D (2000). Absence of significant genotoxicity in lymphocytes and urine from workers exposed to moderate levels of cobalt-containing dust: a cross-sectional study. Environ Mol Mutagen. 36(2):151–60. https://doi.org/10.1002/1098-2280(2000)36:2<151::AID-EM10>3.0.CO;2-V PMID:11013414

Duan W, Xu C, Liu Q, Xu J, Weng Z, Zhang X, et al. (2020). Levels of a mixture of heavy metals in blood and urine and all-cause, cardiovascular disease and cancer mortality: a population-based cohort study. Environ Pollut. 263(Pt A):114630. https ://doi.org/10.1016/j.envpol.2020.114630 PMID :33618481

Dufresne A, Loosereewanich P, Armstrong B, Thériault G, Bégin R (1996). Inorganic particles in the lungs of five aluminum smelter workers with pleuro-pulmonary cancer. Am Ind Hyg Assoc J. 57(4):370–5. https://doi.org/10.1080/15428119691014918 PMID:8901239

Enterline PE, Marsh GM, Esmen NA, Henderson V, Ricci E (1986). Mortality among copper and zinc smelter workers in the United States. Technical Report submitted to the Smelter Environmental Research Association, January 1986.

Enterline PE, Marsh GM, Esmen NA, Henderson VL, Callahan CM, Paik M (1987). Some effects of cigarette smoking, arsenic, and SO_2 on mortality among US copper smelter workers. J Occup Med. 29(10):831–8. PMID:3681494

Environment Agency (2022). Derivation and use of soil screening values for assessing ecological risks. Report – ShARE id26 (revised). Bristol, UK: Environment Agency. Available from: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1047897/Soil_screening_values_for_assessing_ecological_risk_-_report.pdf, accessed 2 March 2022.

European Commission (2009a). Annex 3 to the Commission Staff Working Document accompanying the Report from the Commission in accordance with Article 3.7 of the Groundwater Directive 2006/118/EC on the establishment of groundwater threshold values. Information on the groundwater threshold values of the Member States. Brussels, Belgium: European Commission. Available from: https://ec.europa.eu/environment/water/water-framework/groundwater/pdf/com_swd_annex_iii.pdf, accessed 1 February 2022.

European Commission (2009b). Directive 2009/48/EC of the European Parliament and of the Council of 18 June 2009 on the safety of toys. Brussels, Belgium: European Commission. Available from: https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:02009L0048-20210521&from=EN, accessed 14 March 2022.

European Commission (2010). Directive 2010/75/EU of the European Parliament and of the Council of 24 November 2010 on industrial emissions (integrated pollution prevention and control). Recast. Brussels, Belgium: European Commission. Available from: https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:02010L0075-20110106&from=DE, accessed 15 March 2022.

Fréry N, Saoudi A, Garnier R, Zeghnoun A, Falq G (2011). Exposition de la population française aux substances chimiques de l'environnement. Tome 1: Présentation générale de l'étude. Métaux et métalloïdes. Saint-Maurice, France: Institut de Veille Sanitaire. Available from: https://www.santepubliquefrance.fr/determinants-de-sante/exposition-a-des-substances-chimiques/pesticides/documents/rapport-synthese/exposition-de-la-population-francaise-aux-substances-chimiques-de-l-environnement.-tome-1.-presentation-generale-de-l-etude.-metaux-et-metalloides, accessed 1 February 2022. [French]

FSA (2003). Safe upper levels for vitamins and minerals. Expert Group on Vitamins and Minerals, May 2003. London, UK: Food Standards Agency. https://webarchive.nationalarchives.gov.uk/ukgwa/20121105225356/http://www.food.gov.uk/multimedia/pdfs/vitmin2003.pdf, accessed 30 September 2022.

Gennart JP, Baleux C, Verellen-Dumoulin C, Buchet JP, De Meyer R, Lauwerys R (1993). Increased sister chromatid exchanges and tumor markers in workers exposed to elemental chromium-, cobalt- and nickel-containing dusts. Mutat Res. 299(1):55–61. https://doi.org/10.1016/0165-1218(93)90119-X PMID:7679193

Government of British Columbia (2019). Environmental management act, contaminated sites regulation B.C. Reg. 375/96. Victoria (BC), Canada: Government of British Columbia. Available from: https://www.bclaws.gov.bc.ca/civix/document/id/crbc/crbc/375_96_multi, accessed 30 September 2022.

Hengstler JG, Bolm-Audorff U, Faldum A, Janssen K, Reifenrath M, Götte W, et al. (2003). Occupational exposure to heavy metals: DNA damage induction and DNA repair inhibition prove co-exposures to cadmium, cobalt and lead as more dangerous than hitherto expected. Carcinogenesis. 24(1):63–73. https://doi.org/10.1093/carcin/24.1.63 PMID:12538350

Hogstedt C, Alexandersson R (1990). Causes of death of hard metal workers. Arb Hälsa. 21:1–26. [Swedish]

Hutter HP, Wallner P, Moshammer H, Marsh G (2016). Dust and cobalt levels in the Austrian tungsten industry: workplace and human biomonitoring data. Int J Environ Res Public Health. 13(9):931. https://doi.org/0.3390/ijerph13090931 PMID:27657104

IFA (2021). Cobalt and its compounds. GESTIS International Limit Values database. Germany: Institut für Arbeitsschutz der Deutschen Gesetzlichen Unfallversicherung (Institute for Occupational Safety and Health of the German Social Accident Insurance). Available from: https://www.dguv.de/ifa/gestis/gestis-internationale-grenzwerte-fuer-chemische-substanzen-limit-values-for-chemical-agents/index-2.jsp, accessed 30 September 2022.

Johnstone EB, Louis GM, Parsons PJ, Steuerwald AJ, Palmer CD, Chen Z, et al. (2014). Increased urinary cobalt and whole blood concentrations of cadmium and lead in women with uterine leiomyomata: findings from the ENDO Study. Reprod Toxicol. 49:27–32. https://doi.org/10.1016/j.reprotox.2014.06.007 PMID:24994689

JSOH (2020). Recommendation of occupational exposure limits (2020–2021). Environ Occup Health Practice. 2(1): 1–34. https://doi.org/10.1539/eohp.roel2020

Katsarou A, Baxevanis C, Armenaka M, Volonakis M, Balamotis A, Papamihail M (1997). Study of persistence and loss of patch test reactions to dichromate and cobalt. Contact Dermat. 36(2):87–90. https://doi.org/10.1111/j.1600-0536.1997.tb00419.x PMID:9062743

Kennedy KJ, Esmen NA, Buchanich JM, Zimmerman S, Sleeuwenhoek AJ, Marsh GM (2017). Mortality among hardmetal production workers. Occupational exposures. J Occup Environ Med. 59(12):e297–305. https://doi.org/10.1097/JOM.0000000000001068 PMID:28704227

Klasson M, Bryngelsson IL, Pettersson C, Husby B, Arvidsson H, Westberg H (2016). Occupational exposure to cobalt and tungsten in the Swedish hard metal industry: air concentrations of particle mass, number, and surface area. Ann Occup Hyg. 60(6):684–99. PMID:27143598

Krakowiak A, Dudek W, Tarkowski M, Świderska-Kiełbik S, Nieścierenko E, Pałczyński C (2005). Occupational asthma caused by cobalt chloride in a diamond polisher after cessation of occupational exposure: a case report. Int J Occup Med Environ Health. 18(2):151–8. PMID:16201206

Kresovich JK, Erdal S, Chen HY, Gann PH, Argos M, Rauscher GH (2019). Metallic air pollutants and breast cancer heterogeneity. Environ Res. 177:108639. https://doi.org/10.1016/j.envres.2019.108639 PMID:31419716

Kusaka Y, Kumagai S, Kyono H, Kohyama N, Shirakawa T (1992). Determination of exposure to cobalt and nickel in the atmosphere in the hard metal industry. Ann Occup Hyg. 36(5):497–507. https://doi.org/10.1093/annhyg/36.5.497 PMID:1444069

Kusaka Y, Yokoyama K, Sera Y, Yamamoto S, Sone S, Kyono H, et al. (1986). Respiratory diseases in hard metal workers: an occupational hygiene study in a factory. Br J Ind Med. 43(7):474–85. https://doi.org/10.1136/oem.43.7.474 PMID:3718895

L’vova GN, Chopikashvili LV, Vasil’eva IM, Zasukhina GD (1990). Protective effect of ascorbic acid in cells of people exposed to cobalt chloride. Genetika. 26(7):1316–9. PMID:2121602 [Russian]

Lasfargues G, Wild P, Moulin JJ, Hammon B, Rosmorduc B, Rondeau du Noyer C, et al. (1994). Lung cancer mortality in a French cohort of hard-metal workers. Am J Ind Med. 26(5):585–95. https://doi.org/10.1002/ajim.4700260502 PMID:7832207

Li Z, Long T, Wang R, Feng Y, Hu H, Xu Y, et al. (2021a). Plasma metals and cancer incidence in patients with type 2 diabetes. Sci Total Environ. 758:143616. https://doi.org/10.1016/j.scitotenv.2020.143616 PMID:33218808

Li Z, Wang Z, Xue K, Wang Z, Guo C, Qian Y, et al. (2021b). High concentration of blood cobalt is associated with the impairment of blood-brain barrier permeability. Chemosphere. 273:129579. https://doi.org/10.1016/j.chemosphere.2021.129579 PMID:33493816

Linna A, Oksa P, Groundstroem K, Halkosaari M, Palmroos P, Huikko S, et al. (2004). Exposure to cobalt in the production of cobalt and cobalt compounds and its effect on the heart. Occup Environ Med. 61(11):877–85. https://doi.org/10.1136/oem.2003.009605 PMID:15477280

Linna A, Oksa P, Palmroos P, Roto P, Laippala P, Uitti J (2003). Respiratory health of cobalt production workers. Am J Ind Med. 44(2):124–32. https://doi.org/10.1002/ajim.10258 PMID:12874844

Marsh GM, Buchanich JM, Zimmerman S, Liu Y, Balmert LC, Esmen NA, et al. (2017a). Mortality among hardmetal production workers. US cohort and nested case-control studies. J Occup Environ Med. 59(12):e306–26. https://doi.org/10.1097/JOM.0000000000001075 PMID:29215485

Marsh GM, Buchanich JM, Zimmerman S, Liu Y, Balmert LC, Graves J, et al. (2017b). Mortality among hardmetal production workers. Pooled analysis of cohort data from an international investigation. J Occup Environ Med. 59(12):e342–64. https://doi.org/10.1097/JOM.0000000000001151 PMID:29215487

Marsh GM, Esmen NA, Buchanich JM, Youk AO (2009). Mortality patterns among workers exposed to arsenic, cadmium, and other substances in a copper smelter. Am J Ind Med. 52(8):633–44. https://doi.org/10.1002/ajim.20714 PMID:19533624

Mateuca R, Aka PV, De Boeck M, Hauspie R, Kirsch-Volders M, Lison D (2005). Influence of *hOGG1*, *XRCC1* and *XRCC3* genotypes on biomarkers of genotoxicity in workers exposed to cobalt or hard metal dusts. Toxicol Lett. 156(2):277–88. https://doi.org/10.1016/j.toxlet.2004.12.002 PMID:15737490

Matharu GS, Berryman F, Judge A, Reito A, McConnell J, Lainiala O, et al. (2017). Blood metal ion thresholds to identify patients with metal-on-metal hip implants at risk of adverse reactions to metal debris. An external multicenter validation study of Birmingham hip resurfacing and corail-pinnacle implants. J Bone Joint Surg Am. 99(18):1532–9. https://doi.org/10.2106/JBJS.16.01568 PMID:28926382

McElvenny DM, MacCalman LA, Sleeuwenhoek A, Davis A, Miller BG, Alexander C, et al. (2017). Mortality among hardmetal production workers. UK cohort and nested case-control studies. J Occup Environ Med. 59(12):e275–81. https://doi.org/10.1097/JOM.0000000000001036 PMID:28697060

Mérida-Ortega Á, Rothenberg SJ, Cebrián ME, López-Carrillo L (2022). Breast cancer and urinary metal mixtures in Mexican women. Environ Res. 210:112905 https://doi.org/10.1016/j.envres.2022.112905 PMID:35217012

Morfeld P, Groß JV, Erren TC, Noll B, Yong M, Kennedy KJ, et al. (2017). Mortality among hardmetal production workers. German historical cohort study. J Occup Environ Med. 59(12):e288–96. https://doi.org/10.1097/JOM.0000000000001061 PMID:29215484

Moulin JJ, Clavel T, Roy D, Dananché B, Marquis N, Févotte J, et al. (2000). Risk of lung cancer in workers producing stainless steel and metallic alloys. Int Arch Occup Environ Health. 73(3):171–80. https://doi.org/10.1007/s004200050024 PMID:10787132

Moulin JJ, Wild P, Mur JM, Fournier-Betz M, Mercier-Gallay M (1993). A mortality study of cobalt production workers: an extension of the follow-up. Am J Ind Med. 23(2):281–8. https://doi.org/10.1002/ajim.4700230205 PMID:8427256

Moulin JJ, Wild P, Romazini S, Lasfargues G, Peltier A, Bozec C, et al. (1998). Lung cancer risk in hard-metal workers. Am J Epidemiol. 148(3):241–8. https://doi.org/10.1093/oxfordjournals.aje.a009631 PMID:9690360

Mur JM, Moulin JJ, Charruyer-Seinerra MP, Lafitte J (1987). A cohort mortality study among cobalt and sodium workers in an electrochemical plant. Am J Ind Med. 11(1):75–81. https://doi.org/10.1002/ajim.4700110108 PMID:3812499

Nemery B, Nagels J, Verbeken E, Dinsdale D, Demedts M (1990). Rapidly fatal progression of cobalt lung in a diamond polisher. Am Rev Respir Dis. 141(5 Pt 1):1373–8. https://doi.org/10.1164/ajrccm/141.5_Pt_1.1373 PMID:2160215

Niehoff NM, O'Brien KM, Keil AP, Levine KE, Liyanapatirana C, Haines LG, et al. (2021). Metals and breast cancer risk: a prospective study using toenail biomarkers. Am J Epidemiol. 190(11):2360–73. https://doi.org/10.1093/aje/kwab204 PMID:34268559

Norwegian Scientific Committee for Food and Environment (2007). Risk assessment of health hazards from nickel, cobalt, zinc, iron, copper and manganese migrated from ceramic articles. Opinion of the Panel on Food Additives, Flavourings, Processing Aids, Materials in Contact with Food and Cosmetics of the Norwegian Scientific Committee for Food Safety, Adopted 2 May 2007. Oslo, Norway: Norwegian Scientific Committee for Food and Environment (Vitenskapskomiteen for mat og miljø). Available from: https://vkm.no/download/18.d44969415d027c43cf13da6/1501076192110/ebc8d55983.pdf, accessed 14 March 2022.

NTP (2021). 15th report on carcinogens. Research Triangle Park (NC), USA: United States Department of Health and Human Services, Public Health Service. Available from: https://doi.org/10.22427/NTP-OTHER-1003, accessed 14 March 2022.

O'Rorke MA, Cantwell MM, Abnet CC, Brockman AJ, Murray LJ; FINBAR Study Group (2012). Toenail trace element status and risk of Barrett's oesophagus and oesophageal adenocarcinoma: results from the FINBAR study. Int J Cancer. 131(8):1882–91. https://doi.org/10.1002/ijc.27434 PMID:22262413

Ontario Ministry of Environment and Energy (1996). Scientific criteria document for the development of a provincial water quality objective for cobalt (stable isotope). PIBS 3361E. Toronto (ON), Canada: Ontario Ministry of Environment and Energy. Available from: https://archive.org/details/cobaltscientific00torouoft, accessed 1 February 2022.

Pan D, Wang S, Su M, Sun G, Zhu X, Ghahvechi Chaeipeima M, et al. (2021). Vitamin B_{12} may play a preventive role in esophageal precancerous lesions: a case–control study based on markers in blood and 3-day duplicate diet samples. Eur J Nutr. 60(6):3375–86. https://doi.org/10.1007/s00394-021-02516-0 PMID:33619628

Princivalle A, Iavicoli I, Cerpelloni M, Franceschi A, Manno M, Perbellini L (2017). Biological monitoring of cobalt in hard metal factory workers. Int Arch Occup Environ Health. 90(2):243–54. https://doi.org/10.1007/s00420-016-1190-y PMID:28078438

Rizzato G, Fraioli P, Sabbioni E, Pietra R, Barberis M (1994). The differential diagnosis of hard metal lung disease. Sci Total Environ. 150(1–3):77–83. https://doi.org/10.1016/0048-9697(94)90132-5 PMID:7939613

Rodrigues EG, Herrick RF, Stewart J, Palacios H, Laden F, Clark W, et al. (2020). Case–control study of brain and other central nervous system cancer among workers at semiconductor and storage device manufacturing facilities. Occup Environ Med. 77(4):238–48. https://doi.org/10.1136/oemed-2019-106120 PMID:32019845

Rodrigues EG, Stewart J, Herrick R, Palacios H, Laden F, Clark W, et al. (2019). Retrospective exposure assessment for semiconductor and storage device manufacturing facilities. J Occup Environ Med. 61(4):e132–8. https://doi.org/10.1097/JOM.0000000000001544 PMID:30946698

Rogers MAM, Thomas DB, Davis S, Vaughan TL, Nevissi AE (1993). A case-control study of element levels and cancer of the upper aerodigestive tract. Cancer Epidemiol Biomarkers Prev. 2(4):305–12. PMID:8348053

Saravanabhavan G, Werry K, Walker M, Haines D, Malowany M, Khoury C (2017). Human biomonitoring reference values for metals and trace elements in blood and urine derived from the Canadian Health Measures Survey 2007–2013. Int J Hyg Environ Health. 220(2 Pt A):189–200. https://doi.org/10.1016/j.ijheh.2016.10.006 PMID:27776932

Sauni R, Oksa P, Uitti J, Linna A, Kerttula R, Pukkala E (2017). Cancer incidence among Finnish male cobalt production workers in 1969–2013: a cohort study. BMC Cancer. 17(1):340. https://doi.org/10.1186/s12885-017-3333-2 PMID:28521771

Scharf B, Clement CC, Zolla V, Perino G, Yan B, Elci SG, et al. (2014). Molecular analysis of chromium and cobalt-related toxicity. Sci Rep. 4(1):5729. https://doi.org/10.1038/srep05729 PMID:25034144

Schmitz-Spanke S, Drexler H, Hartwig A, MAK Commission (2019). Addendum to cobalt and cobalt compounds [BAT value documentation, 2018]. In: The MAK-collection for occupational health and safety: annual thresholds and classifications for the workplace. Vol. 4, No. 3. Weinheim, Germany: Wiley-VCH Verlag GmbH Co. KGaA. Available from: https://doi.org/10.1002/3527600418.bb744048vere2319

Shirakawa T, Morimoto K (1997). Interplay of cigarette smoking and occupational exposure on specific immunoglobulin E antibodies to cobalt. Arch Environ Health. 52(2):124–8. https://doi.org/10.1080/00039899709602875 PMID:9124872

Svartengren M, Bryngelsson IL, Marsh G, Buchanich J, Zimmerman S, Kennedy K, et al. (2017). Cancer incidence among hardmetal production workers: the Swedish cohort. J Occup Environ Med. 59(12):e365–73. https://doi.org/10.1097/JOM.0000000000001185 PMID:29215488

Swennen B, Buchet JP, Stánescu D, Lison D, Lauwerys R (1993). Epidemiological survey of workers exposed to cobalt oxides, cobalt salts, and cobalt metal. Br J Ind Med. 50(9):835–42. https://doi.org/10.1136/oem.50.9.835 PMID:8398878

Tilakaratne D, Sidhu S (2015). Heavy metal (monoclonal) bands: a link between cutaneous T-cell lymphoma and contact allergy to potassium dichromate, nickel and cobalt? Australas J Dermatol. 56(1):59–63. https://doi.org/10.1111/ajd.12182 PMID:25303728

Tüchsen F, Jensen MV, Villadsen E, Lynge E (1996). Incidence of lung cancer among cobalt-exposed women. Scand J Work Environ Health. 22(6):444–50. https://doi.org/10.5271/sjweh.166 PMID:9000312

USGS (2021b). US Geological Survey Minerals Yearbook 2019. Cobalt. Washington (DC), USA: United States Department of the Interior. Available from: https://www.usgs.gov/centers/national-minerals-information-center/cobalt-statistics-and-information, accessed 1 October 2021.

Wallner P, Kundi M, Moshammer H, Zimmerman SD, Buchanich JM, Marsh GM (2017). Mortality among hardmetal production workers: a retrospective cohort study in the Austrian hardmetal industry. J Occup Environ Med. 59(12):e282–7. https://doi.org/10.1097/JOM.0000000000001046 PMID:28665836

Walters GI, Moore VC, Robertson AS, Burge CBSG, Vellore AD, Burge PS (2012). An outbreak of occupational asthma due to chromium and cobalt. Occup Med (Lond). 62(7):533–40. https://doi.org/10.1093/occmed/kqs111 PMID:22826555

Wang F, Zhu J, Yao P, Li X, He M, Liu Y, et al. (2013d). Cohort profile: the Dongfeng–Tongji cohort study of retired workers. Int J Epidemiol. 42(3):731–40. https://doi.org/10.1093/ije/dys053 PMID:22531126

Water Quality Australia (2018). Australian and New Zealand guidelines for fresh and marine water quality. Canberra (ACT), Australia: Department of Agriculture, Water and the Environment. Available from: https://www.waterquality.gov.au/anz-guidelines, accessed 1 February 2022.

Westberg H, Bryngelsson IL, Marsh G, Buchanich J, Zimmerman S, Kennedy K, et al. (2017). Mortality among hardmetal production workers. The Swedish cohort. J Occup Environ Med. 59(12):e263–74. https://doi.org/10.1097/JOM.0000000000001054 PMID:29215483

White AJ, O’Brien KM, Niehoff NM, Carroll R, Sandler DP (2019). Metallic air pollutants and breast cancer risk in a nationwide cohort study. Epidemiology. 30(1):20–8. https://doi.org/10.1097/EDE.0000000000000917 PMID:30198937

WHO (2006). Cobalt and inorganic cobalt compounds. Geneva, Switzerland: World Health Organization. https://apps.who.int/iris/bitstream/handle/10665/43426/9241530693_eng.pdf?sequence=1&isAllowed=yhttps://apps.who.int/iris/handle/10665/43426, accessed 6 March 2022.

Wild P, Perdrix A, Romazini S, Moulin JJ, Pellet F (2000). Lung cancer mortality in a site producing hard metals. Occup Environ Med. 57(8):568–73. https://doi.org/10.1136/oem.57.8.568 PMID:10896965

Wultsch G, Nersesyan A, Kundi M, Mišík M, Setayesh T, Waldherr M, et al. (2017). Genotoxic and cytotoxic effects in exfoliated buccal and nasal cells of chromium and cobalt exposed electroplaters. J Toxicol Environ Health A. 80(13–15):651–60. https://doi.org/10.1080/15287394.2017.1286918 PMID:28524814

Xue K, Qian Y, Wang Z, Guo C, Wang Z, Li X, et al. (2021). Cobalt exposure increases the risk of fibrosis of people living near E-waste recycling area. Ecotoxicol Environ Saf. 215:112145. https://doi.org/10.1016/j.ecoenv.2021.112145 PMID:33743401