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Volume 52
Chlorinated Drinking-water; Chlorination By-products; Some Other Halogenated Compounds; Cobalt and Cobalt Compounds

Summary of Data Reported and Evaluation

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CHLORINATED DRINKING-WATER
(Group 3)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 45)

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Water supplies were first chlorinated at the turn of the century, and over the following two decades chlorination was introduced for disinfection of drinking-water in most industrialized countries. In the chlorination process, chlorine reacts mainly with natural water constituents to produce a complex mixture of by-products, including a wide variety of halogenated compounds, the actual levels of which depend on the amount of chlorine added and the type of water source. In general, groundwaters produce lower levels, while surface waters often tend to produce higher levels of chlorination by-products; however, there is some evidence that groundwaters can give higher levels of brominated substances, probably due to higher levels of bromide in the untreated water. Estimates of the total halogenated organic matter generated during chlorination suggest typical levels in the range < 10-250 μg/l as chlorine. The main chlorination by-products are trihalomethanes and chlorinated acetic acids, which usually occur in the range 1-100 μg/l (although higher levels have been reported). Many products occur in the range 1-10 μg/l, while a large number can be detected at levels of < 1 μg/l. The by-products responsible for most of the bacterial mutagenicity found in chlorinated drinking-water, 3-chloro-4-(dichloromethyl)-5-hydroxy-2[5*H*]- furanone (MX) and associated substances, are present at very low concentrations (< 0.1 μg/l).

5.2 Experimental carcinogenicity data

Two series of studies were considered to provide evidence that could support an evaluation of the potential carcinogenicity of chlorinated drinking-water.

Samples of material concentrated from treated and undisinfected or treated and chlorinated water samples were tested in mice in three initiation-promotion experiments (by subcutaneous injection followed by topical application of 12-*O*-tetradecanoylphorbol 13-acetate). None of the concentrates derived from the chlorinated water induced a significantly increased incidence of skin tumours when compared with concentrates derived from undisinfected water samples or with saline.

In one experiment in mice, oral administration of chlorinated humic acids in the drinking-water did not increase the incidence of tumours over that in animals receiving unchlorinated humic acids or in saline-treated controls.

5.3 Human carcinogenicity data

Seven case-control studies conducted in the USA were considered to provide evidence that could support an evaluation. Four of these had community exposure data, and three had individually derived exposure data. The four studies with community exposure data each included several cancer sites. One study showed a significant increase in risk for colon cancer only; another showed a significant increase only for rectal cancer; the other two studies showed no excess risk for cancer.

Of the three case-control studies with individual exposure data, one was a population-based study of urinary bladder cancer carried out by interview in 10 areas of the USA. Many potential confounding factors, including smoking, were taken into account in the analyses. An early analysis of the study showed a significant association between long-term use at home of a chlorinated surface water source (as compared to an unchlorinated groundwater source) and urinary bladder cancer in nonsmokers only. In a subsequent analysis,

tap-water intake was considered in addition to home water source, and consumption level of tap water was significantly associated with urinary bladder cancer; this effect was substantially confined to those who had lived for 40 years or more in a house with a chlorinated surface water source. There were significant and increasing trends in urinary bladder cancer risk with duration of residence in a house with a chlorinated surface water source for both women and nonsmokers whose tap-water consumption was above the median. In a further report based only on Iowa participants in this study, risk for urinary bladder cancer was associated with duration of use of a chlorinated water source, and the association became stronger with increasing accuracy of the exposure measure.

In the second of these case-control studies, carried out in Massachusetts (USA), the authors reported an excess risk for mortality from urinary bladder cancer among people who had lived in areas with chlorinated water supplies as compared with chloraminated supplies. Some confounding factors, including smoking, were taken into account; however, the proportion of eligible subjects for whom exposure could be ascertained was low.

In a third case-control study, based on deaths among members of the New York State Teachers' Retirement System, no association was found between deaths from cancers of the colon and rectum combined and estimated use of surface water or intake of chloroform from domestic and workplace water supplies over the 20 years prior to death. Few confounding variables were taken into acount.

A cohort of the general population in a county in Maryland (USA) was enrolled and surveyed in 1963 and followed up to 12 years. Urinary bladder cancer incidence was found to be higher in both men and women residents supplied mainly by a chlorinated surface water source compared with county residents who obtained their drinking-water from unchlorinated deep wells; but the effects of chlorinated drinking-water could not be distinguished from factors related to urbanicity, and the numbers were too small to rule out a chance effect.

Six correlation studies and one time-trend study were considered by the Working Group to provide some useful data. These studies showed moderately consistent patterns of a positive correlation between use of surface water or of chlorinated water and cancers of the stomach, colon, rectum, urinary bladder and lung, with the most consistent patterns for cancers of the urinary bladder and rectum.

The studies that were considered informative, and therefore included in this summary, were nevertheless difficult to interpret in an evaluation of the carcinogenicity of chlorinated drinking-water. The water variables studied - whether surface or groundwater and others - were generally imperfect surrogates for the subject of this monograph. There is cause for some scepticism about the estimates of exposure to chlorinated drinking-water in all of these studies. Furthermore, very few attempted to document exposure over long periods of the subjects' lives. Chlorination by-products differ according to local conditions and practices of chlorination, and the health effects found in one place may not be found elsewhere. Many variables, such as smoking habits, dietary practices and environmental conditions, influence the risks for cancer, and they may differ between populations served by chlorinated and unchlorinated water supplies. Such factors should ideally be taken into account in an epidemiological study; however, in most of the studies evaluated, there was little if any information available about them. When the data are examined on the basis of individual cancer sites, the evidence of elevated risk is strongest for cancer of the urinary bladder. The strongest study of cancer at this site supports the hypothesis of an elevated risk due to drinking chlorinated surface water compared with unchlorinated groundwater. However, the sum of the evidence from other studies, although showing some degree of consistency, is severely compromised by the weaknesses outlined above.

5.4 Other relevant data

Elevated serum cholesterol levels were reported in women but not in men living in communities served by chlorinated *versus* nonchlorinated water supplies in one study. No difference in the prevalence of ancephaly was observed between villages served by chlorinated and nonchlorinated groundwater in another study.

In regard to studies of genetic and related effects, only those reports were included in which the role of chlorination could be evaluated. Samples of unconcentrated chlorinated drinking-water were not genotoxic in bacteria or in a micronucleus assay in plants and did not induce morphological transformation in cultured

mammalian cells. Samples of organic material concentrated from chlorinated surface waters were usually genotoxic in bacteria and induced sister chromatid exchange, micronuclei and chromosomal aberrations in single studies with cultured mammalian cells. In a single study, no activity was observed in a mammalian cell assay for mutation.

Samples of organic material concentrated from chlorinated groundwaters were less frequently mutagenic in bacteria than those from chlorinated surface waters; in a single study, they induced sister chromatid exchange but not micronuclei in cultured mammalian cells.

Samples of organic material concentrated from surface water treated with either chlorine dioxide or ozone followed by chlorination induced mutation in bacteria in some studies.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of chlorinated drinking-water in humans.

There is *inadequate evidence* for the carcinogenicity of chlorinated drinking-water in experimental animals.

Overall evaluation

Chlorinated drinking-water is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Last updated: 17 November 1997

SODIUM CHLORITE
(Group 3)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 145)

Sodium chlorite
CAS No.: 7758-192

Sodium chlorite trihydrate
CAS No.: 49658-21-1

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Sodium chlorite is the only chlorite salt produced commercially in significant quantities. It is used mainly for the generation of chlorine dioxide *in situ* for bleaching textiles, in pulp and paper processing, and for disinfection. Sodium chlorite is used in a small number of water treatment plants to generate chlorine dioxide; this may result in low residual concentrations of chlorite in drinking-water.

No information was available on occupational exposures to sodium chlorite.

5.2 Experimental carcinogenicity data

Sodium chlorite was tested for carcinogenicity in male and female B6C3F_1 mice and Fischer 344 rats by oral administration in the drinking-water and in a limited study by skin application in female Sencar mice. It was further tested for promoting effects in female Sencar mice by skin application following a single application of 7,12-dimethylbenz[*a*]anthracene. Oral administration of sodium chlorite to mice was associated with a marginal increase in the incidence of lung tumours in treated males. In the study in rats, no significant increase in tumour incidence at any site was seen in treated animals. Skin tumours did not occur in Sencar mice following skin application of sodium chlorite. In the initiation/promotion study, sodium chlorite had a marginal promoting effect.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

Sodium chlorite has been shown to produce haemolytic anaemia in several animal species at concentrations in drinking-water of 100 mg/l or higher. No sign of such effects was seen in humans at much lower doses.

Minimal adverse reproductive effects were observed in rats and mice given sodium chlorite in the drinking-water at concentrations of 100 mg/l or higher.

Single studies indicated that sodium chlorite induced mutations in bacteria and chromosomal aberrations in cultured mammalian cells. In mice treated *in vivo*, conflicting results were obtained with regard to the induction of micronuclei, while a single study showed no induction of aneuploidy, chromosomal aberrations or abnormal sperm morphology.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of sodium chlorite in experimental animals.

No data were available from studies in humans on the carcinogenicity of sodium chlorite.

Overall evaluation

Sodium chlorite is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Synonyms for Sodium chlorite

- Chlorous acid, sodium salt
- Neo Silox D
- Textone

Synonym for Sodium chlorite trihydrate

- Chlorous acid, sodium salt, trihydrate

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HYPOCHLORITE SALTS
(Group 3)

VOL.: 52 (1991) (p. 159)

Calcium hypochlorite
CAS No.: 7778-54-3

Calcium hypochlorite, dibasic
CAS No.: 12394-14-8

Calcium hypochlorite dihydrate
CAS No.: 22464-76-2

Lithium hypochlorite
CAS No.: 13840-33-0

Potassium hypochlorite
CAS No.: 7778-66-7

Sodium hypochlorite
CAS No.: 7681-52-9

Sodium hypochlorite heptahydrate
CAS No.: 6431-03-9

Sodium hypochlorite hydrate (2:5)
CAS No.: 55248-17-4

Sodium hypochlorite pentahydrate
CAS No.: 10022-70-5

Calcium sodium hypochorite
CAS No.: 53053-57-9

5. Summary of Data Reported and Evaluation

5.1 Exposure data

The principal hypochlorite salts produced commercially are calcium, sodium and lithium hypochlorites. Calcium hypochlorite (solid or aqueous solution) is widely used for disinfection in swimming pools and in industrial applications and for pulp and textile bleaching. Sodium hypochlorite (aqueous solution) is used as a household laundry bleach, in commercial laundering, in pulp and paper manufacture, in industrial chemical synthesis and in the disinfection of drinking-water. Lithium hypochlorite (solid) is used in swimming pools for disinfection and in household detergents.

Hypochlorite salts (principally sodium hypochlorite) are used to disinfect drinking-water at many small treatment works. In the disinfection of drinking-water and wastewater, addition of hypochlorite salts and of chlorine gas gives the same chlorine species in solution - i.e., an equilibrium mixture of mainly hypochlorous acid and hypochlorite anion. In this way, much of the general population is exposed to hypochlorite *via* chlorinated drinking-water (see the monograph on Chlorinated drinking-water).

5.2 Experimental carcinogenicity data

Sodium hypochlorite was tested for carcinogenicity in a two-year study in male and female B6C3F_1 mice and Fischer 344 rats by oral administration in drinking-water, in limited studies in female Sencar mice and in female ddN mice by skin application. Sodium hypochlorite was also tested for promoting effects in female Sencar mice following initiation with 7,12-dimethylbenz[*a*]anthracene and in female ddN mice following initiation with 4-nitroquinoline 1-oxide. Sodium hypochlorite administered in the drinking-water did not increase the proportion of rats or mice with tumours. Sodium hypochlorite applied to the skin of Sencar mice or ddN mice did not produce skin tumours. No skin promoting effect was observed in the study with 7,12-dimethylbenz[*a*]anthracene, whereas some effect was seen in the study with 4-nitroquinoline 1-oxide.

Drinking-water containing 100 mg/l chlorine was tested for carcinogenicity in a multigeneration study in male and female BDII rats. No increase in the incidence of tumours was seen in treated animals relative to controls through six generations.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

Sodium hypochlorite induced genotoxic effects in bacteria. In single studies, chromosomal aberrations were observed in cultured mammalian cells, whereas sister chromatid exchange but no chromosomal aberration was seen in cultured human cells. In a single study, micronuclei were induced in newt larvae. In mice, no induction of micronuclei, aneuploidy or chromosomal aberrations was observed in bone marrow, but abnormal sperm morphology was seen after administration of sodium hypochlorite.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of hypochlorite salts in experimental animals.

No data were available from studies in humans on the carcinogenicity of hypochlorite salts.

Overall evaluation

Hypochlorite salts are *not classifiable as to their carcinogenicity to humans (Group 3)*.

Synonyms for Calcium hypochlorite

- Calcium oxychloride
- Camporit
- Chemiclor G
- Chloride of lime
- Chlorinated lime
- Chlorolime chemical
- Eusol BPC
- HTH
- HTH (bleaching agent)
- Hypochlorite acid, calcium salt
- Lime chloride
- Losantin
- Pittchlor
- B-K Powder

- Solvox KS
- T-Eusol

Synonyms for Calcium hypochlorite, dibasic

- Calcium hydroxide hypochlorite
- Lime chloride

Synonym for Calcium hypochlorite dihydrate

- Hypochlorous acid, calcium salt, dihydrate

Synonyms for Lithium hypochlorite

- Hypochlorous acid, lithium salt
- Lithium chloride oxide
- Lithium oxychloride

Synonyms for Potassium hypochlorite

- Hypochlorous acid, potassium salt
- Potassium chloride oxide

Synonyms for Sodium hypochlorite

- Antiformin
- B-K Liquid
- Carrel-Dakin solution
- Chloros
- Clorox
- Dakin's solution
- Deosan
- Hypochlorite
- Hypochlorous acid, sodium salt
- Javex
- Klorocin
- Milton
- Neo-cleaner
- Neoseptal Cl
- Parozone
- Purin B
- Sodium chloride oxide
- Sodium oxychloride
- Surchlor
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Synonym for Sodium hypochlorite hydrate (2:5)

- Hypochlorous acid, sodium salt, hydrate (2:5)

Synonym for Sodium hypochlorite heptahydrate

- Hypochlorous acid, sodium salt, heptahydrate

Synonym for sodium hypochlorite pentahydrate

- Hypochlorous acid, sodium salt, pentahydrate

Synonym for Calcium sodium hypochorite

- Hypochlorous acid, calcium sodium salt (3:1:1)

Last updated: 17 November 1997

BROMODICHLOROMETHANE
(Group 2B)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 179)

CAS No.: 75-27-4

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Bromodichloromethane is found in chlorinated drinking-water as a consequence of the reaction between chlorine, added during water treatment, and natural organic substances in the presence of bromide. The major route of human exposure to bromodichloromethane is *via* drinking-water. It has been detected in chlorinated drinking-water in many parts of the world; it has also been detected in some untreated waters, but at much lower levels. Bromodichloromethane is a major component of the organohalides produced by marine algae.

5.2 Experimental carcinogenicity data

Bromodichloromethane was tested for carcinogenicity in two-year studies in male and female Fischer 344 rats and B6C3F_1 mice by oral gavage, in life-span studies in male and female Wistar rats and in CBA x C57Bl/6 hybrid mice by administration in drinking-water. In the gavage studies, bromodichloromethane increased the incidences of adenomatous polyps and adenocarcinomas of the large intestine and of tubular-cell adenomas and adenocarcinomas of the kidney in male and female rats, of tubular-cell adenomas and adenocarcinomas of the kidney in male mice and of hepatocellular adenomas and carcinomas in female mice. In the study by administration in drinking-water, it induced neoplastic nodules and adenofibrosis of the liver in rats; no increase in tumour incidence was seen in mice. In a screening test for lung adenomas by intraperitoneal injection, bromodichloromethane did not increase the incidence of lung tumours in strain A mice.

5.3 Human carcinogenicity data

No relevant data were available to the Working Group.

5.4 Other relevant data

Repeated exposure of rats and mice to bromodichloromethane resulted in toxic effects in several organs, including the liver and kidney.

A study of developmental toxicity in rats given bromodichloromethane throughout the period of major organogenesis showed skeletal variations in the presence of maternal toxicity but no teratogenic effect.

Bromodichloromethane induced mutations in some studies with bacteria and, in a single study, in cultured mammalian cells. Chromosomal aberrations but not sister chromatid exchange were observed in cultured mammalian cells. In single studies, sister chromatid exchange was observed in cultured human cells and in mouse bone marrow *in vivo*. In one study, bromodichloromethane did not induce micronuclei in bone-marrow cells of mice treated *in vivo*.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of bromodichloromethane in humans.

There is *sufficient evidence* for the carcinogenicity of bromodichloromethane in experimental animals.

Overall evaluation

Bromodichloromethane is *possibly carcinogenic to humans (Group 2B)*.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Vol. 71 (1999)

Synonyms for bromodichloromethane

- Dichlorobromomethane
- Dichloromonobromomethane
- Monobromodichloromethane

Last updated: 13 April 1999

BROMOFORM
(Group 3)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 213)

CAS No.: 75-25-2

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Bromoform has a limited number of industrial uses. It is also found in chlorinated drinking-water as a consequence of the reaction between chlorine, added during water treatment, and natural organic substances in the presence of bromide ion. Bromoform has been detected in chlorinated drinking-water in many parts of the world; it has also been detected in untreated water, but at lower levels. Bromoform is the major organohalide produced by chlorination of seawater during desalination. It is a major component of the organohalides produced by marine algae.

The major route of human exposure to bromoform is from drinking-water, although ambient air is also an important source of exposure in some areas.

5.2 Experimental carcinogenicity data

Bromoform was tested for carcinogenicity in a two-year study by oral gavage in male and female B6C3F_1 mice and Fischer 344 rats. It induced adenomatous polyps and adenocarcinomas of the large intestine in male and female rats. Bromoform did not increase the proportion of mice with tumours. In a screening test by intraperitoneal injection, there was a slight increase in the average number of lung tumours in strain A mice given the middle dose of bromoform.

5.3 Human carcinogenicity data

No relevant data were available to the Working Group.

5.4 Other relevant data

In experimental animals, bromoform induced liver and kidney damage and decreased the immune response.

There is some evidence of developmental toxicity in the absence of maternal toxicity in rats.

Mutagenic effects of bromoform were observed occasionally in bacteria. In single studies, bromoform induced mitotic arrest in plants, mutations in insects and in cultured mammalian cells and sister chromatid exchange in human lymphocytes. Chromosomal aberrations were induced in cultured mammalian cells. In single studies in rodents *in vivo*, bromoform did not bind to DNA or cause chromosomal aberrations. Sister chromatid exchange was induced in rodents *in vivo*.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of bromoform in humans.

There is *limited evidence* for the carcinogenicity of bromoform in experimental animals.

Overall evaluation

Bromoform is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Vol. 71 (1999)

Synonyms

- Methenyl tribromide
- Methyl tribromide
- Tribromomethane

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CHLORODIBROMOMETHANE
(Group 3)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 243)

CAS No.: 124-48-1

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Chlorodibromomethane has limited commercial use but is used industrially as a chemical intermediate. It is found in chlorinated drinking-water as a consequence of the reaction between chlorine, added during drinking-water treatment, and natural organic substances in the presence of bromide. The major route of human exposure is *via* drinking-water. Chlorodibromomethane has been detected in chlorinated drinking-water in many parts of the world; it is not normally present in untreated water. It is a major component of organohalide emissions from marine algae.

5.2 Experimental carcinogenicity data

Chlorodibromomethane was tested for carcinogenicity in two-year studies by oral gavage in male and female $B6C3F_1$ mice and Fischer 344 rats and in a lifetime study in CBA x C57Bl/6 hybrid mice by administration in drinking-water. In $B6C3F_1$ mice, it produced a significant increase in the incidence of hepatocellular neoplasms in females and a marginal increase in males. Chlorodibromomethane did not increase the proportion of rats with tumours at any site relative to that in controls. There was no increase in tumour incidence in CBA x C57Bl/6 hybrid mice given chlorodibromomethane in drinking-water.

5.3 Human carcinogenicity data

No relevant data were available to the Working Group.

5.4 Other relevant data

Chlorodibromomethane was mutagenic to bacteria. In single studies, it induced mitotic recombination, but not mutation, in yeast, chromosomal aberrations in cultured mammalian cells and sister chromatid exchange in cultured human cells. Sister chromatid exchange but not micronuclei were observed in rodents treated *in vivo*.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of chlorodibromomethane in humans.

There is *limited evidence* for the carcinogenicity of chlorodibromomethane in experimental animals.

Overall evaluation

Chlorodibromomethane is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Vol. 71 (1999)

Synonyms

- Dibromomethanechloro
- Dibromomonochloromethane
- Monochlorodibromomethane

Last updated: 13 April 1999

HALOGENATED ACETONITRILES
Bromochloroacetonitrile (Group 3)
Chloroacetonitrile (Group 3)
Dibromoacetonitrile (Group 3)
Dichloroacetonitrile (Group 3)
Trichloroacetonitrile (Group 3)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 269)

Bromochloroacetonitrile
CAS No.: 83463-62-1

Chloroacetonitrile
CAS No.: 107-14-2

Dibromoacetonitrile
CAS No.: 3252-43-5

Dichloroacetonitrile
CAS No.: 3018-12-0

Trichloroacetonitrile
CAS No.: 545-06-2

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Halogenated acetonitriles are not produced on an industrial scale. Chloro- and trichloroacetonitriles have been used on a limited basis in the past as pesticides.

Several halogenated acetonitriles have been detected in chlorinated drinking-water in a number of countries as a consequence of the reaction of chlorine with natural organic substances (and bromine in the case of brominated acetonitriles) present in untreated water. The only known route of human exposure is through chlorinated drinking-water.

5.2 Experimental carcinogenicity data

Halogenated acetonitriles (chloroacetonitrile, dichloroacetonitrile, trichloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile) were tested in a limited carcinogenicity study in female Sencar mice by skin application, in an initiation/promotion study in female Sencar mice by skin application and in a screening assay for lung tumours in female strain A mice by oral administration. No skin tumour was produced by any of the haloacetonitriles after skin application in mice. In the initiation/promotion study, reproducible, significant increases in the numbers of animals with skin tumours were seen only with dibromoacetonitrile; no dose-related increase in the incidence of skin tumours was observed. Marginal increases in the proportion of mice with lung tumours occurred with all of the halogenated acetonitriles, but the increases were significant only with chloroacetonitrile, trichloroacetonitrile and bromochloroacetonitrile.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

In short-term screening studies *in vivo*, chloroacetonitrile, bromochloroacetonitrile and dibromoacetonitrile caused minimal developmental toxicity in the presence of significant maternal toxicity. In developmental toxicity studies, dichloroacetonitrile and trichloroacetonitrile caused malformations and embryolethality in the presence of maternal toxicity; with trichloroacetonitrile, embryolethality was also observed at lower dose levels in the absence of maternal toxicity.

Mutations were induced in bacteria by bromochloroacetonitrile and dichloroacetonitrile but not by chloroacetonitrile, dibromoacetonitrile or trichloroacetonitrile. Mutations were induced in insects by dichloroacetonitrile but not by dibromoacetonitrile.

Sister chromatid exchange was induced in cultured mammalian cells by all five halogenated acetonitriles. DNA strand breaks were induced in human lymphocytes *in vitro* by bromochloroacetonitrile, dibromoacetonitrile and trichloroacetonitrile.

In orally treated mice, neither micronuclei in bone-marrow cells nor sperm-head abnormalities were induced by any of the five halogenated acetonitriles.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of bromochloroacetonitrile in experimental animals.

There is *inadequate evidence* for the carcinogenicity of chloroacetonitrile in experimental animals.

There is *inadequate evidence* for the carcinogenicity of dibromoacetonitrile in experimental animals.

There is *inadequate evidence* for the carcinogenicity of dichloroacetonitrile in experimental animals.

There is *inadequate evidence* for the carcinogenicity of trichloroacetonitrile in experimental animals.

No data were available from studies in humans on the carcinogenicity of halogenated acetonitriles.

Overall Evaluation

Bromochloroacetonitrile is *not classifiable as to its carcinogenicity to humans (Group 3)*.

Chloroacetonitrile is *not classifiable as to its carcinogenicity to humans (Group 3)*.

Dibromoacetonitrile is *not classifiable as to its carcinogenicity to humans (Group 3)*.

Dichloroacetonitrile is *not classifiable as to its carcinogenicity to humans (Group 3)*.

Trichloroacetonitrile is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluations: Vol. 71 (1999) (Bromochloroacetonitrile); (Chloroacetonitrile); (Dibromoacetonitrile); (Dichloroacetonitrile); (Trichloroacetonitrile)

Synonyms for Chloroacetonitrile

- 2-Chloroacetonitrile
- α-Chloroacetonitrile
- Chloromethyl cyanide
- Monochloroacetonitrile
- Monochloromethyl cyanide

Synonym for Dichloroacetonitrile

- Dichloromethyl cyanide

Synonyms for Trichloroacetonitrile

- Cyanotrichloromethane
- 2,2,2-Trichloroacetonitrile
- Trichloromethyl cyanide
- Trichloromethylnitrile

Last updated: 13 April 1999

BROMOETHANE
(Group 3)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 299)

CAS No.: 74-96-4

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Bromoethane has limited commercial use, including that as an ethylating agent. It has been detected in ocean air as a result of emissions by marine algae.

5.2 Experimental carcinogenicity data

Bromoethane was tested for carcinogenicity in a two-year study in male and female Fischer 344 rats and $B6C3F_1$ mice by inhalation. In male rats, there was a significant increase in the incidence of adrenal phaeochromocytomas, which was not dose-related. A marginal increase in the incidence of uncommon brain tumours occurred in treated females. In mice, bromoethane induced neoplasms of the uterine endometrium; a marginal increase in the incidence of lung tumours was observed in males. In a screening study by intraperitoneal injection, bromoethane did not increase the incidence of lung tumours in strain A mice.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

Bromoethane was mutagenic in bacteria but not in insects in a single study. In other single studies, bromoethane caused sister chromatid exchange but not chromosomal aberrations in mammalian cells.

5.5 Evaluation

There is *limited evidence* for the carcinogenicity of bromoethane in experimental animals.

No data were available from studies in humans on the carcinogenicity of bromoethane.

Overall evaluation

Bromoethane is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Vol. 71 (1999)

Synonyms

- Bromic ether
- Ethyl bromide
- Hydrobromic ether
- Monobromoethane

Last updated: 13 April 1999

CHLOROETHANE
(Group 3)

VOL.: 52 (1991) (p. 315)

CAS No.: 75-00-3

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Chloroethane is produced by the hydrochlorination of ethylene. It is used in the manufacture of tetraethyllead, as an industrial ethylating agent, as a blowing agent in the production of polystyrene foam and as a local anaesthetic. Occupational exposure occurs during the production of tetraethyllead, and industrial emissions have led to detectable levels of chloroethane in ambient air.

5.2 Experimental carcinogenicity data

Chloroethane was tested for carcinogenicity in a two-year study in male and female B6C3F$_1$ mice and Fischer 344 rats by inhalation. It induced uterine carcinomas in mice; marginal increases occurred in the incidence of hepatocellular tumours in female mice and in the incidence of alveolar/bronchiolar tumours in male mice. There was a marginal increase in the incidence of skin tumours in male rats, and a few uncommon glial-cell tumours occurred in female rats.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

In single studies, chloroethane was mutagenic to bacteria but did not induce transformation in cultured mammalian cells.

5.5 Evaluation

There is *limited evidence* for the carcinogenicity of chloroethane in experimental animals.

No data were available from studies in humans on the carcinogenicity of chloroethane.

Overall evaluation

Chloroethane is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Subsequent evaluation: Vol. 71 (1999)

Synonyms

- Aethylis
- Aethylis chloridum
- Anodynon
- Chloroethyl
- Chelen
- Chlorene
- Chloridum
- Chloryl
- Chloryl anesthetic
- Cloretilo
- Dublofix
- Ether chloratus
- Ether hydrochloric
- Ether muriatic
- Ethyl chloride
- Hydrochloric ether
- Kelene
- Monochloethane
- Monochloroethane
- Muriatic ether
- Narcotile

Last updated: 13 April 1999

1,1,2-TRICHLOROETHANE
(Group 3)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 337)

CAS No.: 79-00-5

5. Summary of Data Reported and Evaluation

5.1 Exposure data

1,1,2-Trichloroethane is used as an intermediate in the production of vinylidene chloride and, to a lesser extent, as a special-purpose industrial solvent and as a chemical intermediate in other processes. It has been detected in drinking-water as well as in untreated groundwater and surface water in some locations; it may occur mainly as a result of industrial emissions.

5.2 Experimental carcinogenicity data

1,1,2-Trichloroethane was tested for carcinogenicity in a two-year study in male and female B6C3F$_1$ mice and Osborne-Mendel rats by oral administration and in Sprague-Dawley rats by subcutaneous injection. In the studies by oral administration, 1,1,2-trichloroethane produced hepatocellular neoplasms and adrenal phaeochromocytomas in mice of each sex but did not significantly increase the proportion of rats with neoplasms at any site relative to untreated controls. In the study in rats by subcutaneous injection, 1,1,2-trichloroethane did not increase the incidence of neoplasms.

In a screening assay for γ-glutamyltranspeptidase-positive foci in the liver of male Osborne-Mendel rats, 1,1,2-trichloroethane did not increase the number of foci in the liver in the initiation protocol (single injection), but the number was increased in the promotion protocol (repeated injections), with or without initiation by *N*-nitrosodiethylamine.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

1,1,2-Trichloroethane was not mutagenic to bacteria. In single studies, it induced chromosomal malsegregation in a fungus and transformation in cultured mammalian cells. S-Phase induction, but not unscheduled DNA synthesis, was observed in mice after treatment *in vivo*.

5.5 Evaluation

There is *limited evidence* for the carcinogenicity of 1,1,2-trichloroethane in experimental animals.

No data were available from studies in humans on the carcinogenicity of 1,1,2-trichloroethane.

Overall evaluation

1,1,2-Trichloroethane is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see Preamble Evaluation.

Synonyms

- Ethane trichloride
- β-Trichloroethane
- 1,2,2-Trichloroethane
- Vinyl trichloride

Last updated: 17 November 1997

COBALT AND COBALT COMPOUNDS
(Group 2B)

For definition of Groups, see Preamble Evaluation.

VOL.: 52 (1991) (p. 363)

Cobalt
CAS No.: 7440-48-4

Cobalt-chromium alloy
CAS No.: 11114-92-4

Nickel-based cobalt alloy
CAS No.: 11068-91-0

Cobalt-chromium-nickel-tungsten alloy
CAS No.: 12638-07-2

Cobalt-chromium-molybdenum alloy
CAS No.: 12629-02-6

Cobalt[II] acetate
CAS No.: 71-48-7

Cobalt[II] acetate tetrahydrate
CAS No.: 6147-53-1

Cobalt[III] acetate
CAS No.: 917-69-1

Cobalt[II] carbonate
CAS No.: 513-79-1

Cobalt[II] carbonate hydroxide (1:1)
CAS No.: 12069-68-0

Cobalt[II] carbonate hydroxide (2:3)
CAS No.: 12602-23-2

Cobalt[II] carbonate hydroxide (2:3) monohydrate
CAS No.: 51839-24-8

Cobalt[II] chloride
CAS No.: 7646-79-9

Cobalt[II] chloride hexahydrate
CAS No.: 7791-13-1

Cobalt[II] hydroxide
CAS No.: 21041-93-0

Cobalt[III] hydroxide
CAS No.: 1307-86-4

Cobalt[II] naphthenate
CAS No.: 61789-51-3

Cobalt[II] nitrate
CAS No.: 10141-05-6

Cobalt[II] nitrate hexahydrate
CAS No.: 10026-22-9

Cobalt[II] molybdenum[VI] oxide
CAS No.: 13762-14-6

Cobalt[II] oxide
CAS No.: 1307-96-6

Cobalt[II,III] oxide
CAS No.: 1308-06-1

Cobalt[III] oxide
CAS No.: 1308-04-9

Cobalt[III] oxide monohydrate
CAS No.: 12016-80-7

Cobalt[II] sulfate
CAS No.: 10124-43-3

Cobalt sulfide
CAS No.: 1317-42-6

Dicobalt octacarbonyl
CAS No.: 10210-68-1

Tetracobalt dodecacarbonyl
CAS No.: 17786-31-1

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Cobalt is widely distributed in the environment; it is the thirty-third most abundant element in the earth's crust. Cobalt is obtained primarily as a by-product of the mining and processing of copper and nickel ores and is a constituent of about 70 naturally occurring oxide, sulfide, arsenide and sulfoarsenide minerals. Cobalt is extracted from ore and concentrated by pyrometallurgical, hydrometallurgical and electrolytic processes alone or in combination. Refined metallic cobalt is available to the industrial market as cathodes and to a lesser extent as powders; oxides and other compounds are also available.

Cobalt compounds have been used as pigments in glass and ceramics in many countries for thousands of years. Since the beginning of the twentieth century, the major uses of cobalt have been in the production of metal alloys, such as superalloys and magnetic alloys, as well as high-strength steels and hard-metal cemented carbides. At the end of the 1980s, about one-third of the cobalt used was in the production of cobalt chemicals, which are used primarily as catalysts and pigments.

The main route of occupational exposure is *via* the respiratory tract by inhalation of dusts, fumes and mists containing cobalt. Exposures have been measured in hard-metal production, processing and use and in porcelain painting. Occupational exposure to cobalt is regulated in many countries.

Cobalt occurs in vegetables *via* uptake from soil, and vegetables account for the major part of human dietary intake of cobalt. Animal-derived foods, particularly liver, contain cobalt in the form of vitamin B_{12}. Cobalt is also found in air, water and tobacco smoke. Human tissues and fluids normally contain low levels of cobalt, which may be increased as a result of occupational exposures. Cobalt concentrations in tissue, serum and urine can be increased in patients with implants made of cobalt-containing alloys.

Cobalt-containing particles have been detected in tissues immediately adjacent to such prostheses.

5.2 Experimental carcinogenicity data

Cobalt metal powder was tested in two experiments in rats by intramuscular injection and in one experiment by intrathoracic injection, producing sarcomas at the injection site.

A finely powdered *cobalt-chromium-molybdenum alloy* was tested in rats by intramuscular injection, producing sarcomas at the injection site. In two other experiments in rats, coarsely or finely ground cobalt-chromium-molybdenum alloy implanted in muscle or pellets of cobalt-chromium-molybdenum alloy implanted subcutaneously did not induce sarcomas. Implantation in the rat femur of three different *cobalt-containing alloys*, in the form of powder, rod or compacted wire, resulted in a few local sarcomas. In another experiment, intramuscular implantation of polished rods consisting of three different cobalt-containing alloys did not produce local sarcomas. In an experiment in guinea-pigs, intramuscular implantation of a *cobalt-chromium-molybdenum alloy* powder did not produce local tumours.

Intraperitoneal injection of a *cobalt-chromium-aluminium spinel* in rats produced a few local malignant tumours, and intratracheal instillation of this spinel in rats was associated with the occurrence of a few pulmonary squamous-cell carcinomas.

In two experiments in rats, intramuscular injection of *cobalt[II] oxide* powder produced sarcomas at the injection site. In an experiment in mice, intramuscular injection of cobalt oxide powder did not produce local tumours. Intratracheal instillation of cobalt oxide powder in rats was associated with a few benign and malignant pulmonary tumours. In a study limited by poor survival, hamsters administered a cobalt oxide dust by inhalation showed no increase in the incidence of pulmonary tumours. In two experiments in rats by subcutaneous and intraperitoneal injection, cobalt oxide powder produced local malignant tumours.

Cobalt[II] sulfide powder was tested in one study in rats by intramuscular injection, producing a high incidence of local sarcomas.

Cobalt[II] chloride was tested in one study in rats by repeated subcutaneous injection, producing many local and a few distant subcutaneous sarcomas.

Cobalt[II,III] oxide was tested in one experiment in hamsters to determine the effects of various particulates on carcinogenesis induced by *N*-nitrosodiethylamine. Intratracheal instillation of cobalt[II,III] oxide did not increase the incidence of pulmonary tumours over that in appropriate control groups.

Studies in mice and rabbits with *cobalt naphthenate* could not be evaluated.

In a screening test for lung adenomas by intraperitoneal injection, *cobalt[III] acetate* did not increase the incidence of lung tumours in strain A mice.

Interpretation of the available evidence for the carcinogenicity of cobalt in experimental animals was difficult because many of the reports failed to include sufficient details on results of statistical analyses, on survival and on control groups. Further, statistical analyses could not be performed by the Working Group in the absence of specific information on survival and on whether the neoplasms were fatal. Nevertheless, weight was given in the evaluation to the consistent occurrence of tumours at the site of administration and to the histological types of tumours observed.

5.3 Human carcinogenicity data

A number of single cases of malignant tumours, mostly sarcomas, have been reported at the site of orthopaedic implants containing cobalt. In one cohort study of people with a hip prosthesis, there was a significant increase in the incidence of lymphatic and haematopoietic malignancies, and significant deficits of breast and colorectal cancers. Overall cancer incidence was significantly lower than expected in the first 10 years after surgery, but significantly higher than expected after 10 or more years. No data were provided on the composition of the prostheses in this study.

Four cohort studies on the association between industrial exposure to cobalt and death from cancer were reviewed, two of which provided information for the evaluation. In a French electrochemical plant, there was a significant increase in the risk for lung cancer among workers in cobalt production, who were also exposed to nickel and arsenic, but not among workers in other departments of the factory. In a study in Sweden of hard-metal workers with documented exposure to cobalt-containing dusts, a significant increase in lung cancer risk was seen in persons exposed for more than 10 years whose exposure had begun more than 20 years previously.

Interpretation of the available evidence on the possible association between occupational exposure to cobalt and cancer in humans is made difficult by the fact that in three of the four studies there was concurrent exposure to other potentially carcinogenic substances, including forms of nickel and arsenic. In the Swedish study, there was concurrent exposure to other components of hard-metal dust.

5.4 Other relevant data

Occupational exposure to cobalt-containing dusts can cause fibrotic changes in the lung and can precipitate asthma. Cardiotoxic effects have been reported in exposed humans; in particular, cardiomyopathy can occur after prolonged oral intake.

Cobalt[II] chloride reduced fertility in male mice.

Cobalt[II] compounds had weak or no genetic effect in bacteria; some cobalt[III] complexes with heterocyclic ligands were active.

In single studies with an extensive range of eukaryotes, including animal and human cells *in vitro*, cobalt[II] compounds induced DNA damage, mutation, sister chromatid exchange and aneuploidy. Gene conversion and mutation in eukaryotes and DNA damage in human cells were observed in several studies. There was some evidence that these compounds can also induce aneuploidy in hamsters *in vivo*. In single studies, cobalt[II] sulfide induced DNA damage and transformation in cultured mammalian cells.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of cobalt and cobalt compounds in humans.

There is *sufficient evidence* for the carcinogenicity of cobalt metal powder in experimental animals.

There is *limited evidence* for the carcinogenicity of metal alloys containing cobalt, chromium and molybdenum in experimental animals.

There is *sufficient evidence* for the carcinogenicity of cobalt[II] oxide in experimental animals.

There is *limited evidence* for the carcinogenicity of cobalt[II] sulfide in experimental animals.

There is *limited evidence* for the carcinogenicity of cobalt[II] chloride in experimental animals.

There is *inadequate evidence* for the carcinogenicity of cobalt-aluminium-chromium spinel, cobalt[II,III] oxide, cobalt naphthenate and cobalt[III] acetate in experimental animals.

Overall evaluation

Cobalt and cobalt compounds are *possibly carcinogenic to humans (Group 2B)*.

For definition of the italicized terms, see Preamble Evaluation.

Synonyms for Cobalt

- C.I. 77320
- Cobalt element
- Cobalt-59

Synonyms for Cobalt-chromium alloy

- Cobalt alloy [nonbase], Co, Cr
- Chromium alloy (nonbase), Co, Cr

Synonyms for Nickel-based cobalt alloy

- APK 1
- Astroloy
- Cabot 700
- Nickel alloy [base] Ni47-59, Co 17-20, Cr 13-17, Mo 4.5-5.7, Al 3.7-4.7, Ti 3-4, Fe 0-1, C 0-0.1 (AISI 687)
- Nimonic AP1
- NiCo18Cr15MoAlTi
- NK17CADT
- PM-ATS 380
- PWA 1013
- R 77
- Rene 77
- U 700m
- U700PM
- Udimet 700

Synonyms for Cobalt-chromium-nickel-tungsten alloy

- AFNOR K-C25NW
- AMS 5382
- Cobalt alloy [base] Co 48-58, Cr 24-26, Ni 9.5-12, W 7-8, Fe 2, Mn 0-1, Si 0-1, C 0.4-0.6 (ASTM A567-2)
- Co X-40
- CoCrNiW5525
- G-X 55
- 31H114
- Haynes Stellite 31
- HS 31
- KC25NW
- MAS 5382
- PN 31H114
- S-31
- Stellite 31
- Stellite 31 X 40
- Stellite X40
- X 40
- 45VF

Synonyms for Cobalt-chromium-molybdenum alloy

- Cobalt alloy [base], [Co 56-68; Cr 25-29, Mo 5-6, Ni 1.8-3.8, Fe 0-3, Mn 0-1, Si 0-1, C 0.2-0.3]
- Akrit CoMo35
- AMS 5385D
- Celsit 290
- F 75
- Haynes Stellite 21
- HS 21
- Protasul-2
- Stellite 21
- Vinertia
- Vitallium
- Zimalloy

Synonyms for Cobalt[II] acetate

- Acetic acid, cobalt(2+) salt
- Bis(acetato)cobalt
- Cobalt acetate
- Cobalt(2+) acetate
- Cobalt diacetate
- Cobaltous acetate
- Cobaltous diacetate

Synonym for Cobalt[II] acetate tetrahydrate

- Bis(acetato)tetraquacobalt

Synonyms for Cobalt[III] acetate

- Acetic acid, cobalt(3+) salt
- Cobalt(3+) acetate
- Cobaltic acetate
- Cobalt triacetate

Synonyms for Cobalt[II] carbonate

- Carbonic acid, cobalt(2+) salt (1:1)
- C.I. 77353
- Cobalt carbonate (1:1)
- Cobalt(2+) carbonate
- Cobalt monocarbonate
- Cobaltous carbonate

Synonyms for Cobalt[II] carbonate hydroxide (1:1)

- Basic cobalt carbonate
- Carbonic acid, cobalt complex
- Cobalt carbonate hydroxide
- Cobalt(carbonato)dihydroxidi-
- Cobalt, [mu.-[carbonato(2-)-0:0']]dihydroxydi-

Synonyms for Cobalt[II] carbonate hydroxide (2:3)

- Cobalt, bis(carbonato(2-))-hexahydroxypenta-
- Cobalt, bis(carbonato)hexahydroxypenta-
- Cobalt carbonate hydroxide
- Cobalt hydroxide carbonate

Synonyms for Cobalt[II] carbonate hydroxide (2:3) monohydrate

- Basic cobalt carbonate
- Carbonic acid, cobalt(2+) salt, basic
- Cobalt, bis(carbonato(2-))hexahydroxypentamonohydrate
- Cobaltous carbonate, basic

Synonyms for Cobalt[II] chloride

- Cobalt chloride [$COCL_2$]
- Cobalt dichloride
- Cobaltous chloride

Synonyms for Cobalt[II] chloride hexahydrate

- Cobalt chloride, hexahydrate
- Cobalt dichloride hexahydrate
- Cobaltous chloride hexahydrate

Synonyms for Cobalt[II] hydroxide

- Cobalt dihydroxide
- Cobalt hydroxide [$Co(OH)_2$]
- Cobalt(2+) hydroxide
- Cobaltous hydroxide

Synonyms for Cobalt[III] hydroxide

- Cobalt hydroxide [$Co(OH)_3$]
- Cobaltic hydroxide
- Cobalt trihydroxide

Synonyms for Cobalt[II] naphthenate

- Cobalt Nap-All
- Cobalt naphthenates
- Naftolite
- Naphthenic acid, cobalt salt
- Naphthenic acids, cobalt salts
- Naphthex Co
- 85N-Co

Synonyms for Cobalt[II] nitrate

- Cobalt bis(nitrate)
- Cobalt(2+) nitrate
- Cobaltous nitrate
- Nitric acid, cobalt(2+) salt

Synonyms for Cobalt[II] nitrate hexahydrate

- Cobalt dinitrate hexahydrate
- Cobalt nitrate hexahydrate
- Cobalt(2+) nitrate hexahydrate
- Cobalt(II) nitrate hydrate
- Cobaltous nitrate hexahydrate
- Nitric acid, cobalt(2+) salt, hexahydrate

Synonyms for Cobalt[II] molybdenum[VI] oxide

- Cobalt molybdate
- Cobalt molybdate[VI]
- Cobalt(2+) molybdate
- Cobalt molybdenum oxide [$CoMoO_4$]
- Cobaltous molybdate
- Cobalt monomolybdate
- Molybdenum cobaltate
- Molybdenum cobalt oxide
- Molybdic acid [H_2MoO_4], cobalt(2) salt (1:1)

Cobalt[II] oxide

- C.I. 77322
- C.I. Pigment black 13
- Cobalt black
- Cobalt monoxide
- Cobalt oxide [CoO]
- Cobalt(2+) oxide
- Cobaltous oxide
- Monocobalt oxide

Synonyms for Cobalt[II,III] oxide

- Cobalt oxide [Co_3O_4]
- Cobaltic-cobaltous oxide
- Cobalto-cobaltic oxide
- Cobalto-cobaltic tetroxide
- Cobaltosic oxide
- Cobalt tetraoxide
- Tricobalt tetraoxide
- Tricobalt tetroxide

Synonyms for Cobalt[III] oxide

- C.I. 77323
- Cobalt oxide [Co_2O_3]
- Cobaltic oxide
- Cobalt(3+) oxide
- Cobalt peroxide
- Cobalt sesquioxide
- Cobalt trioxide
- Dicobalt oxide
- Dicobalt trioxide

Synonyms for Cobalt[III] oxide monohydrate

- Cobalt hydroxide oxide [Co(OH)O]
- Cobalt[III] hydroxide oxide
- Cobalt oxide hydroxide
- Cobalt oxyhydroxide

Synonyms for Cobalt[II] sulfate

- Cobalt monosulfate
- Cobaltous sulfate
- Cobalt sulfate (1:1)
- Cobalt(2+) sulfate
- Cobalt sulphate
- Sulfuric acid, cobalt(2+) salt (1:1)

Synonyms for Cobalt sulfide

- Cobalt monosulfide
- Cobaltous sulfide
- Cobalt(2+) sulfide

Synonyms for Dicobalt octacarbonyl

- Cobalt, di-.mu.-carbonylhexacarbonyldi-
- Cobalt tetracarbonyl dimer

Synonym for Tetracobalt dodecacarbonyl

- Cobalt, tri-.mu.-carbonylnonacarbonyltetra-

Last updated: 17 November 1997