



WORLD HEALTH ORGANIZATION
INTERNATIONAL AGENCY FOR RESEARCH ON CANCER

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans

Volume 47
**Some Organic Solvents, Resin Monomers and Related
Compounds,
Pigments and Occupational Exposures in Paint
Manufacture
and Painting**

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SOME PETROLEUM SOLVENTS (Group 3)

For definition of Groups, see [Preamble Evaluation](#).

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5. Summary of Data Reported and Evaluation

5.1 Exposure

Petroleum solvents are hydrocarbon mixtures which can be grouped into three broad categories on the basis of their boiling ranges and solvent strengths, as follows: special boiling range solvents, boiling range, 30-160 °C; white spirits, 130-220 °C; and high-boiling aromatic solvents, 160-300 °C. Within these broad solvent categories, individual solvents (typically boiling within narrower ranges of 15-30 °C) are composed of aliphatic, alicyclic and aromatic hydrocarbons in varying amounts, depending on refining process and end use. Although the content of benzene in petroleum solvents is now generally less than 1% in nonhydrogenated special boiling range solvents and less than 0.1% in other solvents, higher amounts were commonly present in the past.

Exposure to petroleum solvents is widespread in many occupations, including painting, printing, use of adhesives, rubber processing and degreasing. High exposure levels have been measured in many of these occupational environments.

5.2 Experimental carcinogenicity data

A single study in rats exposed by inhalation to a high-boiling aromatic solvent was of insufficient duration to allow an evaluation of carcinogenicity.

5.3 Human carcinogenicity data

In a single case-control study of cancer at many sites, potential long, high exposure to 'mineral spirits' was associated with increased risks for squamous-cell lung cancer and prostatic cancer. In two case-control studies, one of primary liver cancer and one of Hodgkin's disease, an association with organic solvents, including white spirits, was seen. The results of these studies could not be evaluated with regard to petroleum solvents themselves.

5.4 Other relevant data

In humans, petroleum solvents cause nonallergic contact dermatitis and adverse effects on the central nervous system.

In experimental animals, samples of petroleum solvents with a high aromatic content had greater acute toxicity and were more irritating than those that were virtually aromatic-free. A special boiling range solvent containing *n*-hexane induced chronic toxicity in the peripheral nervous system of experimental animals.

In two studies of malformations in the children of women who had been exposed to petroleum solvents during the first trimester of pregnancy, the numbers of cases were small and the mothers had also been exposed to other substances.

A rubber solvent (special boiling range solvent) induced chromosomal aberrations but not sister chromatid

exchange in cultured human cells. Another special boiling range solvent did not induce chromosomal aberrations in cultured mammalian cells, gene conversion in yeast or mutation in bacteria. A sample of white spirits did not induce chromosomal aberrations in mice *in vivo*, sister chromatid exchange in human cells or mutation in bacteria.

5.5 Evaluation

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

There is *inadequate evidence* for the carcinogenicity of high-boiling aromatic solvents in experimental animals.

No data were available on the carcinogenicity of special boiling range solvents or white spirits in experimental animals.

Overall evaluation

Petroleum solvents are *not classifiable as to their carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see [Preamble Evaluation](#).

Synonyms

Special boiling-range solvents

- Benzine
- Canadol
- Clairsol
- Essence
- Exxsol DSP
- Halpasol
- High-boiling petroleum ether
- Hydrosol
- Indusol
- Lacquer diluent
- Light ligroin
- Ligroin
- Naphtha
- Naphtha 76
- Petroleum benzin
- Petroleum ether
- Refined solvent naphtha
- Rubber solvent
- SBP
- Shellsol
- Solfina
- Special boiling-point solvents
- Special naphtholite
- Spezialbenzine
- Varnish makers' and painters' naphtha
- VM & P naphtha

White spirits

- B.A.S.

- C.A.S.
- DAWS
- Dearomatized white spirits
- Dilutine
- Exxsol D
- 140 Flash solvent
- Halpasol
- HAWS
- High aromatic white spirits
- Hydrosol
- Indusol
- Kristalloel
- Lacquer petrol
- LAWS
- Light petrol
- Low aromatic white spirits
- Mineral solvent
- Mineral spirits
- Mineral turpentine
- Odourless mineral spirit
- Petroleum spirits
- Sangajol
- Shellsol D
- Solfina
- Solnap
- Solvent naphtha
- Spirdane
- Stoddard solvent
- Terpentina
- Tetrasol
- Turpentine substitute
- Varsol

High-boiling aromatic solvents

- A-100
- A-150
- Caromax
- Hydrosol
- Indusol
- Naphtha
- Shellsol
- Solvantar
- Solvarex
- Solvesso

CYCLOHEXANONE

(Group 3)

For definition of Groups, see [Preamble Evaluation](#).

VOL.: 47 (1989) (p. 157)

CAS No.: 108-94-1

Chem. Abstr. Name: Cyclohexanone

5. Summary of Data Reported and Evaluation

5.1 Exposures

Cyclohexanone is a synthetic organic liquid used primarily as an intermediate in the production of nylon. Other minor applications are as an intermediate, additive and solvent in a variety of products. Occupational exposure levels have been measured in some industries.

5.2 Experimental carcinogenicity data

Cyclohexanone was tested for carcinogenicity by oral administration in the drinking-water in one strain of mice and one strain of rats. In mice, there was a slight increase in the incidence of tumours that occur commonly in this strain, only in animals given the low dose. In rats, a slight increase in the incidence of adrenal cortical adenomas occurred in males treated with the low dose.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

No significant systemic toxicity was reported in humans or experimental animals. No significant prenatal toxicity was observed in mice.

Cyclohexanone induced chromosomal aberrations and ploidy changes in cultured human cells and in rats. It did not induce mutation in bacteria.

5.5 Evaluation

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

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

Overall evaluation

Cyclohexanone 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

- Anon
 - Anone
 - Hexanon
 - Hytrol O
 - Ketoexamethylene
 - Nadone
 - Pimelic ketone
 - Pimelin ketone
 - Sextone
-

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MORPHOLINE

(Group 3)

For definition of Groups, see [Preamble Evaluation](#).

VOL.: 47 (1989) (p. 199)

CAS No.: 110-91-8

Chem. Abstr. Name: Morpholine

5. Summary of Data Reported and Evaluation

5.1 Exposures

Morpholine is a synthetic organic liquid used mainly as an intermediate in the production of rubber chemicals and optical brighteners, as a corrosion inhibitor in steam condensate systems, as an ingredient in waxes and polishes and as a component of protective coatings on fresh fruits and vegetables. Occupational exposure may occur during the production of morpholine and in its various uses, but data on exposure levels are sparse. It has been detected in samples of foodstuffs and beverages.

5.2 Experimental carcinogenicity data

Morpholine was tested for carcinogenicity by oral administration in two strains of mice, one strain of rats and one strain of hamsters. The studies in one of the strains of mice and in hamsters were considered inadequate for evaluation. In the other strain of mice, no significant increase in the incidence of tumours was seen in treated animals. In the study in rats, a few tumours of the liver and lung occurred in treated animals. Morpholine was also tested by inhalation exposure in rats; it did not increase the incidence of tumours over that in controls.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

Morpholine is an irritant in humans and experimental animals. It caused kidney damage in experimental animals.

Morpholine did not induce micronuclei, chromosomal aberrations or mutation in hamsters. It did not induce morphological transformation, chromosomal aberrations or DNA damage in cultured animal cells. It did not induce mutations in bacteria.

5.5 Evaluation

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

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

Morpholine 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

- BASF 238
 - Diethylene imidoxide
 - Diethylene oximide
 - Diethylenimide oxide
 - Drewamine
 - 1-Oxa-4-azacyclohexane
 - Tetrahydro-*para*-isoxazine
 - Tetrahydro-1,4-isoxazine
 - Tetrahydro-1,4-oxazine
 - Tetrahydro-(2*H*)-1,4-oxazine
 - Tetrahydro-(4*H*)-1,4-oxazine
 - Tetrahydro-*para*-oxazine
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BIS(2,3-EPOXYCYCLOPENTYL)ETHER (Group 3)

For definition of Groups, see [Preamble Evaluation](#).

VOL.: 47 (1989) (p. 231)

CAS No.: 2386-90-5

Chem. Abstr. Name: 2,2'-Oxybis(6-oxabicyclo[3.1.0]hexane)

5. Summary of Data Reported and Evaluation

5.1 Exposures

Bis(2,3-epoxycyclopentyl)ether is a synthetic organic liquid which has been used as a component and modifier of epoxy resins. Measurements of occupational exposure levels have not been reported.

5.2 Experimental carcinogenicity data

Bis(2,3-epoxycyclopentyl)ether was tested for carcinogenicity by skin application in one experiment in two strains of mice, producing a small number of skin tumours in both strains; an increased incidence of lung tumours was observed in females of one strain. Another experiment by skin application in mice was inadequate for evaluation.

5.3 Human carcinogenicity data

No data were available to the Working Group.

5.4 Other relevant data

Bis(2,3-epoxycyclopentyl)ether induced sister chromatid exchange in cultured human cells and micronuclei in mice. It was mutagenic to bacteria.

5.5 Evaluation

There is *limited evidence* for the carcinogenicity of bis(2,3-epoxycyclopentyl)ether in experimental animals.

No data were available from studies in humans on the carcinogenicity of bis(2,3-epoxycyclopentyl)ether.

Overall evaluation

Bis(2,3-epoxycyclopentyl)ether 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

- ERL^R 4205
 - W95
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SOME GLYCIDYL ETHERS

Phenyl glycidyl ether (Group 2B)

Bisphenol A diglycidyl ether (Group 3)

For definition of Groups, see [Preamble Evaluation](#).

VOL.: 47 (1989) (p. 237)

Bisphenol A diglycidyl ether

CAS No.: 1675-54-3

Chem. Abstr. Name: 2,2'-[(1-Methylethylidene)bis(4,1-phenyleneoxymethylene)]bis(oxirane)

Phenyl glycidyl ether

CAS No.: 122-60-1

Chem. Abstr. Name: (Phenoxyethyl)oxirane

5. Summary of Data Reported and Evaluation

5.1 Exposures

Glycidyl ethers are basic components of epoxy resins which have been commercially available since the late 1940s. Bisphenol A diglycidyl ether and its oligomers are major components of epoxy resins. Other glycidyl ethers, including phenyl glycidyl ether, are frequently incorporated into epoxy resin systems as reactive modifiers. Epoxy resins based on bisphenol A diglycidyl ether are widely used in protective coatings, including paints, in reinforced plastic laminates and composites, in tooling, casting and moulding resins, in bonding materials and adhesives, and in floorings and aggregates. Occupational exposure to bisphenol A diglycidyl ether and phenyl glycidyl ether may occur during their production, during the production of epoxy products and during various uses of epoxy products, but data on exposure levels are sparse.

5.2 Experimental carcinogenicity data

Bisphenol A diglycidyl ether of various technical grades was tested by skin application in mice in five studies. In one of the studies, an increased incidence of epidermal tumours was found in one of two strains tested. In another study, a small increase in the incidence of epidermal tumours and small increases in the incidences of kidney tumours in male mice and of lymphoreticular/haematopoietic tumours in female mice were observed. No increase in the incidence of skin tumours was observed in two further studies, and the other study was inadequate for evaluation. Following subcutaneous injection of technical-grade bisphenol A diglycidyl ether to rats, a small number of local fibrosarcomas was observed. Following application of technical-grade bisphenol A diglycidyl ether to the skin of rabbits, no skin tumour was observed.

Pure bisphenol A diglycidyl ether was tested in one experiment by skin application in mice; no epidermal but a few dermal tumours were observed in males, and there was a small increase in the incidence of lymphoreticular/haematopoietic tumours in females.

Pure phenyl glycidyl ether was tested for carcinogenicity by inhalation exposure in male and female rats of one strain, producing carcinomas of the nasal cavity in animals of each sex.

5.3 Human data

No data were available to the Working Group.

5.4 Other relevant data

Some glycidyl ethers have been shown to cause allergic contact dermatitis in humans. Glycidyl ethers generally cause skin sensitization in experimental animals. Necrosis of the mucous membranes of the nasal cavities was induced in mice exposed to allyl glycidyl ether.

Prenatal toxicity was not induced in rats exposed by inhalation to phenyl glycidyl ether or in rabbits exposed dermally to bisphenol A diglycidyl ether.

One study of workers exposed to bisphenol A diglycidyl ether showed no increase in the incidence of chromosomal aberrations in peripheral lymphocytes. A study of workers with mixed exposures was inconclusive with regard to the effects of specific glycidyl ethers. Phenyl glycidyl ether, but not *n*-butyl glycidyl ether, induced morphological transformation in mammalian cells *in vitro*. *n*-Butyl glycidyl ether induced micronuclei in mice *in vivo* following intraperitoneal but not oral administration. Phenyl glycidyl ether did not induce micronuclei or chromosomal aberrations *in vivo* or chromosomal aberrations in animal cells *in vitro*. Alkyl C₁₂ or C₁₄ glycidyl ether did not induce DNA damage in cultured human cells or mutation in cultured animal cells. Allyl glycidyl ether induced mutation in *Drosophila*. The glycidyl ethers were generally mutagenic to bacteria.

5.5 Evaluation

There is *sufficient evidence* for the carcinogenicity of phenyl glycidyl ether in experimental animals.

There is *limited evidence* for the carcinogenicity of bisphenol A diglycidyl ether in experimental animals.

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

Overall evaluation

Phenyl glycidyl ether is *possibly carcinogenic to humans (Group 2B)*.

Bisphenol A diglycidyl ether is *not classifiable as to its carcinogenicity to humans (Group 3)*.

For definition of the italicized terms, see [Preamble Evaluation](#).

Subsequent evaluation: [Vol. 71 \(1999\) \(Bisphenol A diglycidyl ether\)](#); [\(Phenyl glycidyl ether\)](#)

Synonyms for Bisphenol A diglycidyl ether

- Araldite 6005
- Araldite^R GY 250
- Araldite^R GY 6010
- Bis(4-glycidyloxyphenyl)dimethylmethane
- 2,2-Bis(4-glycidyloxyphenyl)propane
- 2,2-Bis(4-hydroxyphenyl)propane diglycidyl ether
- 2,2-Bis(*para*-glycidyloxyphenyl)propane
- 2,2-Bis(*para*-hydroxyphenyl)propane diglycidyl ether
- 2,2-Bis[4-(2,3-epoxypropoxy)phenyl]propane
- 2,2-Bis[*para*-(2,3-epoxypropoxy)phenyl]propane
- 4,4'-Bis(2,3-epoxypropoxy)diphenyldimethylmethane

- 4,4'-Dihydroxydiphenyldimethylmethane diglycidyl ether
- 4,4'-Isopropylidenebis[1-(2,3-epoxypropoxy)benzene]
- 4,4'-Isopropylidenediphenol diglycidyl ether
- Bis(4-hydroxyphenyl)dimethylmethane diglycidyl ether
- BPDGE
- D.E.R.^R 331
- Dian diglycidyl ether
- Diglycidyl bisphenol A
- Diglycidyl bisphenol A ether
- Diglycidyl diphenylolpropane ether
- Diglycidyl ether of 2,2-bis-(4-hydroxyphenyl)propane
- Diglycidyl ether of 2,2-bis-(*para*-hydroxyphenyl)propane
- Diglycidyl ether of 4,4'-isopropylidenediphenol
- Diglycidyl ether of bisphenol A
- *para,para'*-Dihydroxydiphenyldimethylmethane diglycidyl ether
- Diomethane diglycidyl ether
- EPI-REZ^R 510
- Epikote^R 815
- Epikote^R 828
- EPON^R 828
- EPOTUF^R 37-140
- Epoxide A
- Oligomer 340

Synonyms for Phenyl glycidyl ether

- 1,2-Epoxy-3-phenoxypropane
- 2,3-Epoxypropoxybenzene
- 2,3-Epoxypropyl phenyl ether
- Glycidol phenyl ether
- Glycidyl phenyl ether
- Heloxy^R WC 63
- PGE
- Phenol glycidyl ether
- 1-Phenoxy-2,3-epoxypropane
- 3-Phenoxy-1,2-epoxypropane
- Phenoxypropene oxide
- Phenoxypropylene oxide
- -Phenoxypropylene oxide
- 3-Phenoxy-1,2-propylene oxide
- Phenyl 2,3-epoxypropyl ether
- 3-Phenyloxy-1,2-epoxypropane

ANTIMONY TRIOXIDE AND ANTIMONY TRISULFIDE

Antimony trioxide (Group 2B)

Antimony trisulfide (Group 3)

For definition of Groups, see [Preamble Evaluation](#).

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Antimony trioxide

CAS Nos: 1309-64-4 - Antimony oxide; 1317-98-2 - Valentinite; 12412-52-1 - Senarmontite

Chem. Abstr. Name: Antimony oxide

Antimony trisulfide

CAS Nos: 1345-04-6 - Antimony sulfide; 1317-86-8 - Stibnite

Chem. Abstr. Name: Antimony sulfide

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Antimony trioxide is produced from stibnite ores (antimony trisulfide) or as a by-product of lead smelting and production. It is used mainly in fire-retardant formulations for plastics, rubbers, textiles, paper and paints. It is also used as an additive in glass and ceramic products and as a catalyst in the chemical industry.

Occupational exposure may occur during mining, processing and smelting of antimony ores, in glass and ceramics production, and during the manufacture and use of products containing antimony trioxide.

Antimony trisulfide is used in the production of explosives, pigments, antimony salts and ruby glass. Occupational exposure may occur during these processes and also during the mining, processing and smelting of ores containing antimony trisulfide.

5.2 Experimental carcinogenicity data

Antimony trioxide was tested for carcinogenicity by inhalation exposure in male and female rats of one strain and in female rats of another strain, producing a significant increase in the incidence of lung tumours (scirrhous and squamous-cell carcinomas and bronchioloalveolar tumours) in females in both studies. No lung tumour was seen in male rats.

Antimony ore concentrate (mainly antimony trisulfide) was tested for carcinogenicity by inhalation exposure in male and female rats of one strain, producing a significant increase in the incidence of lung tumours (scirrhous and squamous-cell carcinomas and bronchioloalveolar tumours) in females. No lung tumour was seen in males.

5.3 Human carcinogenicity data

The available data were inconclusive.

5.4 Other relevant data

Antimony trioxide causes pneumoconiosis in humans. One study of women exposed to dusts containing metallic antimony, antimony trioxide and antimony pentasulfide suggested that they may have had an excess incidence of premature births and spontaneous abortions and that their children's growth may have been retarded.

Antimony trioxide induced DNA damage in bacteria.

5.5 Evaluation

There is *inadequate evidence* for the carcinogenicity of antimony trioxide and antimony trisulfide in humans.

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

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

Overall evaluations

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

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

For definition of the italicized terms, see [Preamble Evaluation](#).

Synonyms for Antimony trioxide

- A 1582
- A 1588 LP
- Amspec-KR
- Antimonious oxide
- Antimony sesquioxide
- Antimony white
- Antimony[III] oxide
- Anzon-TMS
- AP 50
- Asarco antimony oxide [LT, HT, VHT]
- Blue Star
- CI Pigment White 11
- Dechlorane A-O
- Diantimony trioxide
- Exitelite
- Extrema
- Flowers of antimony
- Laurel Fire Shield
- Senarmontite
- Thermoguard
- Thermoguard S
- Twinkling Star
- Valentinite
- White Star

Synonyms for Antimony trisulfide

- Antimonous sulfide

- Antimony glance
 - Antimony needles
 - Antimony orange
 - Antimony sesquisulfide
 - Antimony trisulfide colloid
 - Antimony vermilion
 - Black antimony
 - CI Pigment Red 107
 - Crimson antimony sulfide
 - Diantimony trisulfide
 - Lymphoscan
 - Needle antimony
 - Stibnite
-

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TITANIUM DIOXIDE (Group 3)

For definition of Groups, see [Preamble Evaluation](#).

VOL.: 47 (1989) (p. 307)

CAS Nos.: 13463-67-7 - Titanium dioxide; 1317-70-0 - Anatase titanium dioxide; 1317-80-2 - Rutile titanium dioxide

Chem. Abstr. Name: Titanium dioxide

5. Summary of Data Reported and Evaluation

5.1 Exposures

Titanium dioxide is a white pigment produced mainly from ilmenite (iron titanate) and natural rutile (titanium dioxide). It is widely used in paints, paper, plastics, ceramics, rubber, inks and a variety of other products. Occupational exposure to titanium dioxide during its production, the production of paints, in painting trades and during other industrial use is likely to be extensive, but there is a paucity of data on levels, both occupational and environmental.

5.2 Experimental carcinogenicity data

Titanium dioxide was tested for carcinogenicity by oral administration in one strain of mice and in one strain of rats, by inhalation in two strains of rats, by intratracheal administration in one strain of hamsters, by subcutaneous injection in one strain of rats and by intraperitoneal administration in one strain of male mice and two strains of female rats. Increased incidences of lung adenomas in rats of both sexes and of cystic keratinizing lesions diagnosed as squamous-cell carcinomas in female rats were observed in animals that had inhaled the high but not the low doses of titanium dioxide. Oral, subcutaneous, intratracheal and intraperitoneal administration did not produce a significant increase in the frequency of any type of tumour in any species. Intratracheal administration of titanium dioxide in combination with benzo[*a*]pyrene to hamsters resulted in an increase in the incidence of benign and malignant tumours of the larynx, trachea and lungs over that in benzo[*a*]pyrene-treated controls.

5.3 Human carcinogenicity data

The only available epidemiological study provided inconclusive results.

5.4 Other relevant data

Titanium dioxide did not induce morphological transformation in mammalian cells or mutation in bacteria.

5.5 Evaluation

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

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

Overall evaluation

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

For definition of the italicized terms, see [Preamble Evaluation](#).

Synonyms

- A-fil Cream
- Atlas white titanium dioxide
- Austiox
- Bayertitan
- C-Weiss 7
- Calcotone White T
- Cosmetic White C47-5175
- Cosmetic White C47-9623
- E 171
- Flamenco
- Hombitan
- Horse Head A-410
- Horse Head A-420
- Horse Head R-710
- KH 360
- Kronos titanium dioxide
- Levnox White RKB
- NCI-CO4240
- Pigment White 6
- Rayox
- Runa RH20
- Rutile
- Tichlor
- Tiofine
- Tiona T.D.
- Tioxide
- Tipaque
- Ti-Pure
- Titafrance
- Titandioxid
- Titania
- Titanium[IV] oxide
- Titanox
- Titanox 2010
- Trioxide(s)
- Tronox
- Unitane products [various]
- 1700 White
- Zopaque

OCCUPATIONAL EXPOSURES IN PAINT MANUFACTURE AND PAINTING

Occupational exposure as a painter (Group 1)

Occupational exposure in paint manufacture (Group 3)

For definition of Groups, see [Preamble Evaluation](#).

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5. Summary of Data Reported and Evaluation

5.1 Exposures

Approximately 200 000 workers worldwide are employed in paint manufacture. The total number of painters is probably several millions, a major group being construction painters. Other industries in which large numbers of painters are employed include manufacture of transportation equipment and metal products, automotive and other refinishing operations and furniture manufacture.

Thousands of chemical compounds are used in paint products as pigments, extenders, binders, solvents and additives. Painters are commonly exposed by inhalation to solvents and other volatile paint components; inhalation of less volatile and nonvolatile components is common during spray painting. Dermal contact is the other major source of exposure. Painters may be exposed to other chemical agents that they or their coworkers use.

Painters are commonly exposed to solvents, the main ones being petroleum solvents, toluene, xylene, ketones, alcohols, esters and glycol ethers. Chlorinated hydrocarbons are used in paint strippers and less frequently in paint formulations. Benzene was used as a paint solvent in the past but is currently found in only small amounts in some petroleum solvent-based paints. Titanium dioxide and chromium and iron compounds are used widely as paint pigments, while lead was used commonly in the past. Asbestos has been used as a paint filler and may occur in spackling and taping compounds; painters in the construction industry and shipyards may also be exposed to asbestos. Exposure to silica may occur during the preparation of surfaces in construction and metal painting.

Workers in paint manufacture are potentially exposed to the chemicals that are found in paint products, although the patterns and levels of exposure to individual agents may differ from those of painters. Construction painters may be exposed to dusts and pyrolysis products during the preparation of surfaces and to solvents in paints, although water-based paints have become widely used recently. In metal and automobile painting, metal-based antirust paints and solvent-based paints are often applied by spraying; in addition, newer resin systems, such as epoxy and polyurethane, are commonly used. In contrast to other painting trades, furniture finishing involves the use of more varnishes, which have evolved from cellulose-based to synthetic resin varnishes, including amino resins which may release formaldehyde.

5.2 Human carcinogenicity data

The reports most relevant for assessing the risk for cancer associated with occupational exposures in paint manufacture and painting are three large cohort studies of painters and collections of national statistics on cancer incidence and mortality in which data on cancer at many sites were presented for painters. These show a consistent excess of all cancers, at about 20% above the national average, and a consistent excess of lung cancers, at about 40% above the national average. The available evidence on the prevalence of smoking in painters, although limited, indicates that an excess risk for lung cancer of this magnitude cannot be explained by smoking alone. The risks for cancers of the oesophagus, stomach and bladder were raised in many of the studies, but the excesses were generally smaller and more variable than those for lung cancer. Some of the studies also reported excess risks for leukaemia and for cancers of the buccal cavity and larynx.

Several other small cohort and census-based studies in painters provided estimates of risk for cancer at one or several sites. The risk for lung cancer was reported to be raised in eight, that for stomach cancer in two, that for bladder cancer in two, that for leukaemia in four, that for malignancies of the lymphatic system in three, that for buccal cancer in three, that for laryngeal cancer in one, that for skin cancer in one, and that for prostatic cancer in three. In many studies, risks for cancer were reported only for sites for which the result was statistically significant.

In the three cohort studies of workers involved in the manufacture of paint, two of which were small, there was little to suggest an excess risk of lung cancer or of cancer at any other anatomical site.

Eleven case-control and related studies of lung cancer could be evaluated. All of the studies showed an increased risk for lung cancer among painters. The five studies in which smoking was taken into account showed an increase of 30% or more in risk for lung cancer. Two studies suggested increased risks among painters for laryngeal cancer, and one indicated an increased risk for mesothelioma.

Cancer of the urinary tract has been examined in relation to exposure to paint in 15 case-control and related studies. Eight showed an excess risk for bladder cancer in all painters. In certain studies, specific aspects of exposure to paint were examined: car painters were addressed in two studies, one indicating an excess risk; spray painters were evaluated in three studies, two of which showed an excess risk; and exposure to lacquer and chromium was associated with a risk in one study.

In a study of occupational histories of patients with oesophageal and stomach cancers, high risks were seen for painters. A further study also identified a risk for stomach cancer and another a risk for oesophageal cancer. One study of cancer of the gall-bladder and of the biliary tract showed associations with the occupation of painting. A study of pancreatic cancer reported a high risk for exposure to paint thinners.

Five studies of leukaemia mentioned painters. Two studies showed excess risks. Two small studies of Hodgkin's disease and three studies of multiple myeloma showed increased risks in association with the occupation of painter or with any exposure to paints, paint-related products or organic solvents.

A single study of prostatic cancer showed a significant excess risk for manufacturers of paints and varnishes, and one study reported a high risk for testicular cancer among spray painters.

Twelve studies of childhood cancer mentioned paternal exposure to paint and related substances; four of these also presented data on maternal exposure. Three studies showed an excess of childhood leukaemia in association with paternal exposure and one in association with maternal exposure. Two studies showed an excess risk for brain tumours in the children of male painters. One small study of children with Wilms' tumour showed an excess in those whose fathers were painters. All of these excesses are based on small numbers of children whose parents had been exposed, even in the larger studies. In the other studies, no association was seen between parental exposure to paint and childhood cancers. The type and timing of exposure varied among these studies.

5.3 Other relevant data

Painters may suffer from allergic and nonallergic contact dermatitis, chronic bronchitis and asthma, and adverse effects on the nervous system. There is also some indication of adverse effects in the liver, kidney, blood and blood-forming organs. Many of these effects are also seen in paint production workers.

Of three studies on the fertility of painters, two showed no adverse effect and the third a possible excess frequency of infertility in men. One study reported an excess frequency of spontaneous abortion in female painters, based on self-reported data. Studies of birth weight, perinatal mortality rates and congenital malformations in the offspring of male painters generally showed no adverse effects; few data on female painters were available.

No increase in the frequency of sister chromatid exchange in peripheral lymphocytes was found in one study of painters or in one study of paint manufacturing workers.

5.4 Evaluation

There is *sufficient evidence* for the carcinogenicity of occupational exposure as a painter.

There is *inadequate evidence* for the carcinogenicity of occupational exposure in paint manufacture.

Overall evaluation

Occupational exposure as a painter *is carcinogenic (Group 1)*.

Occupational exposure in paint manufacture *is not classifiable as to its carcinogenicity (Group 3)*.

For definition of the italicized terms, see [Preamble Evaluation](#).

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