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Beryllium

DESIGNATIONS

CAS No.: 7440-41-7
Registry name: Beryllium
Chemical name: Beryllium
Synonyms, Trade names: Glycinium
Chemical name (German): Beryllium
Chemical name (French): Bryllium
Appearance: hard, shiny, silvery white metal

BASIC CHEMICAL AND PHYSICAL DATA

Chemical symbol:	Be
Rel. atomic mass:	9.01 g
Density:	1.848 g/cm^3 at 20C
Boiling point:	2,970C

Melting point:	1,287C
Vapour pressure:	4.84 Pa at 1287C
Explosion limits:	beryllium dusts may form explosive mixtures with air
Odour threshold:	none
Solvolysis/solubility:	beryllium itself is virtually insoluble in water but soluble in diluted mineral acids; beryllium hydroxide, beryllium oxide: insoluble in water, beryllium nitrate: 1,033 g/l (at 20C), beryllium sulphate 424 g/l (at 25C)

ORIGIN AND USE

Usage:
Beryllium is used in nuclear engineering, the construction of aircrafts and rockets, radiology and metallurgy.

Origin/derivation:
Beryllium is found in nature in various mineral compounds. It makes up approx. 0.006 % of the Earth's crust. It can be obtained as a pure metal by the sulphate, fluoride or chloride process from beryl ($3BeO.Al_2O_3.6SiO_2$)

Production figures:
The annual worldwide production of beryllium and its compounds is 3,000 - 4,000 t (acc. KOCH, 1989).

Emissions:
Approx. 8,000 t per annum (acc. KOCH, 1989)

Toxicity

Humans:	TCLo 300 mg/m^3, inhalation	acc. UBA, 1986
	LDLo 0.1 mg/m^3, inhalation	acc. KOCH, 1989
	Absorption of 0.025 mg/m^3 no toxic effect	acc. KOCH, 1989
Mammals:		
Rat	LD_{50} 9.7 mg/kg, oral	acc. KOCH, 1989
	LD_{50} 0.44 mg/kg, intravenous	acc. KOCH, 1989
	LD_{50} 0.50 mg/kg, intravenous	acc. UBA, 1986
	LD_{50} 0.19 mg/m^3, inhalation	acc. KOCH, 1989
Aquatic organisms:		
Small crustaceans	LC_{10} 10 mg/l	acc. UBA, 1986
	LC_{50} 18 mg/l	acc. UBA, 1986

	LC_{100} 50 mg/l	acc. UBA, 1986

Characteristic effects:

Humans/mammals: **Beryllium and its compounds are extremely poisonous. Poisoning is caused mainly by the inhalation of dust or by skin contact and causes irritation of and damage to the respiratory organs (bronchitis, pneumonia, dermatitis, the so called "beryllium disease"). Metal splinters or dusts which get into the skin cause beryllium ulcers and result in the most serious of known skin diseases. Oral application seldom causes poisoning since there is only slight resorption of beryllium. Chronic poisoning can be fatal. Pulmonary carcinomas induced in animal experiments. In the Federal Republic of Germany occupational illness/disease due to beryllium or its compounds must be reported to the appropriate authorities.**

Long-term exposure causes beryllium accumulation in the bones and liver. With chronic intake, the latent period may be more than 5 years.

ENVIRONMENTAL BEHAVIOUR

Water:
In water, the substance is found almost exclusively as mineral grains. A solution may be formed under acid conditions which inhibits the self-purification capability of surface water and groundwater from a concentration of 0.01 mg/l. Thus, beryllium poses a great hazard to fish and micro-organisms.

Air:
Beryllium is released into the atmosphere by emissions from companies processing beryllium. It is also liberated when coal is burned (on average 0.1-7 mg/kg; acc. DVGW, 1985).

Soil:
Soils are accumulation sinks.

Food chain:

Beryllium is accumulated in aquatic organisms (bioaccumulation factor: 1,000). In addition to foodstuffs, beryllium may also be absorbed in considerable quantities from drinking water.

ENVIRONMENTAL STANDARDS

Medium/acceptor	Sector	Country/organ.	Status	Value	Cat.	Remarks	Source
Water:	Surface	D	(G)	0.0001 mg/l		For natural treatm.	acc. DVGW, 1985
	Surface	D	(G)	0.0002 mg/l		For phys.-chem. treatm.	acc. DVGW, 1985
	Drinkw	SU		0.0002 mg/l		1970	acc. DVGW, 1985
	Irrigation	D	(G)	0.1 mg/l		For field culture	acc. DVGW, 1985
	Irrigation	D	(G)	0.05 mg/l		For cultivation under glass	acc. DVGW, 1985
	Irrigation	USA	(G)	0.5 mg/l		1968	acc. DVGW, 1985
	Irrigation	USA	(G)	1 mg/l		1968, short-time value	acc. DVGW, 1985
Soil:		D	G	10 mg/kg		in cultivated	acc. KLOKE,

						soils	1988
Air:	Emiss.	D	L	0.1 mg/m^3		mass flow > 0.5 g/h1)	acc. TA Luft, 1986
		IL		0.00001 mg/m^3		24 h	acc. STERN, 1986
		USA		0.00001 mg/m^3		24 h	acc. MERIAN, 1984
		YU		0.00001 mg/m^3		24 h	acc. MERIAN, 1984
	Workp	AUS	(L)	0.002 mg/m^3			acc. MERIAN, 1984
	Workp	B	(L)	0.002 mg/m^3			acc. MERIAN, 1984
	Workp	BG	(L)	0.001 mg/m^3			acc. MERIAN, 1984
	Workp	CH	(L)	0.002 mg/m^3			acc. MERIAN, 1984
	Workp	CS	(L)	0.001 mg/m^3		Long-time value	acc. MERIAN, 1984
			(L)	0.002			

Workp	CS	(L)	mg/m^3		Short-time value	acc. MERIAN, 1984
Workp	D	L	0.005 mg/m^3	TRK	Grinding of metal	DFG, 1989
Workp	D	L	0.002 mg/m^3	TRK	Other work	DFG, 1989
Workp	DDR	(L)	0.002 mg/m^3	MAK	Short & long-time value	acc. MERIAN, 1984
Workp	H	(L)	0.001 mg/m^3			acc. MERIAN, 1984
Workp	I	(L)	0.002 mg/m^3			acc. MERIAN, 1984
Workp	J	(L)	0.002 mg/m^3			acc. MERIAN, 1984
Workp	NL	(L)	0.002 mg/m^3			acc. MERIAN, 1984
Workp	PL	(L)	0.001 mg/m^3			acc. MERIAN, 1984
Workp	RO	(L)	0.001		Short-time	acc. MERIAN,

				mg/m^3		value	1984
	Workp	S	(L)	0.002 mg/m^3			acc. MERIAN, 1984
	Workp	SF	(L)	0.002 mg/m^3			acc. MERIAN, 1984
	Workp	SU	(L)	0.001 mg/m^3			acc. SORBE, 1989
	Workp	USA	(L)	0.002 mg/m^3	TWA		ACGIH, 1986
	Workp	YU	(L)	0.002 mg/m^3			acc. MERIAN, 1984

Note:

The use of beryllium compounds in cosmetics is prohibited in the Federal Republic of Germany.

1) Be and its compounds in respirable form, stated as Be

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Surface/groundwater:			
Drinking water (1961-1966)	USA	0.01-0.7 g/l	acc. DVGW, 1985

Upper Palatinate Forest	D	1-12 g/l	acc. DVGW, 1985
Lake Constance (1971-1973)	D	< 0.2 g/l	acc. DVGW, 1985
Rhine (Lobith, 1983)	D	0.01-0.09 g/l	acc. DVGW, 1985
Sediments:			
Lake Baldeney, Ruhr (1975)	D	1.4-1.7 mg/kg	acc. DVGW, 1985
Air:			
Atmosphere		0.5-0.8 ng/m^3	acc. KOCH, 1989
Cigarette smoke		0.47-0.74 g/cigarette	acc. KOCH, 1989

Assessment/comments

Due to the toxicity and the carcinogenic potential of beryllium, it is to be ensured that there is no long-term contamination of drinking water in particular. Industrial waste water should be filtered and the beryllium recycled. Direct skin contact is to be avoided when handling beryllium and its compounds.

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Bromine

DESIGNATIONS

***CAS No.:* 7726-95-6**
***Registry name:* Bromine**
***Chemical name :* Bromine**
***Synonyms, Trade names:* Bromine**
***Chemical name (German):* Brom**
***Chemical name (French):* Brome**
***Appearance:* dark, reddish-brown, fuming, highly volatile liquid or vapour (below -7.2C metal-like crystals, colourless at -252C)**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	Br_2
Rel. molecular mass:	159.80 g
Density:	3.12 g/cm^3
Relative gas density:	5.5
Boiling point:	58.78C
Melting point:	-7.2C

Vapour pressure:	220 hPa at 20C; 340 hPa at 30C; 740 hPa at 50 C
Odour threshold:	1 ppm in air
Solvolysis/solubility:	in water 42 g/l at 0C
	35.5 g/l at 20C
	soluble in benzene, gasoline, chloroform, ethanol, ether and hydrogen sulphide
Conversion factors:	1 mg/m^3 = 0.150 ml/m^3
	1 ml/m^3 = 6.658 mg/m^3
Note:	Apart from mercury, bromine is the only element which is liquid at room temperature. Bromine is highly reactive: it reacts explosively with several metals, corrodes many organic substances and reacts as an oxidising agent.

ORIGIN AND USE

Usage:

Most bromine (about 30 %) is used to produce 1,2-dibromoethane which is added to fuels containing tetraethyl lead to stop lead deposits collecting on the valves of internal-combustion engines. Further uses are the production of oil well packs and completion fluids as calcium bromide (22 %) and in flame retardants (16 %). It is used as starting material for the organic synthesis of pesticides, dyes, medicines, photographic chemicals and contrast media.

Origin/derivation:

Bromine is mainly obtained from waste lyes in the potassium industry; seawater is a further source. Waste lyes, brine and mother liquids etc. with a bromine content of > 1 g/l are processed by means of hot debromination, with cold debromination being used for seawater. Subsequent distillation and drying remove chlorine, residual water and impurities from the raw bromine segregated from the aqueous phase (99% yield).

Production figures:

Worldwide

1982 = 381,000 t (ULLMANN, 1985)
1983 = 364,200 t
1984 = 373,000 t

Toxicity

Humans:	LDLo 14 mg/kg, oral	acc. UBA, 1986
	LCLo 1000 ppm, res. air (inhal.) = lethal	acc. UBA, 1986
Mammals:		
Mouse	LC_{50} 750 ppm (9 min), inhalation	acc. UBA, 1986
Cat	LCLo 140 ppm (7 h), inhalation	acc. UBA, 1986
Rabbit	LCLo 180 ppm (6.5 h), inhalation	acc. UBA, 1986
Aquatic organisms:		

Goldfish	20 mg/l = lethal	acc. UBA, 1986
Small crustaceans	10 mg/l = lethal	acc. UBA, 1986

Characteristic effects:

Humans/mammals: The liquid produces severe, poorly healing irritation and burning of the eyes, the respiratory organs, the skin and the gastro-intestinal tract. Deep, painful necroses form on the skin and the mucous membranes. High concentrations cause oedemas of the glottis, larynx and lungs as well as inflammation of the lungs. Poison class 2 (ROTH, 1989). Bromine vapours are even more hazardous as they produce bronchial spasms and pneumonia.

Plants: Bromine is a trace element. Bromides do not damage plants, but are not necessarily required.

ENVIRONMENTAL BEHAVIOUR

Water:
Bromine is denser than water, but dissolves slightly in water and colours it brown. This is followed by the generation of bromine vapours. There is a strong hazard to all types of water, in particular to drinking water, service water and waste water on account of the high toxicity linked to oxidising and corrosive properties. Bromides are found as accompanying ions in potassium and sodium deposits. The bromide content rises with increasing salinity. In coastal areas, higher bromide concentrations in the groundwater can be attributed to the infiltration of seawater. Water hazard class 2 (ROTH, 1989). Bromine prevents the formation of algae in water.

Air:
The liquid readily evaporates and forms caustic vapours. The vapours are denser than air and creep along the ground.

ENVIRONMENTAL STANDARDS

Medium/acceptor	Sector	Country/organ.	Status	Value	Cat.	Remarks	Source
Soil:		NL	G	20 mg/kg AD			acc. KLOKE, 1988
		NL	G	50 mg/kg AD		Investigation	acc. KLOKE, 1988
		NL	G	300 mg/kg AD		Rehabilitation	acc. KLOKE, 1988
Air:	Workp	D	L	0.1 ml/m^3	MAK		DFG, 1989
	Workp	USA	(L)	0.7 mg/m^3	TWA		acc. ACGIH, 1986
	Workp	USA	(L)	2 mg/m^3	STEL		acc. UBA, 1986
	Workp	SU	(L)	0.075 ml/m^3			acc. UBA, 1986
	Workp	SU	(L)	1 mg/m^3		Skin resorption	acc. KETTNER, 1979
	Emiss.	**D**	**L**	**5 mg/m^3**		**mass flow > 50 g/h**	**acc. TA Luft, 1986**

Comparison/reference values

Medium/origin	Country	Value	Source
Seawater		0.065 kg/m^3	acc. ULLMANN, 1978
Fly ash (coal)	USA	0.3-21 mg/kg	acc. HOCK, 1988
Plants		15 mg/kg	acc. HOCK, 1988

Assessment/comments

The biological action of bromine and its compounds is quite similar to that of chlorine and its compounds. However, most bromine compounds are more toxic than the corresponding chlorine compounds while chlorine itself is more hazardous than bromine because of its reactivity.

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Cadmium

DESIGNATIONS

CAS No.: **7440-43-9**
Registry name: **Cadmium**
Chemical name: **Cadmium**
Synonyms, Trade names: **Cadmium**
Chemical name (German): **Cadmium**
Chemical name (French): **Cadmium**
Appearance: **soft heavy metal with silvery white sheen**

BASIC CHEMICAL AND PHYSICAL DATA

Chemical symbol: **Cd**
Rel. atomic mass: **112.41 g**
Density: **8.642 g/cm^3 at 20C**
Boiling point: **767C**
Melting point: **320.9C**
Vapour pressure: **0.013 Pa at 180C**
Solvolysis/solubility: **Cd readily dissolves in nitric acid, but only slowly in hydrochloric and sulphuric acid**

Basic data of selected compounds

CAS No:	1306-19-0	10108-64-2
Chemical name:	Cadmium oxide	Cadmium chloride
Synonyms, Trade names:	Cadmium orange, Cadmium yellow, Aurora yellow	Cadmium dichloride, Caddy
Chemical name (German):	Cadmiumoxid	Cadmiumchlorid

Chemical name (French):	Oxyde de cadmium	Chlorure de cadmium
Appearance:	yellow to brown solid	colourless, crystalline solid
Empirical formula:	CdO	$CdCl_2$
Rel. molecular mass:	128.41 g	183.32 g
Density:	6.95 g/cm^3	4.05 g/cm^3
Boiling point:		967C
Melting point:	sublimation above 700C	568C
Solvolysis/solubility:	in water: very slightly (0.005 wt%) readily soluble in diluted acids	in water: readily soluble (1,400 g/l)

ORIGIN AND USE

Usage:
Metallic cadmium is used to coat iron in order to improve corrosion resistance and in alloys in the automobile industry. Its compounds are used as pigments (primarily as cadmium sulphide) and as plastic stabilisers in PVC, in batteries and in fungicides; also applied for Ni-Cd batteries as well as moderators and control elements in nuclear reactors.

Origin/derivation:
Cadmium minerals are rare; frequently found in isomorphic form in nearly all zinc ores (95 % of cadmium production). Cadmium is separated from Zn by distillation or precipitation from sulphate solution using zinc dust.

Production figures:

1980 = 18 x 10^3 t (worldwide); MERIAN, 1984

Toxicity

Mammals:		
Rat	LDLo 15 mg/kg, ims	acc. KOCH, 1989
Rat	TDLo 70 mg/kg, ims	acc. KOCH, 1989
Rat	LD_{50} 88 mg/kg , (cadmium chloride)	acc. DVGW, 1988
Rat	LD_{50} 72 mg/kg , (cadmium oxide)	acc. DVGW, 1988
Rabbit	LD_{50} 70-150 mg/kg , (cadmium chloride)	acc. DVGW, 1988
Guinea pig	LD 150 mg/kg , cadmium fluoride	acc. DVGW, 1988
Aquatic organisms:		
Daphnia	0.1 mg/l (harmful)	acc. DVGW, 1988
Stickleback	0.3 mg/l (harmful)	acc. DVGW, 1988
Minnow	LD 5600 mg/l (1 h)	acc. DVGW, 1988

Goldfish	LD 0.017 mg/l (9-18 h)	acc. DVGW, 1988
Fish (without spec. differentiation)	LC_{50} minimal 23 ppm (over 264 h)	acc. KOCH, 1989
	LC_{50} average 140 ppm (over 24 h)	acc. KOCH, 1989

Characteristic effects:

Humans/mammals: **Apart from the gastro-intestinal tract and the lungs, the organ most susceptible to chronic cadmium exposure is the kidney. Cadmium is a cumulative poison; the ready solvolyse of the element in weak acids is an important precondition for its absorption in the organism: 5 % of cadmium is resorbed via the gastro-intestinal tract and accumulated in liver and kidneys. In Asia, "Itai-Itai" disease is caused by high cadmium concentrations in rice. Damage is caused by erythrocyte destruction, proteinuria, rhinitis, emphysema and chronic bronchitis. Cadmium and its compounds are carcinogenic. A typical symptom of chronic poisoning is the excretion of -microglobulin in the urine due to the impairment of the kidney function. Bone deformation may also result.**

Plants: **Cadmium reduces the photosynthesis and transpiration rate whilst increasing the respiratory rate. Small cadmium concentrations in the soil are sufficient to produce marked damage such as shortening of the shoot axis and intensive yellowing of older leaves. Absorption not only takes place via the roots but also through shoots and leaves. In addition to yield shortfalls the main hazard results from the contamination of (accumulation in) crop plants since this is how cadmium as a cumulative poison enters the food chain.**

ENVIRONMENTAL BEHAVIOUR

Water:
2/3 - 3/4 of the cadmium in surface water and groundwater is adsorbed on suspended matter. It can be remobilised from sediment by complexing agents. Fish toxicity is dependent amongst other things on the calcium

content of the water. Generally speaking, a high calcium content in water reduces the toxic effect of cadmium on fish.
The biological self-purification of surface water and groundwater is impaired from 0.1 mg/l (DVGW, 1988).

Air:
Cadmium is stable in air since it forms an oxide layer.

Soil:
The cadmium retention capacity is a function of the number of exchangeable alkalis. There is hardly any elution because of the adsorption on organic soil components. The accumulation horizon for cadmium is in the rhizosphere (roots). The minimum availability is at pH 6.5. The cadmium absorption by plants increases with a decreasing pH of the soil.

Degradation, decomposition products, half-life:
Cadmium accumulates in the organism. 50 % of the accumulated amount is found in the liver and the kidneys. Cadmium is primarily excreted in urine (2 μ g/d on average (0.2 - 3.1 μ g/l)). The biological half-life of cadmium in the human body is between 15 and 25 years (measured in the kidneys; GROKLAUS, 1989).

Food chain:
Humans consume roughly one third of the cadmium to which they are exposed in animal foodstuffs and two thirds in vegetable foodstuffs. Smoking significantly increases the cadmium impact.

Note: **[1] The biological half-life is the time taken for a specified element concentration in the body to be reduced to half its original value by way of degradation and excretion processes.**

ENVIRONMENTAL STANDARDS

Medium/acceptor	Sector	Country/organ.	Status	Value	Cat.	Remarks	Source

Water:	Drinkw	AUS	(L)	10 g/l			acc. MERIAN, 1984
	Drinkw	CH	(L)	5 g/l			acc. MERIAN, 1984
	Drinkw	D	L	5 g/l		Amendment, 1985	acc. DVGW, 1988
	Drinkw	EC	G	5 g/l			acc. LAU-BW, 1989
	Drinkw	SU	(L)	10 g/l			acc. MERIAN, 1984
	Drinkw	USA	(L)	0.01 mg/l			acc. DVGW, 1988
	Drinkw	WHO	G	5 g/l			acc. MERIAN, 1984
	Surface	D	L	50 g/m^3		Investigation	acc. LAU-BW, 1989
	Surface	D	L	100 g/m^3		Rehabilitation	acc. LAU-BW, 1989
	Surface	D	G	0.005 mg/l		1) A	acc. DVGW, 1988
			G				acc. DVGW,

	Surface	D	G	0.01 mg/l		2) B	1988
	Groundw	D(HH)	G	2.5 g/l		Investigation	acc. LAU-BW, 1989
	Groundw	D(HH)	G	10 g/l		Rehabilitation	acc. LAU-BW, 1989
	Groundw	NL	G	0.4 g/l		Reference	acc. TERRA TECH 6/94
	Groundw	NL	L	6 g/l		Intervention	acc. TERRA TECH 6/94
	Waste water	CH	(L)	0.10 g/m^3		Direct/indirect introduc.	acc. LAU-BW, 1989
	Waste water	CH	G	0.005 g/m^3		Quality aim	acc. LAU-BW, 1989
	Waste water	D(BW)	G	1 g/m^3			acc. LAU-BW, 1989
	Trough water	D	L	6 g/l			acc. DVGW, 1988
Soil:		CH	G	0.8 mg/kg AD			acc. LAU-BW, 1989
		CH	G	0.03 mg/kg		Sol. content	acc. LAU-BW, 1989

	D(HH)	G	8 mg/kg DS		Investigation	acc. LAU-BW, 1989
	NL	G	0.8 mg/kg AD		Reference	acc. TERRA TECH 6/94
	NL	L	12 mg/kg AD		Intervention	acc. TERRA TECH 6/94
Sewage sludge	CH	L	30 mg/kg DS		Sludge	acc. LAU-BW, 1989
Sewage sludge	D	L	3 mg/kg AD		Soil	acc. LAU-BW, 1989
Sewage sludge	D	L	20 mg/kg DS		Sludge DS	acc. LAU-BW, 1989
Sewage sludge	EC	L	1-3 mg/kg DS		Soil DS	acc. LAU-BW, 1989
Sewage sludge	EC	L	20 -.40 mg/kg DS		Sludge DS	acc. LAU-BW, 1989
Fertiliser	D	L	4 mg/kg		3)	acc. LAU-BW, 1989
Fertiliser	D	L	3 mg/kg		Soil AD	acc. LAU-BW, 1989
						acc. LAU-BW,

	Fertiliser	D		20 mg/kg		Sludge	1989
	Compost	A	G	1-6 ppm			acc. LAU-BW, 1989
	Compost	CH	L	3 mg/kg			acc. LAU-BW, 1989
	Compost	D	G	3 mg/kg		Soil AD	acc. LAU-BW, 1989
	Compost	D	G	33 g/ha and a			acc. LAU-BW, 1989
Air:		CH	L	10 ng/m^3		7)	acc. LAU-BW, 1989
		CH	L	2 g/m3 and d		In dust deposition	acc. LAU-BW, 1989
		D	L	0.04 g/m^3	IW1	5) 6)	acc. TA Luft, 1986
		D	L	5 g/m3 and d	IW1	5) 7)	acc. TA Luft, 1986
		WHO	G	1-5 ng/m^3		8)	acc. LAU-BW, 1989
		WHO	G	10-20 ng/m^3		9)	acc. LAU-BW, 1989

Emiss.	D	L	0.2 mg/m^3		mass flow > 50 g/h10)	acc. TA Luft, 1986
Workp	AUS	L	0.05 mg/m^3		Dust and sol. salts	acc. MERIAN, 1984
Workp	AUS	L	0.05 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	B	L	0.05 mg/m^3		Dust and sol. salts	acc. MERIAN, 1984
Workp	B	L	0.05 mg/m^3		Manufacture, Cd oxide	acc. MERIAN, 1984
Workp	B	L	0.05 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	BG	L	0.1 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	CS	L	0.1 mg/m^3		Cd oxide, smoke, Cd 11)	acc. MERIAN, 1984
Workp	CS	L	0.5 mg/m^3		Cd oxide, smoke, Cd 12)	acc. MERIAN, 1984
					Cd oxide, smoke,	acc. MERIAN,

Workp	CH	L	0.1 mg/m^3		as Cd	1984
Workp	CH	L	0.2 mg/m^3		Dust and sol. salts	acc. MERIAN, 1984
Workp	D	L	1.5 g/dl	BAT	Whole blood	acc. DVGW, 1988
Workp	D	L	15 g/l	BAT	Urine	acc. DVGW, 1988
Workp	DDR	L	0.1 mg/m^3		Dust and sol. salts 11)	acc. MERIAN, 1984
Workp	DDR	L	0.2 mg/m^3		Dust and sol. salts 12)	acc. MERIAN, 1984
Workp	NL	L	0.05 mg/m^3		Manufacture, Cd oxide	acc. MERIAN, 1984
Workp	NL	L	0.05 mg/m^3		Dust and sol. salts	acc. MERIAN, 1984
Workp	NL	L	0.05 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	H	L	0.1 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
		L	0.05		Dust and sol.	acc. MERIAN,

Workp	I		mg/m^3		salts	1984
Workp	I	L	0.01 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	J	L	0.1 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	PL	L	0.1 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	RO	L	0.2 mg/m^3		Cd oxide, smoke, Cd 12)	acc. MERIAN, 1984
Workp	S	L	0.05 mg/m^3		Dust and sol. salts	acc. MERIAN, 1984
Workp	S	L	0.02 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	SF	L	0.02 mg/m^3		Dust and sol. salts	acc. MERIAN, 1984
Workp	SF	L	0.01 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	SU	L	0.1 mg/m^3		Cd oxide	acc. KETTNER, 1979
		L				acc. KETTNER,

Workp	SU	L	0.3 mg/m^3		Cd hydroxide	1979
Workp	SU	L	0.1 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Workp	USA	L	0.1 mg/m^3		Cd oxide, smoke, Cd 11)	acc. MERIAN, 1984
Workp	USA	L	0.3 mg/m^3		Cd oxide, smoke, Cd 12)	acc. MERIAN, 1984
Workp	USA	(L)	0.05 mg/m^3	TWA		acc. DVGW, 1988
Workp	USA	L	0.2 mg/m^3		Dust and sol. salts 11)	acc. MERIAN, 1984
Workp	USA	L	0.6 mg/m^3		Dust and sol. salts 12)	acc. MERIAN, 1984
Workp	USA	(L)	0.05 mg/m^3	TWA	Smoke, Cd oxide	acc. ACGIH, 1986
Workp	USA	(L)	0.05 mg/m^3	TWA	Manufacture, Cd oxide	acc. ACGIH, 1986
Workp	WHO	L	0.01 mg/m^3		Dust and sol. salts 13)	acc. MERIAN, 1984

	Workp	WHO	L	0.25 mg/m^3		Dust and sol. salts 12)	acc. MERIAN, 1984
	Workp	YU	L	0.1 mg/m^3		Cd oxide, smoke, as Cd	acc. MERIAN, 1984
Foodstuffs:		WHO	G	0.07 mg/d			acc. RMPP, 1979
		D	L	0.005 mg/l		Mineral water	acc. DVGW, 1988
		D	G	0.0025 mg/kg		Milk	acc. GROKLAUS, 1989
		D	G	0.05 mg/kg		Cheese	acc. GROKLAUS, 1989
		D	G	0.1 mg/kg		Beef	acc. GROKLAUS, 1989
		D	G	0.1 mg/kg		Sausage meat	acc. GROKLAUS, 1989

Notes:

1) For drinking water treatment in each case: A = signifies pollution limits up to which water can be used as drinking water

2) For drinking water treatment in each case: B = signifies pollution limits up to which drinking water can be produced with the assistance of currently known and proven chemophysical methods

3) In organ.-mineral, mixed fertiliser

4) Annual arithmetic mean, in suspended dust

5) Annual arithmetic mean on the basis of daily mean values, protection of humans

6) Cd and its anorganic compounds as suspended dust, stated as Cd

7) Cd and its anorganic compounds within dust sediments, stated as Cd

8) Averaging period: 1 year (rural areas); protection of humans

9) Averaging period: 1 year (urban areas); protection of humans

10) dustlike Cd ist and anorganic compounds, stated as Cd

11) Mean value

12) Short-time value

13) Long-time value

14) Tolerable weekly absorption

Further regulations in: Order Governing Cosmetics, 1985: Ban on usage; Law Governing Paints, 1987. Usage prohibited in the manufacture of foodstuffs, semi-luxury goods and consumer goods; the utilisation of Cd compounds as pesticides is prohibited in Germany.

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Surface water			
Rhine (Cologne):	D	0.03 - 0.2 g/l	acc. DVGW, 1988
Rhine (Duisburg):	D	0.05 - 0.5 g/l	acc. DVGW, 1988
Ruhr (Witten):	D	0.2 - 2.1 g/l	acc. DVGW, 1988
Ruhr (Duisburg):	D	0.4 - 0.6 g/l	acc. DVGW, 1988
Fertiliser (in 100 kg P_2O_5):	USA	1.2 - 2.4 g Cd/ha	acc. BREMER UMWELT INST., 1985
	Morocco	3.5 - 7 g Cd/ha	acc. BREMER UMWELT INST., 1985
	SU	3.6 - 7.2 g Cd/ha	acc. BREMER UMWELT INST., 1985
	Senegal	11.4 - 22.8 g Cd/ha	acc. BREMER UMWELT INST., 1985
Fly ash (coal)	USA	0.1-3.9 mg/kg	acc. HOCK, 1988
Plants		0.05-0.2 mg/kg	acc. HOCK, 1988

Assessment/comments

As a trace element, cadmium is subject to constant circulation in biological and non-biological environmental structures. The impact on the environment which cadmium causes by natural means (approx. 40 t/a worldwide) is

slight compared to the estimated anthropologically-induced emissions. Cadmium is regarded as one of the most poisonous metals. There have been documented cases of acute and chronic poisoning due to workplace exposure. Cadmium is one of the trace elements for which there is proof of poisoning of certain groups of people as a consequence of chronic, environment-related exposure.

The proven accumulation of the element in sediments, the associated possibility of remobilisation and the tendency towards bioaccumulation are the major environmental hazards. If possible, waste products containing cadmium should be reprocessed.

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Carbaryl

DESIGNATIONS

CAS No.: 63-25-2
Registry name: Carbaryl
Chemical name: 1-Naphthyl-methylcarbamate
Synonyms, Trade names: 1-Naphthalenyl-methylcarbamate, Sevin, Atoxan, Caprolin, Compound 7744, Gamonil, Panam, Sevidol and many others
Chemical name (German): Carbaryl, 1-Naphthyl-N-methylcarbamat
Chemical name (French): Carbaryl, 1-naphthyl mthylcarbamate

Appearance: **colourless, crystalline solid (pure substance)**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$C_{12}H_{11}NO_2$
Rel. molecular mass:	201.23 g
Density:	1.232 g/cm^3
Boiling point:	not distillable
Melting point:	142C
Vapour pressure:	<0.7 Pa at 25C
Solvolysis/solubility:	in water: < 1g/l in acetone: 200-300 g/l; in cyclohexanone: 200-250 g/l; in ether: 200 g/l; in isopropanol: 100 g/l; in xylene: 100 g/l

ORIGIN AND USE

Usage:

Carbaryl is a contact insecticide with a long lasting effect. It is used as a spraying powder (50% + 12.5% Tetradifon; 37.5 % + 11.2 % Dioxathion) in winegrowing e.g. against mites and in combination with lindane against potato-bugs.

Origin/derivation:

There are no natural sources. Carbaryl is obtained from 1-naphthol, either by reaction with methylisocyanate or by treatment with phosgene and subsequent reaction of the resulting chloroformate with methylamine.

Production figures:

World production of carbaryl:

25,000 t	(1971)	(ULLMANN, 1989)
1,000-1,500 t	(1989)	(KOCH, 1989)

Toxicity

Mammals:		
Rat	LD_{50} 400-850 mg/kg, oral	acc. PERKOW, 1994
	LD_{50} >4000 mg/kg, dermal	acc. PERKOW, 1994
	NOEC 2,000mg/kg feed, (2a)	acc. PERKOW, 1994
Rabbit	LD_{50} 710 mg/kg, oral	acc. PERKOW, 1994
	LD_{50} >2,000 mg/kg, dermal	acc. PERKOW, 1994
Guinea pig	LD_{50} 280 mg/kg, oral	acc. KOCH, 1989

Aquatic organisms:		
Fish (various)	LC$_{50}$ 1.75-4.25 mg/l (24 h)	acc. PERKOW, 1994
	LC$_{50}$ 0.1-13 mg/l (24 h)	acc. KOCH, 1989
	LC$_{50}$ 2-20 mg/l (24 h)	acc. VERSCHUEREN, 1983
Goldfish	LC$_{50}$ 28 mg/l (24 h)	acc. PERKOW, 1994
	LC$_{50}$ 13.2 mg/l (96 h)	acc. VERSCHUEREN, 1983
Insects:		
Bee	LD$_{50}$ 1.3 g/bee, contact	acc. PERKOW, 1994
	LD$_{50}$ 0.14 g/bee, oral	acc. PERKOW, 1994

Characteristic effects:

***Humans/mammals:* Carbaryl belongs to the group of the carbamates which are quickly degraded and not accumulated in humans. Poisoning may be due to oral intake or resorption via the skin. The physiological effects result from the (reversible) inhibition of the enzyme acetylcholinesterase causing convulsions and other neuromuscular manifestations. These effects take place very quickly but last only for a short time. Carbaryl is readily hydrolysed forming 1-naphthol which is excreted.**

ENVIRONMENTAL BEHAVIOUR

Soil:

Due to its solubility in water, carbaryl is expected to be very mobile in soil. Thus, there is hardly any accumulation in this medium. Generally, the persistence of carbamate insecticides in soil is considered to be low to moderate (one to four months) due to their mobility and biodegradability [ULLMANN, 1989].

Water:

In aquatic systems hydrolysis takes place at pH>7 forming 1-naphthol and methylamine. Although degradation is relatively fast the possibility of migration of the substance into the groundwater has to be taken into account [KOCH, 1989]. In Germany, the substance is classified in water hazard class 2.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/organ.	Status	Value	Cat.	Remarks	Source
Water:	Drinkw	D	L	0.1 g/l		single substance	acc. KOCH, 1989
	Drinkw	SU	L	100 g/l			acc. KOCH, 1989
	Groundw	NL	L	0.1 g/l		Intervention	acc. TERRA TECH, 6/94
Soil:		NL	L	5 mg/kg		Intervention	acc. TERRA TECH, 6/94
Air:	Workp	D	L	5 mg/m^3	MAK	Skin	acc. AUER TECHNIKUM,

							1988
	Workp	USA	(L)	5 mg/m^3	TWA		acc. AUER TECHNIKUM, 1988
	Workp	SU	(L)	1 mg/m^3			acc. AUER TECHNIKUM, 1988
	Emiss.	D	L	5 mg/m^3		mass flow > 25 g/h	acc. TA Luft, 1986
Foodstuffs:		WHO	G	0.01 mg/kg/d	ADI		acc. KOCH, 1989
Kiwi		D	L	10 mg/kg			acc. PERKOW, 1994
Apple, apricot, pear, peach, plum, grape, cabbage, salad		D	L	3 mg/kg			acc. PERKOW, 1994
Remaining fruits and vegetable, rice		D	L	1 mg/kg			acc. PERKOW, 1994
Remaining grain		D	L	0.5 mg/kg			acc. PERKOW, 1994
Remaining vegetable food		D	L	0.1 mg/kg			acc. PERKOW,

						1994
						acc. PERKOW, 1994

Note:
In Germany , the use of carbaryl is prohibited (1994)

Assessment/comments

Carbaryl features a relatively low toxicity for humans, no tendency towards bioaccumulation and a low persistence in soil due to its biodegradability. On the other hand, it has a toxic effect on bees and aquatic organisms even in small quantities. Thus, its use should be reduced as far as possible.

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Carbon monoxide

DESIGNATIONS

CAS No.: **630-08-0**
Registry name: **Carbon monoxide**
Chemical name: **Carbon monoxide**

Synonyms, Trade names: **Carbon oxide, carbon(II) oxide**
Chemical name (German): **Kohlenmonoxid, Kohlenoxid**
Chemical name (French): **Oxyde de carbone, monoxyde de carbone**
Appearance: **colourless, odourless gas**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	CO		
Rel. molecular mass:	28.01 g		
Density:	1.25 g/l at 0C		
Relative gas density:	0.97		
Boiling point:	-191.5C		
Melting point:	-199C		
Ignition temperature:	605C		
Explosion limit:	12.5 - 74 vol.%		
Max. explosion pressure:	7.3×10^5 Pa		
Odour threshold:	none		
Solvolysis/solubility:	in water:	33 ml/l	(at 0C)

		23 ml/l	(at 20C)
	soluble in ethyl acetate, chloroform, glacial acetic acid, ethyl acetate and other organic solvents		
Conversion factors:	1ppm = 1.164 mg/m^3		
	1 mg/m^3 = 0.859 ppm		

ORIGIN AND USE

Usage:
The most important use of CO in production involves its reaction with steam at elevated temperature to form syngas which is e.g. used to produce methanol. Carbon monoxide is also used in the reduction of oxides to form pure metals. It is, however, used on a very small scale.

Origin/derivation:
Carbon monoxide is an unwanted by-product of numerous thermal processes. It is produced in the course of all oxygen-undersaturated combustion processes involving carbon and its compounds. The natural sources of carbon monoxide predominate (90% of total emissions); the remaining 10% are made up of motor-vehicle emissions (55%), industry (11%) and other emitters (HORN, 1989).
Carbon monoxide is a component of "town gas".

Toxicity

Humans:	LCLo 4,000 ppm, inhalation (30 min)	acc. UBA, 1986

	TCLo 650 ppm, inhalation (45 min)	acc. UBA, 1986
Mammals:		
Rat:	LC_{50} 1,807 ppm, inhalation (4 h)	acc. UBA, 1986
Mouse:	LC_{50} 2,444 ppm, inhalation (4 h)	acc. UBA, 1986
Cat:	MLC 10,040 mg/m^3, inhalation (35 min)	acc. HORN, 1989
Guinea pig:	LC_{50} 2,811 mg/m^3, inhalation (4 h)	acc. HORN, 1989
Aquatic organisms:		
Fish:	LD > 1.2 mg/l	acc. UBA, 1986

Characteristic effects:

Humans/mammals: **The toxicity in humans and animals is caused by the extraordinary affinity of carbon monoxide for haemoglobin which is responsible for the oxygen transport (approx. 250 times greater affinity of carbon monoxide compared to oxygen [ULLMANN/RMPP]). Intake is exclusively by way of inhalation. Carbon monoxide cannot be perceived on the basis of odour, colour, taste, irritation of the mucous membranes or other effects. Thus, poisoning due to town gas or vehicle emissions may take place (frequently suicide results).**

Acute poisoning takes the form of headaches, retching, muscular weakness, loss of consciousness, shortness of breath and finally death, depending on the concentration and time of exposure.

Plants: Carbon monoxide does not poison plants since it is rapidly oxidised to form carbon dioxide which is used for photosynthesis.

ENVIRONMENTAL BEHAVIOUR

Water:

Carbon monoxide is only slightly soluble in water. Depressurizing the compressed gas quickly leads to the formation of explosive mixtures over the surface of the water. Carbon monoxide is listed under water hazard class 0 in the Federal Republic of Germany (no hazard to water). It has a toxic effect on fish.

Air:

Carbon monoxide is about as dense as air. It ingresses into the atmosphere by way of exhaust gases and is rapidly oxidised to form carbon dioxide. The substance is particularly hazardous because of ist widespread dispersion and the high toxicity level for humans and animals. Particular attention is therefore to be paid to the CO concentration in breathing air in smog areas.

Soil:

Oxygen-undersaturated soils have been found to have a higher concentration of carbon dioxide oxidised from carbon monoxide. CO accelerates the oxidation of NO to form NO_2. Roughly 80 t CO/km^2 are converted every year by soil bacteria.

Half-life:

The dwell time of CO in the atmosphere is between 1 and 2 months on average (HORN, 1989). The half-life of carbon monoxide bonded in blood is about 250 minutes (HORN, 1989).

Degradation, decomposition products:

Carbon monoxide rapidly oxidises to form carbon dioxide. Especially at higher temperatures, it reacts explosively with numerous substances (e.g. aluminium dust, potassium, nitrogen dioxide) with heat being produced (e.g.

bromine trifluoride, silver oxide). Plants metabolise CO to form CO_2 or methane.

Food chain:

There is no evidence of residues in foodstuffs or semi-luxury goods. Smokers inhale considerable quantities of carbon monoxide in cigarette smoke.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/ organ.	Status	Value	Cat.	Remarks	Source
Air:		AUS	(L)	30 ppm		2 h	acc. STERN, 1986
		AUS	(L)	10 ppm		8 h	acc. STERN, 1986
		B	(L)	6 mg/m^3		8 h	acc. MEINL et al., 1985
		B	(L)	15 mg/m^3		1 h	acc. MEINL et al., 1985
		BG	(L)	3 mg/m^3		30 min$^{1)}$	acc. STERN, 1986
		BG	(L)	1 mg/m^3		24 h$^{1)}$	acc. STERN, 1986
		CH	(L)	8 mg/m^3		24 h	acc. BUB, 1986
		CDN	(L)	35 mg/m^3		2 h	acc. STERN, 1986
		CDN	(L)	15 mg/m^3		8 h	acc. STERN, 1986
			(L)				

	CS	(L)	6 mg/m^3		30 min	acc. STERN, 1986
	CS	(L)	1 mg/m^3		24 h	acc. STERN, 1986
	D	L	10 mg/m^3	MIK	Long-time value2)	acc. BAUM, 1988
	D	L	50 mg/m^3	MIK	Short-time value3)	acc. BAUM, 1988
	D	L	10 mg/m^3	IW 1		acc. TA Luft, 1986
	D	L	30 mg/m^3	IW 2		acc. TA Luft, 1986
	DDR	(L)	3 mg/m^3	MIK$_D$		acc. HORN, 1989
	DDR	(L)	5 mg/m^3	MIK$_K$		acc. HORN, 1989
	E	(L)	45 mg/m^3		30 min	acc. STERN, 1986
	E	(L)	15 mg/m^3		8 h	acc. STERN, 1986
	GB	(L)	10 mg/m^3		8 h	acc. BUB, 1986
	GB	(L)	40 mg/m^3		1 h	acc. BUB, 1986
	GR	(L)	15 mg/m^3		8 h, smog warning	acc. MEINL et al., 1985
	GR	(L)	25 mg/m^3		8 h, smog alarm	acc. MEINL et al., 1985

					stage 1	
	GR	(L)	35 mg/m^3		8 h, smog alarm stage II	acc. MEINL et al., 1985
	H	(L)	1 mg/m^3		30 min5)	acc. STERN, 1986
	H	(L)	3 mg/m^3		30 min1)	acc. STERN, 1986
	H	(L)	6 mg/m^3		30 min6)	acc. STERN, 1986
	I	(L)	40 mg/m^3		2 h	acc. STERN, 1986
	I	(L)	10 mg/m^3		8 h	acc. MEINL et al., 1985
	IL	(L)	30 ppm		30 min	acc. STERN, 1986
	IL	(L)	10 ppm		8 h	acc. STERN, 1986
	J	(L)	10 ppm		24 h	acc.STERN, 1986
	J	(L)	20 ppm		8 h	acc. STERN, 1986
	J	(L)	58 mg/m^3		1 h, priority stage II	acc. MEINL et al., 1985
	N	(L)	25 mg/m^3		3 h	acc. STERN, 1986
	N	(L)	10 mg/m^3		8 h	acc. STERN, 1986
	NL	(L)	40 mg/m^3		2 h	acc. STERN, 1986

	NZ	(L)	30 ppm		2 h	acc. STERN, 1986
	NZ	(L)	10 ppm		24 h	acc. STERN, 1986
	RC	(L)	1 ppm		60 min	acc. STERN, 1986
	RP	(L)	30 ppm		2 h	acc. STERN, 1986
	RP	(L)	9 ppm		8 h	acc. STERN, 1986
	SA	(L)	40 mg/m^3		2 h	acc. STERN, 1986
	SA	(L)	10 mg/m^3		8 h	acc. STERN, 1986
	SF	(L)	40 mg/m^3		2 h	acc. STERN, 1986
	SF	(L)	10 mg/m^3		8 h	acc. STERN, 1986
	SU	(L)	3 mg/m^3		30 min1)	acc. STERN, 1986
	TJ	(L)	6 mg/m^3		6 min	acc. STERN, 1986
	TJ	(L)	2 mg/m^3		24 h	acc. STERN, 1986
	WHO	G	10 mg/m^3		8 h	acc. UBA, 1988
	WHO	G	30 mg/m^3		1 h	acc. UBA, 1988
	WHO	G	60 mg/m^3		1/2 h	acc. UBA, 1988

		YU	(L)	10 mg/m^3		30 min3)	acc. STERN, 1986
		YU	(L)	30 mg/m^3		30 min4)	acc. STERN, 1986
		YU	(L)	40 mg/m^3		1 h	acc. STERN, 1986
		YU	(L)	10 mg/m^3		8 h	acc. STERN, 1986
		YV	(L)	10 mg/m^3		8 h	acc. STERN, 1986
	Workp	D	L	33 mg/m^3	MAK		DFG, 1989
	Workp	DDR	(L)	55 mg/m^3		Long-time value	acc. HORN, 1989
	Workp	DDR	(L)	110 mg/m^3		Short-time value	acc. HORN, 1989
	Workp	SU	(L)	20 mg/m^3			acc. SORBE, 1989
	Workp	USA	(L)	55 mg/m^3	TWA		ACGIH, 1986
	Workp	USA	(L)	440 mg/m^3	STEL		ACGIH, 1986
	Workp	D	L	5 %7)	BAT	Whole blood, end of shift	DFG, 1989

Notes:

1) **Designated protection areas**

2) **1/2 hour mean value (may be exceeded once per month at most)**

3) **Annual arithmetic mean for human health**

4) **98% value of 1/2 hour mean values in one year**

5) **Specially designated protection areas**

6) **Areas other than those designated/specially designated as protection areas**

7) **CO haemoglobin**

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Air:			
Rural areas	DDR	0.01-0.9 mg/m^3	acc. HORN, 1989
Atmosphere up to altitude of 10 km		0.15 mg/m^3	acc. HORN, 1989
Urban areas	DDR	10-60 mg/m^3 (daily average)	acc. HORN, 1989
Berlin, daily mean value	D	15 mg/m^3	acc. UBA, 1977
Cologne, daily mean value	D	12 mg/m^3	acc. UBA, 1977
Tunnels, garages	DDR	115-570 mg/m^3	acc. HORN, 1989

Gasworks, mines	DDR	< 660 mg/m^3	acc. HORN, 1989

Assessment/comments

Carbon monoxide is released into the environment by combustion processes particularly in road traffic. In view of the fact that inhaled carbon monoxide does considerable harm to humans and animals, the emissions must be regulated by filters and catalysts. In addition to its toxic effect, carbon monoxide is probably partly responsible for changes in the world climate (higher atmospheric temperatures) due to its rapid oxidation to form carbon dioxide.

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Chlordane

DESIGNATIONS

CAS No.: 12789-03-6
Registry name: Chlordane
Chemical name: 1,2,4,5,6,7,8,8-Octachloro-3a,4,7,7a-tetrahydro-4,7-endo-methanoindan
Synonyms, Trade names:
Chemical name (German): Chlordan, 1,2,4,5,6,7,8,8-Octachlor-3a,4,7,7a-tetrahydro-4,7-endo-methanoindan
Chemical name (French): Chlordane

Appearance: **brown, viscous and odourless liquid (technical product)**

Note: **The technical product contains 60-75% chlordane isomers and 25-40% related compounds (mainly two heptachloro isomers (10-20%)). It is substantially the α -isomer.**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$C_{10}H_6Cl_8$
Rel. molecular mass:	409.83 g
Density:	1.59-1.63 g/cm^3 (technical, 25C)
Boiling point:	not distillable
Melting point:	102-104C α -isomer 104-106C β -isomer
Vapour pressure:	1.3 x 10^{-3} Pa at 25C
Solvolysis/solubility:	in water: 9 g/l (technical product) miscible with most aliphatic and aromatic organic solvents including mineral oils

ORIGIN AND USE

Usage:

Chlordane is widely used as a soil insecticide for termite control and as a household insecticide for the control of

ants and cockroaches together with a limited agricultural use as a soil insecticide. It is used as a feed, contact, or respiratory poison. In Germany, the use of chlordane has been prohibited since 1971; in the USA its registration was withdrawn in 1976.

Origin/derivation:
Chlordane is manufactured by chlorinating hexachlorodicyclopentadiene which is obtained by Diels-Alder reaction. There are no natural sources of chlordane.

Toxicity

Mammals:		
Rat	LD_{50} 250 mg/kg, oral	acc. PERKOW, 1983
	LD_{50} 217 mg/kg, dermal	acc. PERKOW, 1983
Mouse	LD_{50} 430 mg/kg, oral	acc. PERKOW, 1983
	LD_{50} 153 mg/kg, dermal	acc. SAX, 1984
Rabbit	LD_{50} 300 mg/kg, oral	acc. PERKOW, 1983
	LD_{50} 780-840 mg/kg	acc. PERKOW, 1983
Aquatic organisms:		
Rainbow trout	LC_{50} 0.022 ppm (96h)	acc. VERSCHUEREN,1983

Fathead minnow	LC_{50} 36.9 g/l (96h)	acc. VERSCHUEREN, 1983
	ECLo 0.32 g/l	acc. VERSCHUEREN, 1983
Bluegill	LC_{50} 0.022 ppm (96h)	acc. VERSCHUEREN, 1983
Insects:		
Bees	Toxic	acc. PERKOW, 1983

Characteristic effects:

Humans/mammals: **Poisoning may be due to oral intake, inhalation or resorption through the skin (readily). Chlordane irritates the skin and the respiratory tract. Symptoms of acute poisoning are vomiting, dizziness, tremors, disturbances of balance and convulsions. The toxic effect occurs very quickly: a case of death has been reported following the skin contact of 100 ml of a solution containing 25% chlordane before medical aid could be obtained. Symptoms are of longer duration with chlordane than with DDT under similar conditions. Chlordane is accumulated in fatty tissue, the liver and the kidneys. Chronic poisoning results in severe damage to these organs. Chlordane is suspected of having a carcinogenic potential.**

ENVIRONMENTAL BEHAVIOUR

Soil:
Due to its very low water solubility and its resistance to degradation, chlordane has to be considered as very persistent in soil. The average half-life is estimated at 2-4 years [ULLMANN, 1989] depending on the type of soil.

Water:

Chlordane has to be classified as highly persistent in water. Experiments in a sealed glass jar in sunlight and under artificial fluorescent light (initial concentration: 10 g/l) resulted in residues of 85% after 8 weeks. In addition, chlordane is extremely toxic to fish and bioaccumulates in fish up to 10^{5-}fold, thus entering the food chain.

Degradation/Decomposition products:

In the organism of mammals (rats), the elimination of hydrogen chloride and the formation of chlorohydrine and hydroxylated, hydrophilic metabolites of unknown composition take place.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	**Sector**	**Country/organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Water:	Drinkw	EC	L	0.1 g/l		single substance	acc. LAU-BW, 1989
	Drinkw	EC	L	0.5 g/l		sum of pesticides	acc. LAU-BW, 1989
	Drinkw	D	L	0.1 g/l		single substance	acc. LAU-BW, 1989
	Drinkw	D	L	0.5 g/l		sum of pesticides	acc. LAU-BW, 1989
Air:	Workp	D	L	0.5 mg/m^3	MAK	Skin, suspected of carcinogenic potential	acc. AUER TECHNIKUM, 1988
	Workp	SU	(L)	0.01 mg/m^3	PDK	Skin	acc. AUER TECHNIKUM, 1988
			(L)				acc. AUER

	Workp	USA		0.5 mg/m^3	TWA	Skin	TECHNIKUM, 1988

Note:
USA: Withdrawal of registration in 1976 [ULLMANN, 1989]
Germany: Use of chlordane has been prohibited since 1971 [PERKOW, 1983]

Assessment/comments

Like all organochlorine insecticides, chlordane is extremely persistent in the environment and is highly toxic to aquatic organisms and mammals. In addition, it has a bioaccumulative effect. Thus, the use of chlordane has been prohibited in several countries.

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Chlorinated naphthalenes

DESIGNATIONS

CAS No.: **70776-03-3**
Registry name: **Chlorinated naphthalenes**
Chemical name: **Chlorinated naphthalenes**
Synonyms, Trade names; **Chloronaphthalene, chloronaphthalene derivatives, PCN, Halowax**

Chemical name (German): **Chlorierte Naphthaline**
Chemical name (French): **Naphthalnes chlors**
Appearance: **Except for 1-monochloronaphthalene, which is a liquid at room temperature, pure chlorinated naphthalenes are colourless, crystalline compounds**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$C_{10}H_{8-n}Cl_n$ (n=1-8)
Rel. molecular mass:	162.5-404.0 g
Density:	1.2 - 2.0 g/cm^3
Boiling point:	250 - 440C
Melting point:	-25 - 197C
Flash point:	135 - >430C
Solvolysis/solubility:	soluble in aromatic solvents
Note:	The chemical structure of the molecules results in 75 chlorinated naphthalene congeners. The physical and chemical properties are dependent on the degree of chlorination. Whereas the melting and boiling point increase with an increase in the number of chlorine atoms, there is a simultaneous reduction in vapour pressure and water solubility.

ORIGIN AND USE

Usage:

Chlorinated naphthalenes are used in the electrical industry and as additives. Only isomeric mixtures characterised

by their chlorine content are commercially available.

Origin/derivation:

The substances are not found naturally. They are obtained by batchwise chlorination of naphthalene in the presence of ferric chloride as a catalyst.

Production figures:

Worldwide approximately 5,000 t per annum with a declining tendency (acc. KOCH, 1989).

Toxicity

Humans:	TCLo 30 mg/m^3, inhalation (trichloronaphthalene)	acc. KOCH, 1989
Mammals:		
Rat:	LD$_{50}$ 1,540 mg/kg, oral	acc. KOCH, 1989
	LD$_{50}$ 868 mg/kg, oral (2-chloronaphthalene)	acc. KOCH, 1989
Mouse:	LD$_{50}$ 1,091 mg/kg, oral	acc. KOCH, 1989
	LD$_{50}$ 2,087 mg/kg, oral (2-chloronaphthalene)	acc. KOCH, 1989

Characteristic effects:

Humans/mammals: **Poisoning is due to oral application (foodstuffs) or inhalation. The toxicity is largely governed by the degree of chlorination. Chloracne and liver damage are especially caused by pentachloro- and hexachloronaphthalene while chlorinated naphthalenes with 1 to 3 chlorine atoms hardly have any toxic effects.**

ENVIRONMENTAL BEHAVIOUR

Chlorinated naphthalenes are very resistant to degradation. Mobility is slight in water and soil, but there is a pronounced tendency towards bioaccumulation and geoaccumulation depending on the degree of chlorination. Highly chlorinated naphthalenes are very persistent. Incomplete combustion results in the formation of polychlorinated dibenzodioxins and -furans (PCDD/F).

ENVIRONMENTAL STANDARDS

Recommended limits in the USA for drinking water (acc. KOCH, 1989):
Trichloronaphthalene isomers: 3.9 μ g/l
Tetrachloronaphthalene isomers: 1.5 μ g/l
Pentachloronaphthalene isomers: 0.39 μ g/l
Hexachloronaphthalene isomers: 0.15 μ g/l
Octachloronaphthalene isomers: 0.08 μ *g/l*
(Refer also to 'naphthalene')

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Air		up to 25 ng/m^3	acc. KOCH, 1989
Surface water		up to 200 ng/l	acc. KOCH, 1989

Seawater		up to 20 ng/l	acc. KOCH, 1989
Sediments/soil		up to 100 g/kg	acc. KOCH, 1989
Fish		up to 0.12 g/kg	acc. KOCH, 1989
Birds		up to 70 g/kg	acc. KOCH, 1989
Humans (fatty tissue)		up to 15 g/kg	acc. KOCH, 1989

Assessment/comments

Similarly to chlorinated biphenyls, the toxicity and the environmental persistence of chlorinated naphthalenes increase with the degree of chlorination. The use of naphthalene with a high degree of chlorination should therefore be kept to a minimum.

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Chlorine

DESIGNATIONS

***CAS No.:* 7782-50-5**

Registry name: **Chlorine**
Chemical name: **Chlorine**
Synonyms, Trade names: **Chlorum**
Chemical name (German): **Chlor**
Chemical name (French): **Chlore**
Appearance: **greenish-yellow gas with pungent irritating odour**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	Cl_2
Rel. molecular mass:	70.91 g
Density:	0.567 g/cm^3 (liquid, critical density at 144 C); 3.21 g/l (gas) at 0C, 1013 hPa
Relative gas density:	2.49
Boiling point:	-34.05C
Melting point:	-100.98C
Vapour pressure:	6.8 bar at 20C
Odour threshold:	0.05 ppm
Solvolysis/solubility:	in water: 7.3 g/l at 20C

	in tetrachloromethane: 176.5 g/l at 19C
Conversion factors:	1 mg/m^3 = 0.339 ml/m^3
	1 ml/m^3 = 2.947 mg/m^3

ORIGIN AND USE

Usage:
Chlorine is used for numerous applications in the chemical industry, for example in the manufacture of chlorinated organic products (synthetics, solvents, insecticides, herbicides). It is also used in the cellulose/paper industry and in laundries as a bleaching agent. Drinking-water and swimming-pool water disinfection is a further application.

Origin/derivation:
In nature, chlorine is chiefly found as chloride (bonded to sodium, potassium and magnesium). A further important chlorine compound is hydrogen chloride. Nowadays, chlorine is primarily produced by electrolysis of alkali-metal chlorides (mercury method and diaphragm method). Use is also still made of chemical methods (SHELL chlorine method, KEL chlorine process, SOUTHWEST POTASH method).

Production figures (worldwide):
1975 = 22.5 million tons; 1983 = 30 million tons; 1994 = >40 million tons

Toxicity

Humans:	LCLo 837 ppm/30 min, inhalation	acc. UBA, 1986
		acc. TAB. CHEMIE,

	2.5 mg/l air = immediately lethal	1980
Mammals:		
Rat	LC_{50} 293 ppm/1 h, inhalation	acc. UBA, 1986
Mouse	LC_{50} 137 ppm/1 h, inhalation	acc. UBA, 1986
Guinea pig	LCLo 330 ppm/7 h, inhalation	acc. UBA, 1986
Dog	LCLo 800 ppm/30 min, inhalation	acc. UBA, 1986
Aquatic organisms:		
Trout	0.08 ppm/ 168 h/ freshwater; TLm	acc. UBA, 1986
Fish	more than 0.05 mg/l lethal	

Characteristic effects:

Humans/mammals: **Chlorine is an extremely caustic, poisonous gas. Symptoms of poisoning following inhalation are the irritation of the mucous membranes of the respiratory tract combined with breathing difficulties, coughing up of blood and low pulse rate. Humans become accustomed to odour and irritation if exposed for a long time or repeatedly. Symptoms may be delayed. Liquid chlorine is extremely caustic on the skin.**

Plants: **Any description of the damage to plants generally relates to the chloride impact although chlorine gas is also absorbed by plant leaves and destroys plant tissue partly by oxidation and partly by expulsion of hydrogen in**

organic compounds.

ENVIRONMENTAL BEHAVIOUR

Water:

Water hazard class 2. Destroys all aquatic life; kills bacteria as long as free chlorine can be detected. Chlorine reacts with water forming hydrogen chloride/hydrochloric acid depending on water content. Chlorine corrodes several materials when wet.

Air*:*

On expansion, chlorine gas (compressed gas) forms a cold mist which is denser than air; poisonous, corrosive mixtures form in air over the surface of water.

Soil:

Chlorine is only found in soil in ionised form of its salts (chlorides).

ENVIRONMENTAL STANDARDS

Medium/acceptor	**Sector**	**Country/organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Air:		D	L	0.1 mg/m^3	IW 1	1)	acc. TA Luft, 1986
		D	L	0.3 mg/m^3	IW 2	2)	acc. TA Luft, 1986
		D	G	0.10 mg/m^3		Monthly average, plants	acc. LAU-BW

	Emiss.	**D**	**L**	**5 mg/m^3**		**mass flow > 50 g/h**	**acc. TA Luft, 1986**
	Workp	D	L	1.5 mg/m^3	MAK	Peak limit I	DFG, 1989
	Workp	D	L	1.5 mg/m^3	BAT		acc. LAU-BW
	Workp	DDR	L	1 mg/m^3			acc. LAB. CHEMIE, 1980
	Workp	SU	(L)	1 mg/m^3			acc. SORBE, 1988
	Workp	USA	(L)	3 mg/m^3	TWA		acc. SORBE, 1988
	Workp	USA	(L)	9 mg/m^3	STEL		acc. SORBE, 1988

Notes:

1) Annual arithmetic mean

2) 98 % of annual 30-minute average

Assessment/comments

Chlorine is a hazard to all types of water and in particular to drinking water because of its high toxicity and corrosiveness as well as its oxidising effect. Disaster alarm must be given if large quantities of the gas are liberated.

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Chloroform

DESIGNATIONS

CAS No.: 67-66-3
Registry name: Chloroform
Chemical name: Trichloromethane
Synonyms, Trade names: Methyl trichloride, methane trichloride, formyl trichloride, Haloform, R20
Chemical name (German): Chloroform, Trichlormethan
Chemical name (French): Chloroforme
Appearance: clear, colourless, highly refractive liquid with sweet odour

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$CHCl_3$
Rel. molecular mass:	119.4 g
Density:	1.48 g/cm^3
Relative gas density:	4.12
Boiling point:	61C

Melting point:	-63C
Vapour pressure:	21 x 10^3 Pa
Solvolysis/solubility:	in water: 8.1 g/l miscible with solvents such as benzene, pentane, hexane, ethanol and diethyl ether
Conversion factors:	1 ppm = 4.96 mg/m^3 1 mg/m^3 = 0.20 ppm

ORIGIN AND USE

Usage:
Most of the total production of chloroform is used to produce monochlorodifluoromethane (CFC22) which is used as a refrigerant but also as an intermediate in the manufacture of tetrafluoroethene which then may be polymerised (PTFE). Chloroform is also used in the production of dyes, pharmaceuticals and pesticides. Its use as a solvent and application in anaesthesia is rapidly on the decline.

Origin/derivation:
90 - 95 % of chloroform is produced either by the hydrochlorination of methanol or by the chlorination of methane. 5 - 10 % is of natural origin and found in seawater (reaction of methyl iodide with inorganic chlorine). Chlorine bleaching of cellulose produces large quantities of chloroform. Considerable amounts likewise result from water chlorination. Commercially available chloroform contains, amongst other substances, bromochloromethane, bromodichloromethane, methylene chloride, tetrachloromethane, 1,2-dichloroethane, trichloroethylene and tetrachloroethane as contaminants.

Production figures:

Worldwide 1973:	245,000 - 300,000 t	acc. KOCH, 1989
D 1982:	35,500 t	acc. UBA, 1986

Emissions:

The production and application-related emissions amount to some 10,000 t; this is supplemented by annual impacting of surface and groundwater with a further 10,000 t/a (acc. KOCH, 1989).

Toxicity

Humans:	5,000 mg/m^3 after 7 min. dizziness, head congestion	acc. BUA, 1985
	20,000 mg/m^3 feeling faint	acc. BUA, 1985
	69,440 mg/m^3 deep full anaesthesia	acc. BUA, 1985
	> 79,360 mg/m^3 apnoea, death	acc. BUA, 1985
	LDLo 10 ppm, inhalation (1 a)	acc. KOCH, 1989
Mammals:		
Rat:	LD_{50} 1,194 mg/kg, oral	acc. DVGW, 1988

	LCLo 8,000 ppm, inhalation (4 h)	acc. BUA, 1985
Mouse:	LD$_{50}$ 80 mg/kg, oral	acc. BUA, 1985
	LD$_{50}$ 28,000 mg/kg, inhalation	acc. BUA, 1985
Dog:	LD$_{50}$ 1,100 mg/kg, oral	acc. DVGW, 1988
Rabbit:	LDLo 500 mg/kg, oral	acc. BUA, 1985
Guinea pig:	LCLo 20,000 ppm, inhalation (2 h)	acc. BUA, 1985
Aquatic organisms:		
Golden orfe:	LC$_{50}$ 162-191 mg/l (48 h)	acc. UBA, 1986
Rainbow trout:	LC$_{50}$ 18-66.8 mg/l (96 h)	acc. UBA, 1986
Blue perch:	LC$_{50}$ 18-115 mg/l (96 h)	acc. UBA, 1986
Water flea:	LC$_{50}$ 28.9 mg/l (48 h)	acc. UBA, 1986

Characteristic effects:

***Humans/mammals:* Intake is most frequently by inhalation with some of the substance being resorbed in the lungs and the remainder being exhaled. Oral application likewise results in the majority being exhaled or discharged by**

the kidneys.

Chloroform damages the liver and the kidneys, the central nervous system and the heart. Large doses have a narcotic effect.

Chloroform has proved to be carcinogenic in animal experiments. Mutagenic or teratogenic effects have not been established to date.

ENVIRONMENTAL BEHAVIOUR

Water:
The degradation of chloroform in water is extremely slow (water hazard class 3). The high volatility results in degasification over surface water. Bioaccumulation is minimal despite the high fat solubility of chloroform (bioconcentration factor in fish: 6; UBA, 1986).

Air:
Chloroform ingresses into the atmosphere because of its high volatility and is accumulated there in small quantities. The substance is decomposed by photolysis.

Soil:
There is no adsorption on particles thus precluding the possibility of accumulation in soil or sediment.

Degradation, decomposition products:
Biological degradation under anaerobic conditions results in carbon dioxide, chloride and methane; degradation in the organism produces carbon dioxide, chloride and phosgene. The presence of oxidising agents causes chloroform to decompose with phosgene and hydrochloric acid being formed (DVGW, 1988).

Food chain:
Chloroform is ubiquitous and thus may also be found in foodstuffs. Drinking water sometimes contains high

chloroform concentrations caused by water chlorination. The daily chloroform absorption is estimated at an average of 10 μ g/person with one quarter each resulting from drinking water and foodstuffs and the other half from the atmosphere (DVGW, 1988).

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/ organ.	Status	Value	Cat.	Remarks	Source
Water:	Surface	D/NL	(L)	1 mg/l	IAWR	For natural treatment	acc. DVGW, 1988
	Surface	D/NL	(L)	5 mg/l	IAWR	For phys.-chem. treatment	acc. DVGW, 1988
	Drinkw	CDN	(L)	350 g/l		1978	acc. DVGW, 1988
	Drinkw	CH	(L)	25 g/l		1)	acc. DVGW, 1988
	Drinkw	D	(G)	25 g/l		BGA commission1)	acc. DVGW, 1988
	Drinkw	USA	(L)	100 g/l		Sum of trihalomethanes	acc. DVGW, 1988
	Drinkw	WHO	G	30 g/l			acc. DVGW, 1988
Air:	Emiss.	D	L	20 mg/m^3		mass flow > 100 g/h	acc. TA Luft, 1986

Foodstuffs:		D	L	25 g/l		Table, mineral water	acc. DVGW, 1988

Notes:

1) Sum of chloroform, bromoform, bromodichloromethane, dibromochloromethane

2) Substance with a suspected carcinogenic potential
The use of chloroform in cosmetics, medicines and pesticides is banned in Germany

Comparison/reference values

Medium/origin	**Country**	**Concentration**	**Source**
Water:			
Rhine (Wiesbaden, 1986)	D	0.35-2.1 g/l	acc. DVGW, 1988
Rhine (Lobith, 1985)	D	0.5-4 g/l	acc. DVGW, 1988
Main (Sindlingen, 1983)	D	21 g/l	acc. DVGW, 1988
Moselle (1983)	D	0.5-0.7 g/l	acc. DVGW, 1988
Elbe (1983)	D	0.6-9.8 g/l	acc. DVGW, 1988
North Sea coast (Emden)	D	0.56-3.8 g/l	acc. UBA, 1986
Baltic coast	D	0.06-0.17 g/l	acc. UBA, 1986
Sediment:			

Ruhr (1972-1981)	D	1-3 mg/kg	acc. DVGW, 1988
Air:			
Basic atmospheric impact		0.05-0.1 g/m^3	acc. KOCH, 1989
Urban areas		up to 74 g/m^3	acc. KOCH, 1989

Assessment/comments

On account of its ubiquitous distribution and the considerable quantities emitted into the environment every year, chloroform has to be considered as an environmental hazard. There is no information to date on the effect and concentration in soils and soil organisms and the genotoxic potential likewise remains unclear. In particular, water chlorination should be avoided as far as possible in order to reduce the pollution of groundwater and drinking water without neglecting the necessity of hygienic prevention measures.

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Chlorophenols

DESIGNATIONS

CAS No.:
Registry name: **Chlorophenols**
Chemical name: **Chlorophenols**
Synonyms, Tradenames:
Chemical name (German): **Chlorphenole**
Chemical name (French): **Chlorophnols**
Appearance: **colourless solids (except o-chlorophenol) with offensive odour**

Note: **The group of the chlorophenols consists of 19 different compounds. As pentachlorophenol (PCP) is the most important compound in this group, the basic chemical and physical data refer to PCP.**

CAS No.:	87-86-5
Registry name:	Pentachlorophenol
Chemical name:	Pentachlorophenol
Synonyms, Tradenames:	PCP, 2,3,4,5,6-Pentachlorophenol, Dowicide G, Dowicide 7, Penta, Santophen 20, Pentacon, Penchlorol, Pentakil, Sontobrite, Weedone
Chemical name (German):	Pentachlorphenol
Chemical name (French):	Pentachlorophnol
Appearance:	colourless solid with offensive odour

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	C_6Cl_5OH
Rel. molecular mass:	266.35 g
Density:	1.978 g/cm^3
Boiling point:	300C (decomposition)
Melting point:	190C
Vapour pressure:	23 x 10^{-3} Pa
Solvolysis/solubility:	in water: 20 mg/l soluble in alcohol, ether, acetone, benzene
Conversion factors:	1 ppm = 11.1 mg/m^3 1 mg/m^3 = 0.09 ppm

ORIGIN AND USE

Usage:

Because of their broad-spectrum antimicrobial properties, chlorophenols have been used as preservative agents for wood, paints, vegetable fibres and leather and as disinfectants. In addition, they are used as herbicides, fungicides and insecticides and as intermediates in the production of pharmaceuticals and dyes.

Origin/derivation:

Most of the commercially important chlorophenols are obtained by direct chlorination of phenol using chlorine gas. In the technical product, there are impurities of other chlorophenol isomers or chlorophenols with more or less chlorine. The heavy chlorophenols are mainly contaminated by polychlorophenoxyphenols, chlorodibenzoparadioxins and chlorodibenzofurans. Emissions are mainly due to the manufacture, storage, transportation and application of chlorophenols.

Production figures:

World (excluding former COMECON and China)	100,000 t/a
heavy chlorophenols (4-5 chlorine atoms)	35-40,000 t/a
light chlorophenols (1-3 chlorine atoms)	60,000 t/a

(figures taken from ULLMANN 1985)

Toxicity

Humans:	LD 50-500 mg/kg, oral (estimated)	
Mammals:		
Rat	LD$_{50}$ 670 mg/kg, oral (2-chlorophenol)	acc. ULLMANN 1986
	LD$_{50}$ 950 mg/kg, percutaneous (2-chlorophenol)	acc. ULLMANN 1986

Rat		
Rat	LD_{50} 570 mg/kg, oral (3-chlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 1030 mg/kg, percutaneous (3-chlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 261 mg/kg, oral (4-chlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 1390 mg/kg, percutaneous (4-chlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 580 mg/kg, oral (2,4-dichlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 1730 mg/kg, percutaneous (2,4-dichlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 820 mg/kg, oral (2,4,5-trichlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 2260 mg/kg, percutaneous (2,4,5-trichlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 1620 mg/kg, oral (2,4,5-trichlorophenol, sodium salt)	acc. ULLMANN 1986
Rat	LD_{50} 820 mg/kg, oral (2,4,6-trichlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 140 mg/kg, oral (2,3,4,6-tetrachlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 210 mg/kg, percutaneous (2,3,4,6-tetrachlorophenol)	acc. ULLMANN 1986

Rat	LD_{50} 50 mg/kg, oral (pentachlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 100 mg/kg, percutaneous (pentachlorophenol)	acc. ULLMANN 1986
Rat	LD_{50} 210 mg/kg, oral (pentachlorophenol,sodium salt)	acc. ULLMANN 1986
Rat	LD_{50} 72 mg/kg, percutaneous (pentachlorophenol, sodium salt)	acc. ULLMANN 1986
Aquatic organisms:		
Golden orfe	LC_{50} 0.60 mg/l (96 h), pentachlorophenol	acc. RIPPEN 1990
Rainbow trout	LC_{50} 0.12-0,26 mg/l (96 h), pentachlorophenol	acc. RIPPEN 1990
Water flea	LC_{50} 0.33-0.41 mg/l (96 h), pentachlorophenol	acc. RIPPEN 1990
Bacteria (div.)	NOEC 12.3 mg/l (30 min), growth	acc. RIPPEN 1990
Algae	EC_{50} 10-7000 g/l (96h), growth, pentachlorophenol	acc. RIPPEN 1990

Characteristic effects:

Humans/mammals: **Chlorophenols can be absorbed through the lungs, the gastro-intestinal tract and the skin. Some 80% is excreted via the kidneys without undergoing any transformation.**

The toxicity of chlorophenols depends upon the degree of chlorination, the position of the chlorine atoms and the

purity of the sample. Chlorophenols have an irritating effect on the eyes and on the respiratory tract. Toxic doses of chlorophenols cause convulsions, shortness of breath, coma and finally death. After repeated administration, toxic doses may result in damage to the inner organs (primarily liver) and the bone marrow.

Pentachlorophenol has a toxic effect on embryos in animal experiments (lethal at higher concentrations). Technical PCP may possibly be carcinogenic not least due to contamination. Mutagenic potential can not be excluded.

ENVIRONMENTAL BEHAVIOUR

Water:

In the aquatic environment, chlorophenols may be dissolved in free or complexed form or adsorbed on suspended matter. Removal is mainly by way of biodegradation which is rapid when adapted microorganisms are already present. However, PCP is biodegraded much more difficultly than other chlorophenols. Chlorophenols are also removed from water by photodecomposition and volatilisation. Finally, adsorption of chlorophenols on suspended matter plays a role in the amount of chlorophenols in water: light chlorophenols are hardly fixed whereas PCP is fixed very strongly.

Air:

PCP ingresses into the atmosphere on account of its volatility. Volatility increases considerably with increasing temperature, but is likewise dependent on possible additives and e.g. the nature of the treated wood. Burning wood treated with PCP liberates polychlorinated dibenzodioxins and -furans (PCDD/F).

Soil:

The persistence of chlorophenols in soil depends on their adsorption-desorption characteristics. Only the adsorption of PCP has been studied in depth. It is fixed very strongly on soil particles and not easily washed off by rain. Apart from adsorption and desorption, rapid seepage paths can play an important role in the transportation of PCP in soil. Once it reaches the groundwater, it is doubtful whether PCP is degraded. Since 1984 there has been a ban in Germany on the storage of waste products from the manufacture of PCP on open dumps to avoid the

infiltration of PCP-contaminated seepage water.

Degradation, decomposition products, half-life:

Free PCP or PCP dissolved in water is photomineralised within a few days when exposed to sunlight (even more so with adsorption on solids). There is no such degradation if PCP has reached lower soil levels or the groundwater. The degradation in water is always dependent on the pH and the temperature. It is subject to pronounced fluctuations (example: half-life at pH 5.1 = 328 h, at pH 6 = 3,120 h (at 30C in both cases)). Although PCP may be degraded by microorganisms under certain conditions, the substance is to be classed as scarcely biodegradable. Quinone forms in the course of degradation and the process may involve complete mineralisation.

Food chain:

Bioaccumulation in aquatic ecosystems does not only appear to be extremely type-specific, but also dependent to a large extent on the biotope as well as the duration and the intensity of exposure. Excretion is likewise type/organ-specific with half-lives ranging between 7 hours and 7 days. There are contradictory findings as to whether PCP is absorbed directly from the water by fish and other aquatic organisms or whether the food chain is involved. Plants may accumulate PCP stored in the soil over the course of several vegetation periods.

ENVIRONMENTAL STANDARDS

Medium/acceptor	**Sector**	**Country/organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Water:	Drinkw	D	L	0.1 g/l			acc. DVGW, 1988
	Drinkw	EC	L	0.1 g/l			acc. EC, 1980
	Groundw	D(HH)	G	0.3 g/l		Individual substance[1)]	acc. LAU-BW, 1989

Groundw	D(HH)	G	1.5 g/l		Individual substance$^{2)}$	acc. LAU-BW, 1989
Groundw	D(HH)	G	0.5 g/l		Substance group$^{1)}$	acc. LAU-BW, 1989
Groundw	D(HH)	G	2 g/l		Substance group$^{2)}$	acc. LAU-BW, 1989
Groundw	NL	G	0.25 g/l		Monochlorophenol (sum) Reference	acc. TERRA TECH 6/94
Groundw	NL	L	100 g/l		Monochlorophenol (sum) Intervention	acc. TERRA TECH 6/94
Groundw	NL	G	0.08 g/l		Dichlorophenol (sum) Reference	acc. TERRA TECH 6/94
Groundw	NL	L	30 g/l		Dichlorophenol (sum) Intervention	acc. TERRA TECH 6/94
Groundw	NL	G	0.025 g/l		Trichlorophenol (sum) Reference	acc. TERRA TECH 6/94

Groundw	NL	L	10 g/l		Trichlorophenol (sum) Intervention	acc. TERRA TECH 6/94
Groundw	NL	G	0.01 g/l		Tetrachlorophenol (sum) Reference	acc. TERRA TECH 6/94
Groundw	NL	L	10 g/l		Tetrachlorophenol (sum) Intervention	acc. TERRA TECH 6/94
Groundw	NL	G	0.02 g/l		Pentachlorophenol (sum) Reference	acc. TERRA TECH 6/94
Groundw	NL	L	3 g/l		Pentachlorophenol (sum) Intervention	acc. TERRA TECH 6/94
Surface	D	G	1 g/l		Individual substance3)	acc. LAU-BW5), 1989
Surface	D	G	5 g/l		Individual substance3)	acc. LAU-BW6), 1989

	Surface	D	G	2 g/l		Substance group[4)]	acc. LAU-BW[5)], 1989
	Surface	D	G	10 g/l		Substance group[4)]	acc. LAU-BW[6)], 1989
Soil:		NL	L	10 mg/kg AD		Substance group, Intervention	acc. TERRA TECH 6/94
		NL	L	5 mg/kg AD		Pentachlorophenol, Intervention	acc. TERRA TECH 6/94
Air:	Workp	D	L	0.5 mg/m^3	MAK		acc. DFG, 1989
	Workp	SU	(L)	0.1 mg/m^3			acc. DVGW, 1988
	Workp	USA	(L)	0.5 mg/m^3	TWA		acc. DVGW, 1988

Notes:

1) The groundwater quality should be investigated in depth

2) Rehabilitation measures should be taken

3) For drinking water treatment using natural methods

[4] **For drinking water treatment using chemical and physical methods**

[5] **Closer investigation necessary**

[6] **Rehabilitation measures necessary**

The use of all chlorophenols have been banned in Sweden since 1978.

Assessment/comments

Chlorophenols should be avoided where at all possible on account of the high toxicity level for aquatic organisms in some cases. The chlorination of drinking water to sterilise river water is problematic since this can lead to the formation of chlorophenols with penetrating odour and taste. Technical PCP contains impurities of other chlorinated phenols and aromatic substances as well as traces of polychlorinated dibenzo-p-dioxins and -furans (PCDD/F); it is thus one of the major sources of the ingress of these substances into the environment. Part of the toxicity of PCP can be attributed to such impurities.

Refer also to information sheets for 'phenols'.

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Chromium

DESIGNATIONS

CAS No.: **7440-47-3**
Registry name: **Chromium**
Chemical name: **Chromium**
Synonyms, Trade names: **Chromium**
Chemical name (German): **Chrom**
Chemical name (French): **Chrome**
Appearance: **silver grey, hard, ductile metal**

BASIC CHEMICAL AND PHYSICAL DATA

Chemical symbol: **Cr**
Rel. atomic mass: **51.996 g**
Density: **7.19 g/cm^3 at 20C**
Boiling point: **2672C**
Melting point: **1857C**
Vapour pressure: **10^{-6} Pa at 844C**
Solvolysis/solubility: **soluble in diluted hydrochloric and sulphuric acid**

BASIC DATA OF SELECTED COMPOUNDS

CAS No:	7789-12-0	1333-82-0
Chemical name:	Sodium dichromate (dihydrate)	Chromium(VI) oxide
Synonyms, Trade names:		Chromium trioxide, chromic acid anhydride

Chemical name (German):	Natriumdichromat (Dihydrat)		Chrom(VI)-oxid, Chromtrioxid	
Chemical name (French):	Dichromate de sodium		Oxyde de Chrome (VI)	
Appearance:	orange to red needles		dark red crystals (usually flakes) odourless	
Empirical formula:	$Na_2Cr_2O_7$ (2 H_2O)		CrO_3	
Rel. molecular mass:	261.98 g (anhydrous) 298.0 g (dihydrate)		99.99 g	
Density:	2.35-2.52 g/cm^3		2.7 g/cm^3	
Boiling point:	above 400C decomposition		not distillable	
Melting point:	357C (above 86C conversion to anhydrous salt)		198C (decomposition: brownish red vapours with pungent smell)	
Vapour pressure:	0 hPa		0 hPa	
Solvolysis/solubility:	in water:	73.18 wt% at 20C	in water:	1660g/l at 20C
		77.09 wt% at 40C		1990 g/l at 90C
		82.04 wt% at 60C		((ρ) chromic acid)
		88.39 wt% at 80C		
	in alcohol: insoluble			

ORIGIN AND USE

Usage:

Chromium is used as a catalyst in ammonia synthesis, in the production of chromium steels, stainless steels and chromium alloys and for electroplating. Organic complexes are used as development dyes in colour photography. Inorganic chromium compounds are used as paint pigments. Chromium(VI) salts are widely used in wood preservation and leather tannery.

Origin/derivation:

Chromium virtually only occurs in nature in the form of compounds. The most important chromium ore is chromite. Pure chromium is obtained by reducing chromium(III) oxide with aluminium (thermite method), by way of electrolysis or using chromium iodide.

Production figures:

1985 = 9.935 million t (worldwide)

Toxicity

Humans:	0.5-1 g, oral = lethal, (potassium chromate)	acc. MERIAN, 1984
	LD 6-8 g, oral (sodium dichromate)	acc. KOCH, 1989
Mammals:		
Rat	LD_{50} 1800 mg/kg, oral (chromium(III)chloride)	acc. MERIAN, 1984
Rat	LD_{50} 3250 mg/kg, oral (chromium(III)nitrate)	acc. MERIAN, 1984

Aquatic organisms:		
Freshwater fish	LC_{50} 250-400 mg/l (Cr^{VI})	acc. MERIAN, 1984
Saltwater fish	LC_{50} 170-400 mg/l (Cr^{VI})	acc. MERIAN, 1984
Daphnia	LC_{50} 0.05 mg/l (Cr^{VI})	acc. MERIAN, 1984
Algae	LC_{50} 0.032-6.4 mg/l (Cr^{VI})	acc. MERIAN, 1984
Brown and rainbow trout	0.20-0.35 mg/l (Cr^{VI})	acc. DVGW, 1988
Fish (no spec. differentiation)	LD 60-728 mg/l, (sodium dichromate)	acc. KOCH, 1989

Characteristic effects:

Humans/mammals: **Because of its insolubility, metallic chromium is not toxic in water. The various hexavalent chromium compounds represent the major risk especially due to their genetic effects. Chromium(VI) compounds are active in virtually all test systems designed to determine mutagenic action. There is a strong risk for embryos and foetuses because of the proven placenta passage. The carcinogenic effect of chromium(VI) compounds has been substantiated both in animal experiments and by epidemiological studies on groups of the population subject to workplace exposure. The corresponding latency times are given as between 10 and 27 years. In contrast to chromium(VI) compounds there is no clear-cut evidence of the carcinogenic effect of chromium(III) compounds. Acute poisoning with chromium(VI) compounds becomes apparent for example in the form of damage to the**

kidneys. Chronic poisoning results in changes in the gastro-intestinal tract as well as in accumulation in the liver, kidneys, thyroid gland and bone marrow. The rate of excretion is slow.

Plants: **There are known cases of plant root damage for example caused mainly by chromium(VI). There are major differences in terms of chromium absorption and possible damage both amongst the various types of plant and within the different parts of a plant. The toxic effect of chromium on plants has mainly been described on the basis of batch tests. The findings for oats revealed that the roots remained small and the leaves narrow whilst at the same time exhibiting reddish brown discoloration and small necrotic blotches.**

Note: **Trivalent chromium is an important trace element for humans and animals in the insulin metabolism.**

ENVIRONMENTAL BEHAVIOUR

Water:

The toxicity of soluble chromium compounds in aquatic systems varies depending on the temperature, pH and water hardness as well as on the species of the organism. Chromium(VI) compounds are readily soluble in water, but are readily reduced under natural conditions in the presence of organic, oxidisable material to form less water-soluble, stable chromium(III) compounds.

Soil:

The mobility of chromium in the pedosphere can only be assessed taking into account the adsorptive and reducing capacity of soils and sediments. Once they have become sedimented and established in aquatic sediment, chromium(III)hydroxides exhibit only a minimal tendency to remobilise since there is scarcely any natural oxidation of chromium(III) compounds to form chromium(VI) compounds. Relatively small concentrations of chromium(VI) are sufficient to be toxic with the pH value of the soil playing a major role. The use of fertilisers containing phosphate increases the amount of chromium entering the soil.

Food chain:

Chromium(III) compounds absorbed with food are relatively harmless whereas chromium(VI) compounds have an extremely toxic effect. Animals and humans normally only absorb small amounts of chromium through inhalation; the majority of substances containing chromium are absorbed with food and drinking water. Resorption in the intestines is largely dependent on the chemical appearance of chromium: up to approx. 20 - 25 % of chromium complexes are absorbed whereas the figure for inorganic chromium is roughly 0.5 % (MERIAN, 1984).

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/ organ.	Status	Value	Cat.	Remarks	Source
Water:	Drinkw	D	L	50 g/l			acc. KOCH, 1989
	Drinkw	WHO	G	50 g/l			acc. KOCH, 1989
	Groundw	D(HH)	G	0.050 g/m^3		Investigation	acc. LAU-BW, 1989
	Groundw	D(HH)	G	0.20 g/m^3		Rehabilitation	acc. LAU-BW, 1989
	Groundw	NL	G	1 μg/l		Reference	acc. TERRA TECH, 6/94
	Groundw	NL	L	30 μg/l		Intervention	acc. TERRA TECH, 6/94
	Waste water	D	G	2 mg/l			acc. KOCH, 1989
Soil:		CH	G	75 mg/kg		Soil	acc. LAU-BW, 1989
		NL	G	100 mg/kg		Reference	acc. TERRA TECH, 6/94
		NL	L	380 mg/kg		Intervention	acc. TERRA TECH, 6/94

	Sewage sludge	D	L	100 mg/kg AD		Soil	acc. LAU-BW, 1989
	Sewage sludge	D	L	1200 mg/kg DM		Sewage sludge	acc. LAU-BW, 1989
	Sewage sludge	CH	L	1000 mg/kg DM		Sewage sludge	acc. LAU-BW, 1989
	Sewage sludge	EC	L	1-3 mg/kg DM		Soil	acc. LAU-BW, 1989
	Sewage sludge	EC	L	20-40 mg/kg DM		Sludge	acc. LAU-BW, 1989
	Sewage sludge	EC	L	1.5 kg/ha		Load in 10 a	acc. LAU-BW, 1989
	Compost	D	G	100 mg/kg AD		Soil	acc. LAU-BW, 1989
	Compost	D	G	2 kg/(ha*a)		Compost	acc. LAU-BW, 1989
	Compost	CH	L	150 mg/kg DM		Compost	acc. LAU-BW, 1989
	Compost	D(HH)	G	300 mg/kg DM		Investigation	acc. LAU-BW, 1989
Air:	Emiss.	D	L	5 mg/m^3		mass flow > 25 g/h1)	acc. TA Luft, 1986

Emiss	D	L	1 mg/m^3		mass flow > 5 g/h$^{2)}$	acc. TA Luft, 1986
Workp	AUS	L	1 mg/m^3		Cr and insoluble Cr compounds	acc. MERIAN, 1984
Workp	AUS	L	0.5 mg/m^3		Soluble salts	acc. MERIAN, 1984
Workp	B	L	0.5 mg/m^3		Soluble salts	acc. MERIAN, 1984
Workp	CH	L	1 mg/m^3		Cr and insoluble Cr compounds	acc. MERIAN, 1984
Workp	CH	L	0.5 mg/m^3		Soluble salts	acc. MERIAN, 1984
Workp	DDR	L	0.5 mg/m^3		Cr and insoluble Cr compounds	acc. MERIAN, 1984
Workp	I	L	0.5 mg/m^3		Cr and insoluble Cr compounds	acc. MERIAN, 1984
Workp	NL	L	0.5 mg/m^3		Soluble salts	acc. MERIAN, 1984
Workp	SF	L	0.5 mg/m^3		Soluble salts	acc. MERIAN, 1984

	Workp	SF	L	1 mg/m^3		Cr and insoluble Cr compounds	acc. MERIAN, 1984
	Workp	SU	(L)	1 mg/m^3		Chromium oxide	acc. KETTNER, 1979
	Workp	USA	(L)	0.5 mg/m^3	TWA	Metallic Cr	acc. ACGIH, 1979
	Workp	USA	(L)	0.5 mg/m^3	TWA	Cr(III)	acc. ACGIH, 1979
	Workp	USA	(L)	0.5 mg/m^3	TWA	Cr(VI), water soluble	acc. ACGIH, 1979
	Workp	USA	(L)	0.5 mg/m^3	TWA	Cr(VI), water insoluble	acc. ACGIH, 1979
	Workp	YU	L	1 mg/m^3		Cr and insoluble Cr compounds	acc. MERIAN, 1984

Notes:

[1] **Cr(VI)-compounds, stated as Cr**

[2] **Cr(VI)-compounds in respirable forms, stated as Cr**

Alkali chromates: have a suspected carcinogenic potential

Comparison/reference values

Medium/origin	Country	Value	Source
Atmosphere	worldwide	5 pg/m^3	acc. KOCH, 1989
Surface/groundwater	worldwide	0.5 g/l	acc. KOCH, 1989
Fly ash (coal)	USA	43-259 mg/kg	acc. HOCK, 1988
Plants		0.2-1 mg/kg	acc. HOCK, 1988

Assessment/comments

The amounts of chromium found in the hydrosphere, pedosphere, atmosphere and biosphere can primarily be attributed to industrial emissions. The natural emissions into the atmosphere are quoted as being roughly 58,000 t per annum, whereas anthropological emissions are almost up to 100,000 t/a.

As regards environmental behaviour, chromium(III) compounds can be said to have a high degree of stability in contrast to chromium(VI) compounds.

Refuse containing chromium should be critically viewed in particular on account of its behaviour in the geological substrate on waste tips. Chromates are estimated to be stable for up to 50 years in an alkaline milieu and migrate even through tenacious soils to reach the groundwater.

The incineration of sludges containing chromium(III) should therefore be avoided on account of possible chromate formation.

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Cobalt

DESIGNATIONS

CAS No.: 7440-48-4
Registry name: Cobalt
Chemical name: Cobalt
Synonyms, Trade names: Cobalt
Chemical name (German): Cobalt
Chemical name (French): Cobalt
Appearance: steel-grey, shiny, ferromagnetic metal

BASIC CHEMICAL AND PHYSICAL DATA

Chemical symbol: Co
Rel. atomic mass: 58.93 g
Density: 8.85 g/cm^3 at 25C
Boiling point: 2800C +/- 50C
Melting point: 1495C
Vapour pressure: <10^{-5} Pa at 1250C, >10^5 Pa at 3200C

Solvolysis/solubility: **readily soluble in diluted, oxidising acids**

ORIGIN AND USE

Usage:

Dyeing of glass, ceramics and enamel by Co compounds. Production of temperature-resistant, abrasion-proof and non-corroding alloys (stellite). The synthetic radioactive isotope ^{60}Co is used in nuclear engineering and nuclear medicine (treatment of tumours) as well as in other countries to preserve foodstuffs. Co is used in the chemical industry in homogeneous and heterogeneous catalysis to synthesise fuels (Fischer-Tropsch method) as well as alcohols and aldehydes (hydroformylation). A further use is found in the cemented carbides.

Origin/derivation:

Co makes up 0.0023% of the Earth's crust and is found together with Cu, Ni and Fe ores. In most cobalt-containing ores, arsenic is present, too. Co is manufactured by the partial roasting of sulphide ores in the presence of fluxing agents. The resulting raw material contains Cu, Ni and Co sulphides and arsenides at higher concentrations. Further processing then involves heating the raw material in the presence of NaCl.

Production figures:

In 1980: 32,700 t (worldwide) [ULLMANN, 1986]

Toxicity

Mammals:		
Rat	LD_{50} 1750 mg/kg, (Co oxide)	acc. Ullmann, 1986
Rat	LD_{50} 821 mg/kg, (Co acetate)	acc. Ullmann, 1986

Rat	LD_{50} 766 mg/kg, (Co chloride)	acc. Ullmann, 1986
Rat	LD_{50} 691 mg/kg, (Co nitrate)	acc. Ullmann, 1986
Rat	LD_{50} 630 mg/kg, (Co carbonate)	acc. Ullmann, 1986
Rat	LDLo 1500 mg/kg, oral	acc. UBA, 1986
Rat	LDLo 100 mg/kg, intravenous	acc. UBA, 1986
Rabbit	LDLo 20 mg/kg, oral	acc. UBA, 1986
Rabbit	LDLo 100 mg/kg, intravenous	acc. UBA, 1986
Aquatic organisms:		
Daphnia	1-9 mg/l = critical level, (Co chloride)	acc. LAU-BW, 1989

Characteristic effects:

Humans/mammals: **Overdoses reduce the activity of the thyroid gland and can cause the formation of goitres. The number of erythrocytes in the blood increases (polycythemia), the blood vessels are temporally enlarged and blood coagulation is impaired. The nervous system is also frequently affected. Heart damage and pulmonary fibrosis (chron.) may occur. The most important aspect in toxicological terms is the inhalation of Co dust (proven carcinogenic effect; ROTH, 1989) and the hazard of sensitisation (ROTH, 1989). In addition, there is corrosion of the throat and the gastrointestinal tract if cobalt dusts have been inhaled. Poisoning causes disfunctions of the liver and kidneys.**

Plants: **A surplus of Co results in an Fe and Cu deficit (toxic effect = displacement effect). There is an increase in the number of chlorotic leaves which become necrotic and then die off.**

ENVIRONMENTAL BEHAVIOUR

Air:
Cobalt is stable at normal temperatures in air. When heated, it oxidises and burns at white heat to form Co_3O_4.

Soil:
The average Co content is 8 mg/kg. The solubility depends on the pH. There is more elution in acid soils. Cobalt is bonded above all to Mn and Fe oxides with the result that only small quantities are available and thus mobile.

Degradation, decomposition products, half-life:
Renal discharge of inhaled cobalt: majority with a half-life of 10 days, remainder with half-life of 90 days (MERIAN, 1984).

Food chain:
The absorption of cobalt in drinking water is insignificant. Generally, the atmosphere is likewise only contaminated by traces of cobalt. Humans absorb approx. 140 - 580 μ g of cobalt per day. Between 20% and 95% are resorbed. However, most of the Co absorbed does not take the form of the necessary vitamin B_{12}, but is encountered rather as inorganic Co bonded to foodstuff constituents. The absorption of inorganic Co is linked to that of iron.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/ organ.	Status	Value	Cat.	Remarks	Source
Water:	Surface	D	G	0.05 mg/l		1) For A + B	acc. LAU-BW, 1989

	Groundw	D	(G)	50 g/l		Investigation	acc. LAU-BW, 1989
	Groundw	D	(G)	200 g/l		Rehabilitation	acc. LAU-BW, 1989
	Groundw	NL	G	20 μg/l		Reference	acc. TERRA TECH, 6/94
	Groundw	NL	L	100 μg/l		Intervention	acc. TERRA TECH, 6/94
	Waste water	CH	(L)	0.05 mg/l			acc. LAU-BW, 1989
	Waste water	CH	(L)	0.50 mg/l		Direct/indirect introduction	acc. LAU-BW, 1989
	Irrigation	D	G	0.20 mg/l		Field cultivation	acc. LAU-BW, 1989
	Irrigation	D	G	0.20 mg/l		Cultivation under glass	acc. LAU-BW, 1989
	Irrigation	USA	(L)	0.20 mg/l			acc. LAU-BW, 1989
	Irrigation	USA	(L)	10 mg/l		2)	acc. LAU-BW, 1989
Soil:		CH	G	25 mg/kg			acc. LAU-BW, 1989
		D	(G)	50 mg/kg		Investigation	acc. LAU-BW, 1989
		D	G	300 mg/kg		Rehabilitation	acc. LAU-BW, 1989
		D	G	800 mg/kg			acc. HOCK, 1988

		NL	G	20 mg/kg		Reference	acc. TERRA TECH, 6/94
		NL	L	240 mg/kg		Intervention	acc. TERRA TECH, 6/94
		USA	G	8000 mg/kg		TTLC	acc. DVGW, 1988
		USA	G	80 mg/kg		STLC	acc. DVGW, 1988
	Sewage sludge	CH	L	100 mg/kg			acc. LAU-BW, 1989
Air:	Emiss.	D	L	1 mg/m^3		mass flow > 5 g/h3)	acc. TA Luft, 1986
	Workp	AUS	L	0.1 mg/m^3			acc. MERIAN, 1984
	Workp	B	L	0.01 mg/m3			acc. MERIAN, 1984
	Workp	BG	L	0.5 mg/m^3			acc. MERIAN, 1984
	Workp	CH	L	0.1 mg/m^3			acc. MERIAN, 1984
	Workp	CS	L	0.1 mg/m^3		Mean value	acc. MERIAN, 1984
	Workp	CS	L	0.3 mg/m^3		Short-time value	acc. MERIAN, 1984
	Workp	D	L	0.5 mg/m^3	TRK	use of powders	acc. LAU-BW, 1994

	Workp	D	L	0.1 mg/m^3	TRK	all other uses	DFG, 1994
	Workp	DDR	L	0.1 mg/m^3		Mean value	acc. MERIAN, 1984
	Workp	DDR	L	0.1 mg/m^3		Short-time value	acc. MERIAN, 1984
	Workp	SF	L	0.1 mg/m^3			acc. MERIAN, 1984
	Workp	I	L	0.1 mg/m^3		C, S	acc. MERIAN, 1984
	Workp	NL	L	0.1 mg/m^3			acc. MERIAN, 1984
	Workp	PL	L	0.5 mg/m^3			acc. MERIAN, 1984
	Workp	RO	L	0.2 mg/m^3		Mean value	acc. MERIAN, 1984
	Workp	RO	L	0.2 mg/m^3		Short-time value	acc. MERIAN, 1984
	Workp	S	L	0.1 mg/m^3			acc. MERIAN, 1984
	Workp	SU	L	0.5 mg/m^3			acc. MERIAN, 1984
	Workp	USA	(L)	0.1 mg/m^3	TWA	Emission class 3	acc. LAU-BW, 1989
	Workp	YU	L	0.1 mg/m^3			

Notes:

1) For drinking water treatment in each case: A = impact limits up to which drinking water can be produced solely by natural methods; B = impact limits up to which drinking water can be produced with the aid of currently tried-and-tested chemophysical methods

2) Only suitable for short-term irrigation on certain soils.

3) Dustlike/aerosolic Co and its compounds in respirable forms, stated as Co

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Water:			
Lake Constance	D	< 0.2 g/l	acc. DVGW, 1988
Rhine (Mainz):	D	6-12 g/l	acc. DVGW, 1988
Ruhr (Duisburg):	D	< 1 g/l	acc. DVGW, 1988
Seawater		0.1 g/l	acc. DVGW, 1988
Sediment:			
Lake Constance	D	5.7-18.9 mg/kg	acc. DVGW, 1988
Rhine (Wiesbaden):	D	20 mg/kg	acc. DVGW, 1988
Ruhr (Wetter):	D	25 mg/kg	acc. DVGW, 1988

Fly ash (coal)	USA	5-73 mg/kg	acc. HOCK, 1988
Plants		0.3-0.5 mg/kg	acc. HOCK, 1988

Assessment/comments

Cobalt is the central atom in vitamin B_{12} and is an important trace element. The hazards emanating from cobalt compounds are slight when compared to other heavy metals. From the toxicological point of view it is important to avoid the inhalation of cobalt dusts.

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Copper

DESIGNATIONS

CAS No.: **7440-50-8**
Registry name: **Copper**
Chemical name: **Copper**
Synonyms, Trade names: **Copper, cuprum**
Chemical name (German): **Kupfer**
Chemical name (French): **Cuivre**

Appearance: **ductile, soft metal with distinctive reddish colour**

BASIC CHEMICAL AND PHYSICAL DATA

Chemical symbol: **Cu**
Rel. atomic mass: **63.55 g**
Density: **8.9 g/cm^3 at 20C**
Boiling point: **2580C**
Melting point: **1083C**
Vapour pressure: **0 mbar at 20C, 0.073 Pa at 1083C, 0.133 Pa at 1870C**
Solvolysis/solubility: **the metal is only directly attacked by oxidising acids (nitric acid, hot concentrated sulphuric acid).**

BASIC DATA OF SELECTED COMPOUNDS

CAS No:	7758-99-8	1317-39-1
Chemical name:	Copper(II) sulphate pentahydrate	Copper(I) oxide
Synonyms, Trade names:	Bluestone, blue vitriol	Copper oxide
Chemical name (German):	Kupfersulfat - Pentahydrat	Kupfer(I)-oxid, Kupferoxid
Chemical name (French):	Sulfate de cuivre	Oxyde de cuivre(I)
Appearance:	light blue crystalline powder	yellow to red crystalline powder depending on preparation and particle size
Empirical formula:	$CuSO_4$ $5H_2O$	Cu_2O

Rel. molecular mass:	249.61 g		143.09 g
Density:	2.285 g/cm^3		5.8-6.2 g/cm^3
Boiling point:	not distillable		above 1800C decomposition
Melting point:	decomposition (above 88C: subsequent elimination of water)		1235C
Vapour pressure:	0 Pa		0 Pa
Solvolysis/solubility:	in water:	148 g/l at 0C	in water: virtually insoluble;
		231 g/l at 25C	in dilute sulphuric/nitric acid disproportion into soluble copper(II) salts;
		335 g/l at 50C	insoluble in most organic solvents
	in methanol: 156 g/l		
	in ethanol: insoluble		

ORIGIN AND USE

Usage:
Copper is used as a conductor in the electrical industry; for heating and cooling pipes, as container material and as an alloying metal; in the form of Cu_2O as antifouling paint (for ships' hulls); in the form of $CuSO_4$ as a fungicide and algicide as well as to combat types of mildew; as whitewash to ward off vine pest and as a fertiliser in the form

of $CuSO_4 5 H_2O$ or Cu_2O.

Origin/derivation:

Copper is found in elementary form and in ores such as chalcopyrite ($CuFeS_2$), chalcocite (Cu_2S) and cuprite (Cu_2O). Copper is usually purified today using electrolytic refining methods. Fused-salt processes only account for roughly 10%. The copper in sulphide ores is generally separated by means of floatation.

Production figures:

In 1986: 513 million t (worldwide) [FISCHER, 1989]

Toxicity

Humans:	700-2100 mg/g dry liver tissue = lethal	acc. SORBE, 1986
Mammals:		
Rat	LD_{50} 159 mg/kg , oral, (Cu carbonate)	acc. DVGW, 1988
Rat	LD_{50} 140 mg/kg , oral, (Cu chloride)	acc. DVGW, 1988
Rat	LD_{50} 470 mg/kg , oral, (Cu oxide)	acc. DVGW, 1988
Rat	LD_{50} 300 mg/kg , oral, (Cu sulphate)	acc. DVGW, 1988
Aquatic organisms		
Daphnia	LD 0.8 mg/l (18 h), (Cu sulphate)	acc. DVGW, 1988

Trout	LD 0.8 mg/l (2-3 d), (Cu sulphate)	acc. DVGW, 1988
Blue algae	0.03 mg/l Cu_{2+} = damage, (Cu sulphate)	acc. DVGW, 1988
Green algae	1.1 mg/l Cu_{2+} = damage, (Cu sulphate)	acc. DVGW, 1988

Cu is a powerful fish poison, the active concentration of which depends on the make-up of the water. The toxic effect is enhanced still further by cadmium, zinc and mercury.

Characteristic effects:

***Humans/mammals:* As a constituent part of numerous enzymes, Cu is an essential trace element. Poisoning mainly results from the inhalation of Cu dusts and Cu fumes. Poisoning caused by oral intake is rare since it causes nausea. The toxic effect is due to the bonding of free Cu ions with certain proteins and the resulting impairment of their physiological functions. Inhalation of fumes and dust displaces the blood in the nasal and mucous membranes and can cause perforation of the nasal septum. Infants are at a higher risk (possible mortality) where there is a high copper content in the drinking water. Death may be caused by cirrhosis of the liver.**

***Plants:* Damage to roots which attacks the plasmalemma and destroys the normal membrane structure; inhibited root growth and formation of numerous short, brownish secondary roots. Cu becomes accumulated in the skin of the roots and in the cell walls. Chlorosis is produced by Cu displacing Fe from centres of metabolic physiology. In the same ecosystem aquatic plants absorb three times more Cu than plants on dry land.**

ENVIRONMENTAL BEHAVIOUR

Water:

Cu is precipitated in saltwater thus explaining its low content compared to freshwater. Acid rain increases the solubility of copper ores. A high copper level in drinking water with low pH can usually be attributed to pipe corrosion. High copper levels can discolour the water and cause greenish deposits.

Air:

Cu is assigned to emission class 3 in the TA-Luft (ROTH, 1989). A greenish patina forms when Cu is exposed to damp air thus protecting the metal against further chemical action (corrosion).

Soil:

There is considerable retention of Cu by inorganic exchangers. Complex formation takes place with increasing pH. The solubility of Cu in soil is at its lowest at pH 5-6. Cu is subject to extreme accumulation in clay mineral layers. The Cu content in soil decreases with increasing depth of soil. Exchange reactions and the nitrogen content in the soil are important factors in the passive transport of immobile copper.

Degradation, decomposition products, half-life:

Cu(II) salts are the most stable Cu compounds.

Food chain:

Mammals and humans absorb 30% of copper in food via the stomach with 5% actually being resorbed and the rest being excreted again by way of the gallbladder. There is accumulation in the liver, brain and kidneys.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/organ.	Status	Value	Cat.	Remarks	Source
Water:	Drinkw	CH	(L)				acc. LAU-BW,

			1.5 mg/l			1989
Drinkw	EC	G	0.1 mg/l		1)	acc. DVGW, 1988
Drinkw	EC	G	3 mg/l			acc. LAU-BW, 1989
Drinkw	SU	(L)	0.1 mg/l			acc. LAU-BW, 1989
Drinkw	USA	(L)	1 mg/l			acc. LAU-BW, 1989
Drinkw	WHO	G	1 mg/l			acc. LAU-BW, 1989
Groundw	D(HH)	G	0.05 mg/l		Investigation	acc. LAU-BW, 1989
Groundw	D(HH)	G	0.2 mg/l		Rehabilitation	acc. LAU-BW, 1989
Groundw	NL	G	15 μ g/l		Reference	acc. TERRA TECH, 6/94
Groundw	NL	L	75 μ g/l		Intervention	acc. TERRA TECH, 6/94
Surface	D	G	0.05 mg/l		2) B	acc. DVGW, 1988

Surface	D	G	0.30 mg/l		3) A	acc. DVGW, 1988
Surface	EC	G	0.02 mg/l		4) A_1	acc. LAU-BW, 1989
Surface	EC	G	0.05 mg/l		4) A_1	acc. LAU-BW, 1989
Surface	EC	G	0.05 mg/l		5) A_2	acc. LAU-BW, 1989
Surface	EC	G	1 mg/l		6) A_3	acc. LAU-BW, 1989
Surface	EC	G	0.04 mg/l		Salmonoid waters	acc. LAU-BW, 1989
Waste water	CH	G	0.01 mg/l		Quality goal	acc. LAU-BW, 1989
Waste water	CH	(L)	0.5 mg/l		Direct/indirect introduction	acc. LAU-BW, 1989
Waste water	D	G	2 mg/l			acc. LAU-BW, 1989
Irrigation	D	G	0.2 mg/l		Field cultivation	acc. LAU-BW, 1989
		G			Cultivation	acc. LAU-BW,

	Irrigation	D		0.05 mg/l		under glass	1989
	Irrigation	GB	G	0.5 mg/l			acc. LAU-BW, 1989
	Irrigation	USA	(L)	0.2 mg/l			acc. LAU-BW, 1989
	Irrigation	USA	(L)	5 mg/l		7)	acc. LAU-BW, 1989
	Troughw	D	G	0.01 mg/l			acc. LAU-BW, 1989
	Troughw	GB	G	0.2 mg/l			acc. LAU-BW, 1989
	Troughw	USA	(L)	1 mg/l		Cattle breeding	acc. LAU-BW, 1989
Soil:		CH	G	50 mg/kg		Total content	acc. LAU-BW, 1989
		CH	G	0.7 mg/kg		Soluble content	acc. LAU-BW, 1989
		D(HH)	(G)	300 mg/kg		Investigation	acc. LAU-BW, 1989
		NL	G	36 mg/kg AD		Reference	acc. TERRA TECH, 6/94

	NL	L	190 mg/kg AD		Intervention	acc. TERRA TECH, 6/94
Sewage sludge	CH	L	1000 mg/kg DM			acc. LAU-BW, 1989
Sewage sludge	D	L	100 mg/kg AD			acc. LAU-BW, 1989
Sewage sludge	D	L	1200 mg/kg DM			acc. LAU-BW, 1989
Sewage sludge	EC	L	50-140 mg/kg DM		Soil	acc. LAU-BW, 1989
Sewage sludge	EC	L	1000-1750 mg/kg DM			acc. LAU-BW, 1989
Fertiliser	D	L	200 mg/kg		8)	acc. LAU-BW, 1989
Compost	A	G	100-1000 ppm DM			acc. LAU-BW, 1989
Compost	CH	L	150 mg/kg DM			acc. LAU-BW, 1989
Compost	D	G	100 mg/kg AD		Soil	acc. LAU-BW, 1989
		G				acc. LAU-BW,

	Compost	D		2000 g/(haa)			1989
Air:	Emiss.	D	L	20 mg/m^3		Smoke, 9)	acc. LAU-BW, 1989
	Emiss.	D	L	75 mg/m^3		Smoke, 10)	acc. LAU-BW, 1989
	Workp	AUS	L	1 mg/m^3		Dust	acc. MERIAN, 1984
	Workp	AUS	L	0.1 mg/m^3		Smoke	acc. MERIAN, 1984
	Workp	B	L	1 mg/m^3		Dust	acc. MERIAN, 1984
	Workp	B	L	0.2 mg/m^3		Smoke	acc. MERIAN, 1984
	Workp	D	L	0.1 mg/m^3	MAK	Smoke	DFG, 1989
	Workp	D	L	1 mg/m^3	MAK	Dust	DFG, 1989
	Workp	DDR	L	0.2 mg/m^3		Smoke, mean value	acc. MERIAN, 1984
	Workp	DDR	L	0.4 mg/m^3		Smoke, short-time value	acc. MERIAN, 1984

Workp	CH	L	1 mg/m^3		Dust	acc. MERIAN, 1984
Workp	CH	L	0.1 mg/m^3		Smoke	acc. MERIAN, 1984
Workp	I	L	1 mg/m^3		Dust	acc. MERIAN, 1984
Workp	I	L	0.2 mg/m^3		Smoke	acc. MERIAN, 1984
Workp	NL	L	1 mg/m^3		Dust	acc. MERIAN, 1984
Workp	NL	L	0.2 mg/m^3		Smoke	acc. MERIAN, 1984
Workp	PL	L	1 mg/m^3		Dust	acc. MERIAN, 1984
Workp	PL	L	0.1 mg/m^3		Smoke	acc. MERIAN, 1984
Workp	RO	L	0.5 mg/m^3		Dust, mean value	acc. MERIAN, 1984
Workp	RO	L	1.5 mg/m^3		Dust, short-time value	acc. MERIAN, 1984
Workp	RO	L	0.05 mg/m^3		Smoke, mean	acc. MERIAN,

						value	1984
	Workp	RO	L	0.15 mg/m^3		Smoke, short-time value	acc. MERIAN, 1984
	Workp	S	L	1 mg/m^3		Dust	acc. MERIAN, 1984
	Workp	SF	L	1 mg/m^3		Dust	acc. MERIAN, 1984
	Workp	SF	L	0.1 mg/m^3		Smoke	acc. MERIAN, 1984
	Workp	SU	(L)	0.5 mg/m^3			acc. LAU-BW, 1989
	Workp	USA	(L)	0.2 mg/m^3	TWA	Smoke	acc. LAU-BW, 1989
	Workp	USA	(L)	1 mg/m^3	TWA	Dust	acc. MERIAN, 1984
	Workp	YU	L	1 mg/m^3		Dust	acc. MERIAN, 1984
	Workp	YU	L	0.1 mg/m^3		Smoke	acc. MERIAN, 1984
Foodstuffs:	Pectin	CH	(L)	400 ppm			acc. DVGW, 1988
	Tinned		(L)				acc. DVGW,

	spinach	CH	(L)	100 ppm			1988
	Margarine	CH	(L)	100 ppm			acc. DVGW, 1988
	Fruit juice	CH	(L)	5-30 ppm			acc. DVGW, 1988
	Milk	CH	(L)	0.05 ppm			acc. DVGW, 1988
	Beer	CH	(L)	0.2 ppm			acc. DVGW, 1988

Notes:

1) On leaving pumping system

2) For drinking water treatment in each case: B = signifies impact limits up to which drinking water can be produced with the aid of currently tried and tested chemophysical methods

3) For drinking water treatment in each case: A = signifies impact limits up to which drinking water may be produced solely by natural methods

4) For drinking water treatment in each case: A1 = simple physical treatment and sterilisation

5) For drinking water treatment in each case: A2 = normal physical and chemical treatment/sterilisation

6) For drinking water treatment in each case: A3 = physical and refined chemical treatment, oxidation, adsorption and sterilisation

7) Only for short-term irrigation of certain soils

8) In organic, mineral mixed fertilisers

9) With mass flow 0.1 kg/h

10) With mass flow 3 kg/h

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Water:			
Lake Constance	D	0.75-1.1 mg/l	acc. DVGW, 1988
Rhine (Cologne):	D	5-17 mg/l	acc. DVGW, 1988
Rhine (Duisburg):	D	2.9-24.6 mg/l	acc. DVGW, 1988
Ruhr (Essen):	D	14-26 mg/l	acc. DVGW, 1988
Ruhr (Duisburg):	D	6-11 mg/l	acc. DVGW, 1988
Seawater		0.0005-0.03 mg/l	acc. HOCK, 1988
Sediment:			
Rhine	D	250 mg/kg	acc. DVGW, 1988
Ruhr	D	900 mg/kg	acc. DVGW, 1988
Fly ash, coal	USA	45-616 mg/kg	acc. HOCK, 1988
Sewage-sludge refuse	D	50-5000 mg/kg	acc. HOCK, 1988

compost			
Plants		2-12 mg/kg	acc. HOCK, 1988

Assessment/comments

Copper is an important trace element for all living organisms. Humans require approx. 2 mg/day. Poisoning is rare since large quantities cause nausea. However, certain compounds are highly toxic to aquatic organisms.

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Cresols

DESIGNATIONS

CAS No.: 1319-77-3
Registry names: Cresols
Chemical names: o-Cresol, m-cresol, p-cresol
Synonyms, Trade names: ortho-Cresol, meta-cresol, para-cresol, 1,2-cresol, 1,3-cresol, 1,4-cresol, hydroxytoluene, methylphenol, methylhydroxybenzene, tricresol
Chemical name (German): o-Cresol, m-Cresol, p-Cresol (also Kresol)
Chemical name (French): o-Crsol, m-crsol, p-crsol
Appearance: colourless or brownish liquid/crystals; similar smell to Lysol disinfectant

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	C_7H_8O		
Rel. molecular mass:	108.14 g		
Density:	1.03 g/cm^3		
Relative gas density:	3.74		
	meta	ortho	para
Boiling point:	203C	191C	202C
Melting point:	11C	31C	35C
Vapour pressure:	0.065 hPa	0.35 hPa	0.06 hPa
Flash point:	86C	81C	86C
Ignition temperature:	560C	555C	555C
Solvolysis/solubility:	2%	2%	2% (in water)
Conversion factors:	1 ppm = 4.49 mg/m^3 1 mg/m^3 = 0.22 ppm		

ORIGIN AND USE

Usage:

Cresols are used as disinfectants, perfumes, preserving agents or herbicides (98% DNOC, UCPA). They are also used in the textile industry as cleaning agents.

Origin/derivation:

Cresols are derived from coal or petroleum and can be found in wood and other biogenic materials. They are thus released into the environment by combustion processes in motor vehicles and domestic heating systems (asphalt abrasion, vaporisation of plastics, perfumes, metal degreasing etc.). The so called "crude cresol" which is obtained from the heavy oil of coal tar contains considerable amounts of m- and p-cresol.

Toxicity

Mammals:		
Rat:	LD_{50} 1.35 g/kg, oral (o-cresol)	acc. VERSCHUEREN. 1983
	LD_{50} 2.02 g/kg, oral (m-cresol)	acc. VERSCHUEREN. 1983
	LD_{50} 1.8 g/kg, oral (p-cresol)	acc. VERSCHUEREN. 1983
Rabbit:	LD_{50} 0.8 g/kg, oral (o-cresol)	acc. VERSCHUEREN. 1983
	LD_{50} 1.1 g/kg, oral (m-cresol)	acc. VERSCHUEREN. 1983
	LD_{50} 1.1 g/kg, oral (p-cresol)	acc. VERSCHUEREN. 1983
Aquatic organisms:		

Green algae:	LD_0 40 mg/l	acc. VERSCHUEREN. 1983
Blue algae:	LD_0 6.8 mg/l	acc. VERSCHUEREN. 1983
Water flea:	LD_0 16 mg/l (o-cresol)	acc. VERSCHUEREN. 1983
	LD_0 28 mg/l (m-cresol)	acc. VERSCHUEREN. 1983
	LD_0 12 mg/l (p-cresol)	acc. VERSCHUEREN. 1983
Goldfish:	TLm 49.1-19 mg/l (24-96h) (o-cresol)	acc. VERSCHUEREN. 1983
Carp:	TLm 30 mg/l (24h) (o-cresol)	acc. VERSCHUEREN. 1983
	TLm 25 mg/l (24h) (m-cresol)	acc. VERSCHUEREN. 1983
	TLm 21 mg/l (24h) (p-cresol)	acc. VERSCHUEREN, 1983

Characteristic effects:

***Humans/mammals:* Cresols have a disinfectant and corrosive effect due to the degradation of proteins. They ingress into the organism via the skin and mucous membranes and cause skin diseases. Resorptive paralysis of the central nervous system ultimately leads to liver and kidney disease. The intake of small quantities can result in dizziness or loss of consciousness, poisoning, delirium and the secretion of copius amounts of saliva and sweat. The symptoms of cresol poisoning are similar to those of phenol, producing white and subsequently brownish-black scabs on the skin.**

Plants: **Cresols inhibit the degradation of glucose.**

ENVIRONMENTAL BEHAVIOUR

Water:

Cresols sink in water and dissolve very slowly. Even if considerably diluted, they still form poisonous, corrosive mixtures which have a toxic effect on aquatic organisms. If cresols ingress into the groundwater the water will no longer be suitable for drinking. There may be accumulation in sediment due to the adsorption of cresols on clay minerals.

Air:

Strong heat will form explosive mixtures which are denser than air and creep along the ground. Cresols are therefore not transported into the atmosphere and are usually washed out again by precipitation. This effect can cause pollution of groundwater in the vicinity of large-scale emission sources. The majority of cresols are subject to photochemical degradation.

Soil:

Cresols are absorbed by plants and broken down by them. The accumulation of cresols in soil depends on the type of soil involved (adsorption on clay minerals).

Degradation, decomposition products:

Cresols are subject to photochemical degradation.

Food chain:

(refer to information sheets "phenols" and "chlorophenols")

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/ organ.	Status	Value	Cat.	Remarks	Source
Air:		D	L	0.2 mg/m^3	MIK	Long-time value	acc. BAUM, 1988
		D	L	0.6 mg/m^3	MIK	Short-time value	acc. BAUM, 1988
		DDR	(L)	0.03 mg/m^3		Short-time value	acc. HORN, 1989
		DDR	(L)	0.01 mg/m^3		Long-time value	acc. HORN, 1989
	Workp	D	L	22 mg/m^3	MAK		DFG, 1989
	Workp	DDR	(L)	40 mg/m^3		Short-time value	acc. HORN, 1989
	Workp	DDR	(L)	20 mg/m^3		Long-time value	acc. HORN, 1989
	Workp	SU	(L)	0.5 mg/m^3	PDK		acc. SORBE, 1989
	Workp	USA	(L)	22 mg/m^3	TWA		ACGIH, 1986

Notes:

In accordance with the TA-Luft, cresols are assigned to class 1 where the concentration may not exceed 20 mg/m^3 with a mass flow of 0.1 kg/h or more in the Federal Republic of Germany.
For further standards refer to "phenols ".

Assessment/comments

When handling cresols the skin should be protected and the substance is to be kept away from naked flames. Protective clothing and good ventilation have to be provided. Usage should be reduced as far as possible because of its considerable toxicity.

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DDT

DESIGNATIONS

CAS No.:	50-29-3
Registry name:	DDT
Chemical name:	1,1,1-Trichloro-2,2-bis(4-chlorophenyl)ethane
Synonyms, Trade names:	p,p'-Dichlorodiphenyltrichloroethane, p,p'-DDT, 1,1-bis(4-chlorophenyl)-2,2,2-trichloroethane, Dicophane (GB); Chlorophenothane (USA); Anofex; Cezarex; Dinocide; Gesarol; Guesapon; Guesard; Guesarol; Gyron; Ixodex; Neocid; Neocidol; Zerdane

Chemical name (German):	DDT, 1,1,1-Trichlor-2,2-bis(4-chlorphenyl)ethan
Chemical name (French):	DDT
Appearance:	colourless crystals ranging from odourless to slightly aromatic

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$C_{14}H_9Cl_5$
Rel. molecular mass:	354.49 g
Density:	1.55 g/cm^3
Melting point:	109C
Vapour pressure:	25.3 x 10^{-6} Pa
Solvolysis/solubility:	in water: 3 x 10^{-6} g/l readily soluble in numerous organic solvents and in fats
Conversion factors:	1 ppm = 14.7 mg/m^3 1 mg/m^3 = 0.07 ppm

ORIGIN AND USE

Usage:

Contact poison and feed insecticide for combating a wide range of insects (malaria and yellow-fever mosquito, plague flea, clothes louse, tsetse fly etc.). The substance was used on a large scale because of its broad-spectrum effectiveness, its low phytotoxicity, its good long-term effect and its low acute toxicity to warm-blooded animals. Whereas the use of DDT is banned today in most industrial nations, it is still used in numerous Third-World countries since the costs of substitute materials are much higher.

Origin/derivation:

DDT is not found naturally. It was synthesised for the first time in 1874 and has been commercially available by condensation of chloral with chlorobenzene since 1945.

Production figures:

Recent data are not available. The estimated production figures in 1974 (OECD) were about 60,000 t worldwide (WHO, 1979). DDT was originally manufactured in many countries, but by 1979 there were only 3 production facilities left, namely in America, India and France (WHO, 1979). The DVGW, on the other hand, quotes two companies in the EC alone (1988).

Emission figures:

Virtually all the DDT produced is released into the environment. The quantities applied are usually between 1 and 3 kg DDT per hectare; the products used contain between 1 and 10 % active agent.

Toxicity

Humans:	LD approx. 500 mg/kg, oral	acc. RIPPEN, 1989
Mammals:		

Rat	LD_{50} 113 mg/kg, oral	acc. RIPPEN, 1989
	LD_{50} 1900 mg/kg, dermal	acc. RIPPEN, 1989
Mouse	LD_{50} 150-300 mg/kg, oral	acc. DVGW, 1988
Dog	LD_{50} 150-750 mg/kg, oral	acc. DVGW, 1988
Cat	LD_{50} 150-600 mg/kg, oral	acc. DVGW, 1988
Insects:		
House fly (musca domestica)	LD_{50} 0.033 g/animal (24 h)	acc. KORTE, 1980
Aquatic organisms:		
Fish	LC_{50} 8-100 g/l (96 h)	acc. RIPPEN, 1989
Water flea (daphnia magna)	EC_{50} 0.36-4.4 g/l (24-48 h)	acc. RIPPEN, 1989
Algae (skeletonema costatum)	EC_{50} 100 g/l (7 d)	acc. DVGW, 1988

Characteristic effects:

***Humans/mammals:* The exact mechanism of action still has to be completely clarified. DDT is a neurotoxin which**

mainly acts on the central nervous system. The external symptoms indicate a temporary easing of the transmission of stimuli followed by blockage. Various enzymes are inhibited by DDT. It thus also acts as a respiratory poison. It accumulates in fatty tissue. Damage to the liver in particular with high doses; sustained exposure (rats) led to liver, kidney and spleen damage. Mutagenity and carcinogenity are highly likely in humans; carcinogenity discovered in animal experiments.

Plants: **DDT does not usually damage plants. Some sensitive plants show signs of impaired root growth in the event of DDT accumulation in the soil.**

Synergism: **Enhanced action (aquatic animals) due to lindane and alkylbenzene sulphonates.**

ENVIRONMENTAL BEHAVIOUR

Water:

DDT exhibits a strong tendency towards adsorption on solid particles in water. It thus accumulates in sediment and can be transported in running water over long distances.

Air:

DDT is found in the atmosphere as a gas, as an aerosol and adsorbed on dusts. It can still be detected 6 months after application in the air over fields on which it has been used. Adsorbed on dusts, it is sometimes transported over thousands of kilometres and thus dispersed worldwide. DDT has been detected not only in Antarctic snow, but also in precipitation over Scotland and the Shetland Islands. The concentrations found in rain permit to draw the conclusion that the distribution of DDT throughout the world is relatively uniform.

Soil:

When DDT is applied, a considerable amount ends up in the soil. Considerable accumulation has been discovered in the top few centimetres, thus concluding that the mobility in soil is relatively low.

Degradation, decomposition products:

Important metabolites of DDT are DDE (1,1-bis-(4-chlorophenyl)-2,2-dichloroethene), DDA and DDD. DDE is at least as toxic as DDT and would appear to be even more resilient in the environment.

There is little information to date regarding the degradation of DDT in soil. The relationship between biological and chemical decomposition reactions remains to be explained. All in all, DDT and certain transformation products (see above) should be classed as very persistent.

There is also a lack of reliable data concerning the transformation and degradation processes of the substance in the atmosphere. HCl and CO_2 were established as degradation products in laboratory experiments under simulated upper-atmosphere conditions. Rapid decomposition due to UV rays has been reported by various authors. There are no representative data regarding photomineralisation under natural conditions (WHO, 1979).

Food chain:

Considerable accumulation via food chains. It can be detected in the circulation of warm-blooded animals soon after absorption, it is however subsequently removed by organs containing lipids and is stored in, for example, fatty tissue, the brain and the liver.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	**Sector**	**Country/ organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Water:	Drinkw	A	(L)	1 g/l		DDT and isomers	acc. DVGW, 1988
	Drinkw	CDN	(L)	30 g/l	MAC	DDT and isomers	acc. DVGW, 1988

	Drinkw	D	L	0.1 g/l		single substance	acc. DVGW, 1988
	Drinkw	EC	G	0.1 g/l			acc. DVGW, 1988
	Drinkw	WHO	G	1 g/l		DDT and isomers	acc. DVGW, 1988
	Groundw	USA	G	0.05 mg/l		State of Illinois	acc. WAITE, 1984
	Surface	IAWR	G	0.1 g/l		Drinking water1)	acc. DVGW, 1988
	Surface	IAWR	G	0.5 g/l		Drinking water2)	acc. DVGW, 1988
	Surface	D	G	2 g/l		Drinking water1)	acc. DVGW, 1988
	Surface	D	G	10 g/l		Drinking water2)	acc. DVGW, 1988
	Surface	USA	G	0.05 mg/l		State of Illinois	acc. WAITE, 1984
Air:	Workp	D	L	1 mg/m^3	MAK		DFG, 1987

	Workp	USA	(L)	1 mg/m^3	TWA		acc. RIPPEN, 1989
Foodstuffs:							
Tea, spices		D	L	1 mg/kg			acc. DVGW, 1988
Fruit, vegetables		D	L	0.1 mg/kg			acc. DVGW, 1988
Other veg. foodstuffs		D	L	0.05 mg/kg			acc. DVGW, 1988
Fat in meat		D	L	3 mg/kg			acc. DVGW, 1988
Fat in fish		D	L	2-5 mg/kg			acc. DVGW, 1988
Fat in milk		D	L	1 mg/kg			acc. DVGW, 1988
Eggs		D	L	0.5 mg/kg			acc. DVGW, 1988

Notes:

1) Drinking water treatment using natural methods

2) Drinking water treatment using chemophysical methods

The production and use of DDT has been banned in Germany since 1974. Usage is also banned in Sweden

and the USA.

Comparison/reference values

Water:		
Surface water (1977-79)	USA	0.1 ppb; (max. n=604)
Antarctic		40 ppt
Baltic		0.2 ppt
Groundwater (1977-79)	USA	0.9 ppb; (max. n=1074)
Rainwater	GB	104-229 ppt
Sediment:		
Lakes and river (Berlin)	D	0.01-136 ppb (n=8)
Lake	Libya	0.02 ppb
Mediterranean (1981)		<0.01-19 ppb
Air:		
'Clean air'	D	0.2-0.6 ng/m^3
Persian Gulf		0.05-0.58 ng/m^3 (mean value: 0.08 ng/m^3)

Gulf of Mexico		0.010-0.047 ng/m^3
Humans:		
Mother's milk	D	1.5-1.8 mg/kg fat
Fatty tissue		1.1-5.3 mg/kg (mean values)
Animals:		
Fish (Lake Michigan; 1969-78)	USA	0.8-9.9 mg/kg
Fish (North Sea; 1972)		2-73 g/kg
Pike-perch (Havel, Berlin; 1981)	D	2-105 g/kg
Plants:		
Aquatic plants (Danube)		2 g/kg

Note: 1) **If no other source is quoted, the data cited are from RIPPEN, 1989.**

Assessment/comments

The ban of DDT in several countries in the early Seventies shows its hazardous nature which can primarily be attributed to its considerable persistence in all environmental media (a basic precondition of the worldwide distribution of DDT today). Apart from its acute toxicity, the decisive aspect for any assessment of DDT is its accumulation in organisms, soils, surface water and groundwater and thus its incalculable long-term effect. In view of the fact that substitute products already exist (although still expensive), the application of DDT and also its continued production are ecologically intolerable.

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2,4-Dichlorophenoxy acetic acid

DESIGNATIONS

CAS No.: **94-75-7**
Registry name: **2,4-Dichlorophenoxyacetic acid**
Chemical name: **2,4-Dichlorophenoxyacetic acid**
Synonyms, Trade names: **2,4-D**
Chemical name (German): **2,4-Dichlorphenoxyessigsure**
Chemical name (French): **Acide 2,4-dichlorophnoxyacetique**
Appearance: **colourless, crystalline powder with musty odour**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$C_8H_6Cl_2O_3$
Rel. molecular mass:	221.04 g
Density:	1.563 g/cm^3

Boiling point:	160C at 50 Pa
Melting point:	140.5C
Vapour pressure:	$<10^{-5}$ Pa
Solvolysis/solubility:	in water:0.55 g/l in olive oil 0.5 g/l, in benzene 6 g/l, in acetone 850 g/l

ORIGIN AND USE

Usage:
2,4-D, its salts and esters are used as a herbicide to combat broad-leafed plants in particular. The quantity applied is generally between 0.3 and 4.5 kg/ha. They are frequently used together with other herbicides. Besides MCPA (2-methyl-4-chlorophenoxyacetic acid), 2,4-D is one of the most widespread herbicides used in the cultivation of grain. The butyl esters of 2,4-D and 2,4,5-T were used by the USA in the Vietnam War with the designation "Agent Orange" to defoliate the forests of Southern Vietnam.

Origin/derivation:
Probably no known natural sources. 2,4-D is produced by the chlorination of phenol and subsequent conversion with chloroacetic acid. The technical product may contain between 0.1 and 0.6 % of chlorophenols and traces of polychlorinated dibenzodioxins and dibenzofurans.

Production figures:
Worldwide approx. 100,000 t/a; EC (1980) 15,000-20,000 t; USA (1976) 17,000 t

Emission figures (estimated):

All the 2,4-D produced is released into the environment. In addition, 2,4-D is formed following hydrolysis as metabolite of the acid esters used as herbicides.

Toxicity

Humans:	LD_{50} 80 mg/kg, oral	acc. RIPPEN, 1989
	LDLo 50-500 mg/kg, oral	acc. RIPPEN, 1989
	TCLo 0.01 mg/l, inhalation	acc. RIPPEN, 1989
Mammals:		
Mouse	LD_{50} 360-368 mg/kg, oral	acc. DFV, 1986
Rat	LD50 375-1200 mg/kg, oral	acc. DFV, 1986
	LD50 1,500 mg/kg, dermal	acc. RIPPEN, 1989
Rabbit	LD50 800 mg/kg, oral	acc. RIPPEN, 1989
	LD50 > 1,600 mg/kg, dermal	acc. RIPPEN, 1989
Dog	LD50 100 mg/kg, oral	acc. RIPPEN, 1989
Birds:	LD_{50} 540 mg/kg	acc. RIPPEN, 1989

Aquatic organisms:		
Golden orfe	LC$_{50}$ 250 mg/l	acc. RIPPEN, 1989
Rainbow trout	LC50 1.1 mg/l (96h) (free acid)	acc. PERKOW, 1985
	LC50 100 mg/l (96h) (diethylamine salt)	acc. PERKOW, 1985
Young rainbow trout	LC50 0.022-0.033 mg/l (96 h)	acc. RIPPEN, 1989
Water flea (Daphnia magna)	LC50 > 100 mg/l (48 h)	acc. RIPPEN, 1989
Algae	EC50 50 mg/l (10 d, inhibited growth)	acc. RIPPEN, 1989
Other organisms:		
Earthworm	LC$_{50}$ 10-100 mg/cm2, application (48 h)	acc. RIPPEN, 1989
Actinomycetes	EC50 160-184 mg/kg	acc. RIPPEN, 1989
Soil fungi	EC80 75-128 mg/kg	acc. RIPPEN, 1989

Characteristic effects:

Humans/mammals: **Carcinogenic effect still under dispute; proven teratogenity in rats. 2,4-D can be resorbed through the gastro-intestinal tract and via the skin (2,4-D ester also via the lungs). It acts on the central and peripheral nervous system (spasms and paralysis) as well as the motor activity and intervenes in the intermediary**

carbohydrate metabolism. 2,4-D absorbed orally is rapidly excreted without being transformed and is not stored in the body (DVGW, 1988). Severe eye irritation, slight skin irritation.

Plants: 2,4-D interferes with the metabolism of the plant, e.g. in the nucleic-acid metabolism.

ENVIRONMENTAL BEHAVIOUR

Water:
2,4-D esters are hydrolysed in surface water and groundwater to form the appropriate acids.

Soil:
2,4-D, in particular the alkali and amine salts, is extremely mobile in soil on account of its high water solubility. There is thus the possibility of groundwater being polluted by seepage water.

Degradation, decomposition products, half-life:
UV transformation in water results in products similar to humic acid via chlorophenols and polyphenols. 2,4-dichlorophenol has been detected under anaerobic conditions in sediment and in aquatic organisms whereas 4-chlorophenol has been discovered (as an intermediate product) in digested sludge.

Half-lives: 4-29 d in soil, roughly 5 d in plants (depending on type). The persistence of 2,4-D in surface water or groundwater would appear to be subject to considerable fluctuation depending on the season. The data vary between complete degradation within 36 days and a loss of only 8 % after 78 days in a laboratory experiment. RIPPEN (1989) quotes half-lives in surface water of between < 12 up to 50 days.

Food chain:
Bioaccumulation in algae (chlorella fusca)

ENVIRONMENTAL STANDARDS

Medium/ acceptor	**Sector**	**Country/ organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Water:	Drinkw	A	(L)	50 g/l			acc. DVGW, 1988
	Drinkw	CDN	(L)	100 g/l	MAC		acc. DVGW, 1988
	Drinkw	D	L	100 g/l			acc. DVGW, 1988
	Drinkw	EC	G	100 g/l			acc. DVGW, 1988
	Drinkw	USA	G	100 g/l			acc. RIPPEN, 1988
	Drinkw	WHO	G	100 g/l			acc. RIPPEN, 1988
	Surface	AUS	G	4 g/l		Protection of aquatic	acc. CES, 1985
	Surface	MEX	(L)	0.1 mg/l		organisms (incl. deriv.), estuaries	acc. CES, 1985
	Surface	MEX	(L)	0.01 mg/l		Coastal waters	acc. CES, 1985
Air:	Workp	D	L	10 mg/m^3		Incl. salts and esters	DFG, 1989
	Workp	SU	(L)	1 mg/m^3			acc. DVGW, 1988
	Workp	USA	(L)	10 mg/m^3	TWA		acc. RIPPEN, 1989
Foodstuffs:		WHO	G	0.3 mg/(kgd)	ADI		acc. RIPPEN, 1989
			(G)1)				

		D	(G)[1]	0.1 mg/(kgd)	DTA		acc. DFG, 1986
Citrus fruits		D	L	2 mg/kg			acc. DVGW, 1988
Other foodstuffs		D	L	0.1 mg/kg			acc. DVGW, 1988

Note:

1) "Toxicology" Working Party of German Research Association
Usage banned for example in Czechoslovakia and Sweden; restricted in Great Britain.

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Water:			
Drinking water (1983)	USA	0.04 g/l	acc. RIPPEN, 1989
Rhine (km 865, 1978)		< 0.01 g/l	acc. RIPPEN, 1989
Surface water (1983)	USA	100 g/l, (max.)	acc. RIPPEN, 1989
Drain water under forest	D	2000 g/l (appl. 4.5 kg/ha, ester)	acc. RIPPEN, 1989
Soil/sediment:			
Sewage sludge	USA	0.55-7,300 g/kg (n = 55 of 223)	acc. RIPPEN, 1989

Assessment/comments

The relatively high mobility of 2,4-D implies a hazard to water - including groundwater - in the area of application. This should be considered if the water resource is to serve the drinking water supply. As can be seen from the quoted standards, the assessments of the toxicity of 2,4-D differ widely. The figures for drinking water vary by three orders of magnitude.

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Dichlorvos

DESIGNATIONS

CAS No.: 62-73-7
Registry name: Dichlorvos
Chemical name: 2,2-Dichlorovinyl dimethyl phosphate
Synonyms, Trade names: 2,2-Dichloroethenyl dimethyl phosphate, O,O-dimethyl O-(2,2-dichlorovinyl) phosphate, DDVP, Nuvan, Vapona
Chemical name (German): Dichlorvos
Chemical name (French): Dichlorvos
Appearance: colourless to yellowy liquid

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$C_4H_7Cl_2O_4P$
Rel. molecular mass:	220.98 g
Density:	1.314 g/cm^3
Relative gas density:	7.63
Boiling point:	74C (at 1.32 hPa)
Vapour pressure:	1.6 Pa
Solvolysis/solubility:	in water: 10 g/l miscible with most organic solvents
Conversion factors:	1 ppm = 9.19 mg/m^3 1 mg/m^3 = 0.11 ppm

ORIGIN AND USE

Usage:
Dichlorvos is an insecticide that mainly acts via the gas phase due to its relatively high vapour pressure. It may also be used as a contact insecticide. Dichlorvos is applied in form of aerosols or baits against flies and mosquitos.

Toxicity

Mammals:		
Rat:	LD_{50} 56-108 mg/kg, oral	acc. WHO, 1986
Rat:	LD_{50} 75-210 mg/kg, dermal	acc. WHO, 1986
Rat:	LD_{50} 56-80 mg/kg, oral	acc. WIRTH, 1981
Aquatic organisms:		
Carp:	TLM > 40 mg/l (48 h)	acc. WHO, 1986
Goldfish:	TLM 10-40 mg/l (48 h)	acc. WHO, 1986
Water flea:	TLM 2.8 mg/l (3 h)	acc. WHO, 1986

Characteristic effects:

***Humans/mammals:* Intake takes place either by inhalation or via the gastro-intestinal tract. As is the case with phosphoric acid esters, dichlorvos is only slightly resorbed in the body. Above all, dichlorvos inhibits acetylcholinesterase and thus impairs the central nervous system. The symptoms are comparable to those of paraquat. According to WIRTH (1981), dichlorvos does not represent a genetic risk.**

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/ organ.	Status	Value	Cat.	Remarks	Source

Air:	Workp	D	L	1 mg/m^3	MAK		DFG, 1989
	Workp	SU	(L)	0.2 mg/m^3	PDK	Skin	acc. KETTNER, 1979
	Workp	USA	(L)	1 mg/m^3	TWA		ACGIH, 1986
Foodstuffs:		WHO	G	0.004 mg/kg/d	ADI		acc. WHO, 1986

Assessment/comments

Dichlorvos is one of the organophosphoric insecticides for which the residue problems are similar to those of halogenated insecticides. Direct contact with dichlorvos is to be avoided on account of the fact that it inhibits the respiratory system.

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Dieldrin

DESIGNATIONS

CAS No.: **60-57-1**
Systematic name: **Dieldrin**

Chemical name: **1,2,3,4,10,10-Hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4,5,8-dimethanonaphthalene**

Synonyms, Trade names: **Alvit, Heod, Compound 497, Octalox, ENT 16,225**

Chemical name (German): **Dieldrin; 1,2,3,4,10,10-Hexachlor-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4,5,8-dimethanonaphthalin**

Chemical name (French): **Dieldrine**

Appearance: **white, odourless crystals**

BASIC CHEMICOPHYSICAL DATA

Empirical formula:	$C_{12}H_8Cl_6O$
Rel. molecular mass:	380.91 g
Density:	1.70 g/cm^3
Boiling point:	(decomposition)
Melting point:	176-177C
Vapour pressure:	24 x 10^{-6} Pa at 25C
Solvolysis/solubility:	in water: 0.1 mg/l miscible with petroleum, acetone and aromatic compounds
Conversion factors:	1 ppm = 15.8 mg/m^3

	1 mg/m^3 = 0.06 ppm

ORIGIN AND USE

Usage:
Dieldrin is an insecticide used in particular to protect cotton plants.

Origin/derivation:
Dieldrin is produced by epoxidation of aldrin, a process which also occurs naturally.

Toxicity

Humans:	LD_{50} 64 mg/kg (estimated)	acc. MERCIER, 1981
Mammals:		
Rat:	LD_{50} 46-63 mg/kg, oral	acc. VERSCHUEREN, 1983
	LD_{50} 52-117 mg/kg, dermal	acc. VERSCHUEREN, 1983
Mouse:	LD_{50} 38-77 mg/kg, oral	acc. MERCIER, 1981
Dog:	LD_{50} 56-120 mg/kg, oral	acc. MERCIER, 1981
Rabbit:	LD_{50} 45-50 mg/kg, oral	acc. MERCIER, 1981

Cow:	LD_{50} 25 mg/kg, oral	acc. MERCIER, 1981
Aquatic organisms:		
Cyprinodont	LC_{50} 5 ppb (96 h)	acc. VERSCHUEREN, 1983
Mugilidae	LC_{50} 23 ppb (96 h)	acc. VERSCHUEREN, 1983
American minnow	LC_{50} 16 mg/l (96 h)	acc. VERSCHUEREN, 1983
Blue perch	LC_{50} 8 mg/l (96 h)	acc. VERSCHUEREN, 1983
Rainbow trout	LC_{50} 10 mg/l (96 h)	acc. VERSCHUEREN, 1983
Water flea	LC_{50} 250 mg/l (48 h)	acc. VERSCHUEREN, 1983
Crawfish	LC_{50} 460 mg/l (96 h)	acc. VERSCHUEREN, 1983
Insects:		
Pteronarcys california	LC_{50} 0.5-39 g/l (96 h)	acc. VERSCHUEREN, 1983

Characteristic effects:

Humans/mammals: **Dieldrin can cause poisoning following resorption via the skin, oral intake or inhalation. It acts as a stimulant to the central nervous system and accumulates in fatty tissue causing severe damage to the liver and**

kidneys. Animal experiments have revealed a carcinogenic effect, but as yet no teratogenic action.

Plants: **Dieldrin does not have a toxic effect on plants (MERCIER, 1981).**

ENVIRONMENTAL BEHAVIOUR

Water:
Accumulation takes place in aqueous systems on account of the good solubility of the substance. Dieldrin is assigned in Germany to water hazard class 3 (highly water-polluting) as a result of the high toxicity in aquatic organisms.

Soil:
Dieldrin accumulates in soils depending on their texture and water content.

Half-life:
Roughly 95 % of an applied quantity of between 3.1 and 5.6 kg/ha disappears from the soil after 12.8 years on average. Only some 9 % evaporates out of loamy or sandy soils in 60 days. Between 75 and 100 % of dieldrin is degraded or decomposed in 3 - 25 years [VERSCHUEREN, 1983].

Environmental behaviour

Degradation, decomposition products:
Dieldrin is metabolised in the body to form 1,2-dihydroxy-dieldrin and 4,5-aldrin-trans-dihydrodial. UV light causes decomposition to form CO_2.

Food chain:
Dieldrin accumulates in fatty tissue and in the mammary glands of human (WIRTH, 1981).

ENVIRONMENTAL STANDARDS

Medium/ acceptor	**Sector**	**Country/ organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Water:	Drinkw	D	L	0.1 g/l		single substance	acc. RIPPEN, 1992
	Drinkw	D	L	0.5 g/l		sum of pesticides	acc. RIPPEN, 1992
	Drinkw	EC	L	0.1 g/l		single substance	acc. RIPPEN, 1992
	Drinkw	EC	L	0.5 g/l		sum of pesticides	acc. RIPPEN, 1992
	Drinkw	USA	G	1 g/l		In state of Illinois	acc. WAITE, 1984
Soil:		NL	G	0.5 g/kg		Single substance, Reference	acc. TERRA TECH 6/94
		NL	L	2.5 g/kg		Aldrin+Dieldrin+Endrin Intervention	acc. TERRA TECH 6/94
Air:	Workp	D	L	0.25 mg/m^3	MAK	Skin	DFG, 1989
	Workp	SU	(L)	0.01 mg/m^3		Skin	acc. KETTNER, 1979
	Workp	USA	(L)	0.25 mg/m^3	TWA	Skin	ACGIH, 1986

Note:

There has been a total ban on the use of dieldrin in the Federal Republic of Germany since 1988 (Order Governing Use of Pesticides).

Comparison/reference values

Medium/origin	**Country**	**Value**	**Source**
Surface/groundwater			
Irish Sea (suspension)	IRL	0.2-140 ng/g	acc. VERSCHUEREN, 1983
Hawaii (sediment)	USA	2-39.5 ppb	acc. VERSCHUEREN, 1983
Los Angeles (harbour)	USA	0.6 x 4.5 ppb	acc. VERSCHUEREN, 1983
Western Baltic (surface)		0.17×10^{-9} g/l	acc. VERSCHUEREN, 1983
North Sea, SE England/Holland		0.4-17 ppb (1974-76)	acc. VERSCHUEREN, 1983

Assessment/comments

Dieldrin is a highly toxic substance towards aquatic organisms and is highly persistent in the environment. In addition, it accumulates in fatty tissue and can also cause severe poisoning in humans. Therefore, its use should be restricted as far as possible.

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Dinitro-o-cresol

DESIGNATIONS

CAS No.: 534-52-1
Systematic name: Dinitro-o-cresol
Chemical name: 2-Methyl-4,6-dinitrophenol
Synonyms, Trade names: 4,6-Dinitro-o-cresol, DNOC, DNC, Detal, Etzel, fruit-tree carboline
Chemical name (German): Dinitro-o-kresol, 2-Methyl-4,6-dinitrophenol
Chemical name (French): Dinitro-o-crsol
Appearance: yellow powder or crystals with bitter taste

BASIC CHEMICOPHYSICAL DATA

Empirical formula:	$C_7H_6N_2O_5$
Rel. molecular mass:	198.14 g
Boiling point:	(decomposition)
Melting point:	86-86.9C
Vapour pressure:	6.5×10^{-3} Pa at 25C
Flash point:	limited combustibility
Solvolysis/solubility:	in water: slightly soluble 125 ppm (at 25C); in acetone: 100.6 g/100 g;

	in ethanol: 4.3 g/100 g; in benzene: 37.5 g/100 g; in chloroform: 37.2 g/100 g; soluble in diethylether, methanol, petroleum ether, carbon tetrachloride

ORIGIN AND USE

Usage:
DNOC is a selective herbicide used in the cultivation of grain, hops, vines and fruit (insecticide, acaricide, with fungicidal secondary effects).

Origin/derivation:
DNOC is only produced synthetically. Commercially available preparations contain formulations of the alkali, ammonia, or amine salts of DNOC which are normally highly soluble in water.

Toxicity

Humans:	LD 0.35-3.0 g	acc. DFG, 1986
Mammals:		
Rat:	LD_{50} 25-85 mg/kg, oral	acc. DFG, 1986
	LD_{50} 28.5 mg/kg, intraperitoneal	acc. DFG, 1986
	LD_{50} 23.1-26.1 mg/kg, subcutaneous	acc. DFG, 1986

Mouse:	LD_{50}20.0 mg/kg, oral	acc. DFG, 1986
	LD_{50} 21.5-27.3 mg/kg, subcutaneous	acc. DFG, 1986
	LD_{50} 1,000 mg/kg, cutaneous	acc. DFG, 1986
	LD_{50} 24-26 mg/kg, intraperitoneal	acc. DFG, 1986
Guinea pig:	LD_{100} 500 mg/kg, cutaneous	acc. DFG, 1986
Dog:	LD_{50} 15 mg/kg, intravenous	acc. DFG, 1986
	LD_{50} 10-23.5 mg/kg, intraperitoneal	acc. DFG, 1986
Aquatic organisms:		
American minnow:	1.5-2 mg/l lethal (6 h)	acc. DVGW, 1988
Stickleback:	3 mg/l lethal	acc. DVGW, 1988
Water flea:	EC_{50} 0.013 mg/l	acc. DVGW, 1988
Blue algae:	$EC1_{0}$ 0.15 mg/l	acc. DVGW, 1988
Green algae:	EC_{10} 13 mg/l	acc. DVGW, 1988

Characteristic effects:

Humans/mammals: **DNOC is a powerful, cumulative-action poison which can even prove fatal. Absorption is primarily by way of the lungs, but also via the gastro-intestinal tract and through the skin. Assimilated DNOC is only slowly excreted.**

The first symptoms of poisoning are an increase in body temperature (high ambient temperatures enhance the severity), sweating, rapid breathing and accelerated pulse, extreme thirst, painful colics, diarrhoea and nausea. Typical symptoms of the effects on the central nervous system are euphoria to be followed by dizziness, possible collapse, anxiety, unrest, disorientation, loss of consciousness and terminal spasms.

Chronic poisoning takes the form of headaches, fatigue and noticeable weight loss. There is also damage to the heart, liver and kidneys. Liver damage is encountered primarily in the case of oral intake.

Plants: **The effect on plants is based on the separation of cell respiration and oxidative phosphorylation.**

ENVIRONMENTAL BEHAVIOUR

Water:
Despite the fact that the solubility in water is extremely low, surface water may be polluted by the elution of soil treated with DNOC. It is more harmful to plankton and microorganisms than to fish. Threshold concentration for water fleas is 3 mg/l, for mosquitoes 500 mg/l.

The toxicity of DNOC solutions is highly dependent on the pH. Acid solutions are more toxic than alkaline solutions (DFG, 1986).

Soil:
DNOC is highly mobile in soil and is only subject to slow microbial degradation. Most soil organisms are not

adversely affected by DNOC; CO_2 production is not impaired; the effect on microarthropodes (e.g. mites) and earth worms is however lethal.

Degradation, decomposition products:
Numerous metabolites have been found in the organism, some of which have a detoxifying effect, whereas others are even more toxic than DNOC itself (e.g. 6-amino-4-nitro-o-cresol or 4,6-diamino-o-cresol). There are no data available metabolites in plants and soil.

DNOC can be traced in soil for a period of up to 14 weeks (acc. DFG, 1986).

Food chain:
There is evidence of residues in parts of plants.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	Sector	Country/ organ.	Status	Value	Cat.	Remarks	Source
Water:	Surface	EC	(L)	0.001 mg/l		All pesticides1)	acc. DVGW, 1988
	Surface	EC	(L)	0.0025 mg/l		All pesticides2)	acc. DVGW, 1988
	Surface	EC	(L)	0.005 mg/l		All pesticides3)	acc. DVGW, 1988
	Drinkw	EC	(L)	0.1 g/l			acc. DVGW, 1988
	Drinkw	FRG	L	0.1 g/l			acc. DVGW, 1988
Air:	Workp	FRG	L	0.2 mg/m^3	MAK		DFG, 1989

	Workp	SU	(L)	0.05 mg/m^3			acc. KETTNER, 1979
	Workp	USA	(L)	0.2 mg/m^3	TWA		ACGIH, 1986
Foodstuffs:		FRG	(G)	0.01 ng/kg.d		Provisional DTA	acc. DFG, 1986

Notes:

DNOC cannot be used as an insecticide in the Federal Republic of Germany in water catchment areas. Its use as a herbicide is banned in water protection zones I and II. Use in the immediate vicinity of stretches of water is forbidden.

1) Mandatory value for simple physical treatment and sterilisation

2) Mandatory value for normal physical and chemical treatment and sterilisation

3) Mandatory value for physical and refined chemical treatment, oxidation, adsorption and sterilisation

Assessment/comments

DNOC has an extremely high acute toxicity and represents a particular hazard. The substance is readily absorbed by the lungs and through the skin. Thus protective measures are required during application. The use of DNOC near large areas of water should particularly be avoided.

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Dioxins

DESIGNATIONS

> ***Note:*** **Strictly speaking, the designation "dioxins" refers to the group of the polychlorinated dibenzo-p-dioxins (PCDD). Chemically and toxicologically, this group is closely related to the group of the polychlorinated dibenzo-p-furans (PCDF). Thus, in a less strict sense, the designation "dioxins" may cover both groups (PCDD/F). The PCDD/F substance group consists of 210 congeners which differ in the number and position of the chlorine atoms. The basic data refer to the most toxic and best described congener 2,3,7,8-tetrachlorodibenzo-p-dioxin which is often named "dioxin" in general usage.**
>
> ***CAS No.:*** **1746-01-6**
> ***Systematic name:*** **2,3,7,8-Tetrachlorodibenzo-p-dioxin**
> ***Chemical name:*** **2,3,7,8-Tetrachlorodibenzo-p-dioxin**
> ***Synonyms, Trade names:*** **Dioxin, 2,3,7,8-TCDD, TCDD, 2,3,7,8-Tetrachlorodibenzo-1,4-dioxin**
> ***Chemical name (German):*** **2,3,7,8-Tetrachlordibenzo-p-dioxin, Dioxin**
> ***Chemical name (French):*** **Dioxine**
> ***Appearance:*** **colourless, crystalline slivers**

BASIC CHEMICOPHYSICAL DATA

Empirical formula:	$C_{12}H_4Cl_4O_2$
Rel. molecular mass:	321.96 g
Density:	1.83 g/cm^3
Boiling point:	approx. 900C

Boiling point:	approx. 900C
Melting point:	approx. 300C
Vapour pressure:	150×10^{-9} Pa
Solvolysis/solubility:	13×10^{-9} g/l
Conversion factors:	1 ppm = 13.38 mg/m^3 1 mg/m^3 = 0.08 ppm

Origin and use

Usage:
2,3,7,8-TCDD (like all PCDD/F) is an unwanted by-product and is produced only as an analytical standard.

Origin/derivation:
There are no known geogenic sources at present. Dioxins are created in thermal processes with lack of oxygen and temperatures of 300-800C from organically and inorganically bonded chlorine (e.g. waste incineration or pyrolysis). Dioxins are likewise encountered during the industrial production of chlorinated compounds. Therefore, these substances may contain impurities of dioxins. Special attention has to be given to the production and processing of chlorinated aromatic substances such as chlorophenols and chlorobenzenes. Dioxins require temperatures above 1000C to decompose completely.

Toxicity

Mammals:		

Rat	LD_{50} 22-100 g/kg, oral	acc. RIPPEN, 1989
Mouse	LD_{50} 70 g/kg, oral	acc. RIPPEN, 1989
Monkey	LD_{50} 114-280 g/kg, oral	acc. RIPPEN, 1989
Mouse	LDLo 80 g/kg, dermal	acc. RIPPEN, 1989
Rabbit	LDLo 10 g/kg, oral	acc. RIPPEN, 1989
Rabbit	LD_{50} 100-115 g/kg, oral	acc. RIPPEN, 1989
Rabbit	LDLo 275 g/kg, dermal	acc. RIPPEN, 1989
Hamster	LD_{50} 1,160-5,000 mg/kg, oral	acc. RIPPEN, 1989
Guinea pig	LD_{50} 0.5-2.0 g/kg, oral	acc. RIPPEN, 1989
Dog	LD_{50} 30-300 g/kg, oral	acc. RIPPEN, 1989
Birds:		
Chicken	LD_{50} 25-50 g/kg, oral	acc. RIPPEN, 1989

***Note:* The majority of the toxicological data available refers to 2,3,7,8-TCDD. In view of the fact that the toxic profiles of the PCDD/F are very similar, their potency is described by introducing toxicity equivalence factors (TEF)**

relative to 2,3,7,8-TCDD.

Characteristic effects:

Humans/mammals: **2,3,7,8-TCDD is the compound with the most severe toxic effect of all PCDD/F. Acute poisoning mainly affects the skin and the liver as well as the peripheral and central nervous system. Additionally, psychic disorientation and effects on the immune system (in animal experiments) have been observed. Chloracne is a typical and long-lasting symptom of acute poisoning by TCDD. The affected skin may also show overpigmentation. Liver damage results in an increased level of transaminase and fat content in the blood. Intestinal disturbances with diarrhoea have been observed as well as damage to the coronary vessels and the urinary tracts. The effects on the nervous system manifest themselves in excitability, nervousness, headaches, temporary insomnia and decrease of the visual and aural capacity. As yet, carcinogenic effects have only been established in animal experiments; teratogenic effects have not yet been proven.**

ENVIRONMENTAL BEHAVIOUR

Water:
Dioxins are readily adsorbed on suspended matter because they are practically insoluble in water. The bio-availability is low, however the toxic effect on aquatic organisms is considerable.

Air:
Dioxins are found in the atmosphere adsorbed on dust particles (fly ash).

Soil:
Mobility is extremely low because of the low water solubility and high adsorption capability. Dioxins thus accumulate in soil.

Half-life:

The half-life of dioxins in soil is more than 10 years (ROTARD, 1987). The half-life in the human body is up to 6 years (BECK et al., 1987).

Degradation, decomposition products:

There is only slight degradation of dioxin by mircoorganisms. Photodegradation may occur.

Food chain:

Accumulation of dioxins in the food chain is a consequence of their solubility in fats. Bioaccumulation is high in fish as well as in fat and in the liver of terrestrial organisms. However, accumulation in plants is moderate.

ENVIRONMENTAL STANDARDS

Medium/ acceptor	**Sector**	**Country/ organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Soil:		D	G	5 ng/kg		unrestricted use	acc. RIPPEN, 1991
		D	G	40 ng/kg		unrestricted agricultural use	acc. RIPPEN, 1991
		D	G	100 ng/kg		exchange of soil on children's playground	acc. RIPPEN, 1991
		D	G	1000 ng/kg		exchange of soil in settlement areas	acc. RIPPEN, 1991
		D	G	10,000 ng/kg		exchange of soil irrespective of site	acc. RIPPEN, 1991
Air:	Workp	D	L	1)	MAK		acc.RIPPEN, 1991

1) not stipulated because of proven carcinogenic effects in animal experiments (Group III A2) 1990
Limit value

Assessment/comments

Due to their extreme toxicity and the suspected carcinogenic and teratogenic effects, the emission of dioxins should be avoided as far as possible. As waste incineration and the production of chlorinated aromatic compounds are the primary sources of dioxin emissions, measures should be taken especially in these fields. Further investigations on the formation of dioxins, on technical measures to reduce dioxin emissions and on their toxicological effects are in process.

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Endosulfan

DESIGNATIONS

CAS No.: 115-29-7
Registry names: Endosulfan
Chemical name: 6,7,8,9,10,10-Hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin-3-oxide

Synonyms, Trade names: **Beosit, Thiodan, Thiofor, Malix**
Chemical name (German): **Endosulfan, 6,7,8,9,10,10-Hexachlor-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin-3-oxid**
Chemical name (French): **Endosulfane**
Appearance: **yellow to brownish-yellow crystalline solid; odour like SO_2**
Note: **Technical endosulfan is a mixture of endosulfan isomers (80% α -isomer / 20% β -isomer)**

BASIC CHEMICAL AND PHYSICAL DATA

Empirical formula:	$C_9H_6Cl_6O_3S$
Rel. molecular mass:	406.95 g
Density:	1.745 g/cm^3
Relative gas density:	14.1
Boiling point:	106C at 0.9 hPa (partial decomposition)
Melting point:	technical 70-100C α -isomer 108-109C β -isomer 206-208C
Vapour pressure:	$< 1 \times 10^{-3}$ Pa
Solvolysis/solubility:	in water 1.4 mg/l; in benzene 33 g/l;

	in xylene 45 g/l; in chloroform 50 g/l; in tetrachloromethane 29 g/l; in methanol 11 g/l.

ORIGIN AND USE

Usage:
Endosulfan is an insecticide.

Origin/derivation:
Endosulfan is produced from hexachlorocyclopentadiene, by Diels-Alder reaction with butenediol, followed by cyclisation with thionyl chloride.

Production figures:
KOCH (1989) estimates the annual production in the Federal Republic of Germany as approx. 2,500 t.

Toxicity

Mammals:		
Rat:	LD_{50} 30-110 g/kg, oral	acc. PERKOW, 1992
	LC_{50} 10-30 g/m^3, inhalation (4 h)	acc. PERKOW, 1992
	LD_{50} 730 g/kg, dermal	acc. PERKOW, 1992

Mouse:	LD_{50} 6.9-13.5 mg/kg, oral	acc. KOCH, 1989
Aquatic organisms:		
Fish	LC_{50} 1.2-1.5 mg/l (96h)	acc. PERKOW, 1992

Characteristic effects:

***Humans/mammals:* Resorption following oral intake is a slow process which is however promoted by fats. Rapid metabolic degradation takes place in the organism with the formation of endosulfandiol. Non-metabolised endosulfan is excreted with urine as are the degradation products. According to KOCH (1989), bioaccumulation is not to be expected. Liver and kidney damage have only been found to date in animal experiments. There are no data available on mutagenic or carcinogenic potentials.**

ENVIRONMENTAL BEHAVIOUR

Endosulfan is stable under normal conditions. Hydrolysis takes place in an acetic or alkaline aqueous medium with the formation of less toxic diol and sulphur dioxide. The chemical structure of endosulfan is such that it is more reactive than DDT or lindane.

The environmental behaviour is determined by the poor solubility in water and the volatility of the substance. It is not accumulated in biotic and abiotic media because of its reactivity. Rapid degradation takes place.

Attention should be paid to the high toxicity in aquatic organisms (water hazard class 3).

ENVIRONMENTAL STANDARDS

Medium/ acceptor	**Sector**	**Country/ organ.**	**Status**	**Value**	**Cat.**	**Remarks**	**Source**
Water:	Drinkw	D	L	0.1 mg/l			acc. KOCH, 1989
	Drinkw	D	L	0.5 mg/l		As sum total for pesticides	acc. KOCH, 1989
	Drinkw	DDR	L	5 mg/l			acc. KOCH, 1989
		SU		1-3 ng/l		Fish breeding waters	acc. KOCH, 1989
Air:	Workp	SU	(L)	0.1 mg/m^3	PDK		acc. SORBE, 1989
	Workp	USA	(L)	0.1 mg/m^3	TWA		ACGIH, 1989

Assessment/comments

The use of endosulfan in the vicinity of surface waters should be the subject of extremely critical assessment on account of its high toxicity in aquatic organisms.

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