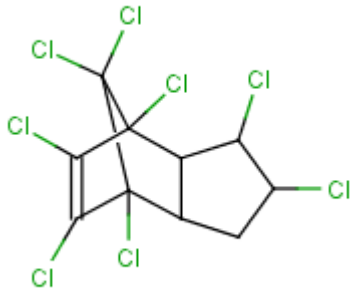
CHLORDANE

$C_{10}H_6Cl_8$ CAS Nr. 57-74-9

Chemical structure



Chemical formule[1]

Relative molecular mass: 409.8

The term 'chlordane' commonly refers to a complex mixture of chlordane isomers, other chlorinated hydrocarbons and by-products. Technical chlordane is a mixture, which consists of at least 147 compounds and the composition varies with the manufacturing process. It contains 43 –75 % *cis*- and *trans*-chlordane and lesser amounts of heptachlor, *cis*- and *trans*-nonachlor and chlordenes. From 1970 onwards, a more refined formulation containing >95 % of *cis*- and *trans*chlordane has also been produced with a ratio of *cis*- to *trans*-chlordane of about 3:1 (EFSA, 2007).

Properties

Chlordane has very low volatility and is essentially insoluble in water (table 1). The biodegradation in soil is very slow. The half-life of chlordane in air, soil and water has been estimated to be in the range of months to many years. Congeners with higher numbers of chlorine atoms (≥8) have a higher persistency. These chemicals can be expected to accumulate in sediment long after application has ceased (IARC, 2001). Because of their lipophilic properties and persistence in the environment, chlordane and related compounds are bioaccumulated and biomagnified along the food chain (EFSA, 2007).

Table 1: Properties of technical chlordane (EFSA, 2007)

Solubility in water	56 µg/L at 25°C.
Melting point	<25°C
Henry's law constant	4.9 Pa m3/mol at 25°C
log Koc	4.58-5.57
log Kow	6.00
Vapour pressure	1.3 × 10-3 Pa at 30°

Chlordane is among the 12 persistent organic pollutants being considered for international action to reduce or eliminate their releases under the Stockholm convention signed in 2001.

Contamination source

Agricultural and non agricultural use

Chlordane is a persistent chlorinated non-systemic (not taken up in the plant), contact and ingested insecticide, which was extensively used from 1947 onwards. It has been shown to non-competitively block GABA receptors in cockroach, *Periplaneta americana*, and other insects. It is non-phytotoxic at insecticidal concentrations. According to the WHO (1984) chlordane has not been produced in Europe or Japan. Chlordane was mainly used as an agricultural insecticide but also for non-agricultural purposes. In Europe it was mainly used for the protection of potato crops vegetables, small grains and sugar beets although no information on the amounts used has been identified

[1] http://chem.sis.nlm.nih.gov/chemidplus/ProxyServlet?objectHandle=Search&actionHandle=getAll3DMViewFiles&nextPage=jsp%2Fcommon%2FChemFull.jsp%3FcalledFrom%3Dlite&chemid=0000057749&formatType=_3D

(EFSA, 2007).
The use of chlordane as a pesticide has been banned in the EU since 1981 by Council Directive 79/117/EEC of 21 December 1978 which prohibited the placing on the market and use of plant protection products containing certain substances.
Chlordane use for non-agricultural purposes was primarily for the protection of structures, but also on lawns and turf, ornamental trees and drainage ditches, mainly in the USA (EFSA, 2007).
Chlordane has also been used as an ingredient in veterinary preparations for the protection of livestock from different pests (EFSA, 2007). Since the mid-1970s, the use of chlordane has been increasingly restricted in many countries.

It has recently been reported that chlordane is extensively used against termites with an estimated amount of over 200 tons/year in China (Xu *et al.,* 2004). Furthermore, particularly outside Europe it appears that there are still considerable amounts in wooden building materials as well as in stockpiles that all eventually could contaminate the environment (EFSA, 2007).

Bioaccumulation and bioconcentration
Biomagnification of chlordane is a complex issue as the compositional pattern (including metabolites) varies with trophic level and food chain. This is due to differences in uptake and metabolism of the different constituents of the technical mixture. Accumulation and transformations of chlordanes have been shown to be enantiomer-selective. As a general rule for environmental samples, the deviation of the enantiomeric fraction from 0.5 (racemate) show similar patterns, i.e. air < water < soil < biota (source: EFSA,2007). In biota, the order is: lower trophic level < higher trophic level and liver or kidney tissue < brain tissue. Recently, information on enantiomeric fractions in reference materials, such as fish homogenates, has been made available. Generally, the composition of chlordane compounds changes in comparison with the technical mixtures when moving up the food chain. *Trans*-nonachlor and in particular oxychlordane concentrations increase at higher trophic levels. In fish, chlordane shows a high potential for bioaccumulation with bioconcentration factors (BCFs) between 3000 and 18,500 (EFSA, 2007).

Analytical method

Determination of chlordane residues is difficult because of the complex nature of the components and the fact that each component degrades independently.
Extraction from crops, other plant products, dairy products, plants and oils has been achieved with an 80–100% efficiency with the use of acetonitrile for extraction, petroleum ether for partitioning and clean-up on a Florisil column. Gel-permeation chromatography can also be used for clean-up, particularly of human adipose tissue. The method of choice for the qualitative and quantitative estimation of chlordane isomers and heptachlor is gas chromatography with electron-capture detection. Gas chromatographic analyses can be confirmed by gas chromatography–mass spectrometry, a method that can also provide good determination of some of the components, such as heptachlor epoxide. Analysis for total organically bound chlorine remains the preferred method for determination of technical-grade chlordane and heptachlor and of the active ingredient in formulations (EFSA, 2007).
A number of other well-proven, validated multi-residue methods are available for the quantitative determination of chlordane in various environmental matrices, including food, feed and other biological specimens (Muir and Sverko, 2006).
Due to the high electro negativity caused by the seven or more chlorine atoms of chlordane and related compounds, high-resolution gas chromatography with electron capture detection (HRGC-ECD) is the analytical method most commonly used. An efficient separation of chlordane compounds from other interfering substances, such as other organochlorine pesticides and PCBs is especially important when using HRGC-ECD (EFSA, 2007).

Toxicity

Main risks and target organs
Chlordane is likely to causes cancer and may cause liver cancer, can cause behavioral disorders in children if they were exposed before birth or while nursing, harms the endocrine system, nervous system, digestive system, and liver (EPA, 2011).
In mammals, the main target organs are the nervous system and the liver. Chlordane causes liver tumours in mice, probably via nongenotoxic mechanisms. The liver and the kidney are the other organs significantly affected by chlordane (WHO, 2000).
Oxychlordane (a major metabolite of *cis*- and *trans*-chlordane) and nonachlor are more toxic than

cis- and *trans*-chlordane.

Toxicokinetics and tissue residues
Chlordane is readily absorbed after oral exposure. Although no specific studies were carried out to determine the intestinal absorption of chlordane in humans, the presence of chlordane residues in the adipose tissue, brain, liver and plasma of individuals after accidental ingestion indicates that the compound is absorbed from the gastrointestinal tract in humans (EFSA, 2007).

Chlordane is primarily metabolized to oxychlordane and to a minor extent may also be dehydrochlorinated to heptachlor. Oxychlordane is the most relevant animal metabolite, being more persistent and toxic than the parent compound (WHO, 1984). Heptachlor, which is also a component of technical-grade chlordane, is biotransformed to its epoxide. Subsequent dechlorination reactions lead to hydroxylated compounds, which are excreted primarily as glucuronides. Minor metabolites include heptachlor and heptachlor epoxide (IARC, 2001). These substances are very slowly excreted from the body in the stools, urine, and in nursing females also in the milk (Bondy et al., 2003; Campoy et al., 2001; Hirai & Tomokuni, 1991).

Oxychlordane and *trans*-nonachlor are generally the major residues of chlordane compounds in animal tissues and animal products (EFSA, 2007).

Acute toxicity
Chlordane has moderate acute toxicity with oral LD_{50} values for mice and rats of 335 – 430 mg/kg bw, and higher values (1720 mg/kg bw) in the hamster. *Cis*-chlordane is more toxic than *trans*-chlordane. Oxychlordane has high acute toxicity with a LD_{50} in the rat of 20 mg/kg bw Other constituents of the technical mixture and metabolites tested (chlordene, 3-chlorochlordene, 1-hydroxychlordene, chlordene epoxide, 1-hydroxy,2,3 epoxychlordene, 2-chlorochlordene) have lower acute toxicity than chlordane (WHO, 1984).
Chlordane stimulates the central nervous system. The first symptom of intoxication is a sharper sensitivity to external stimuli. Later on restlessness, tremor, even epileptic spasms are observed. Symptoms of acute poisoning appear 45 min after consumption, although sometimes they appear as late as several hours afterwards. Chlordane damages the parenchyma organs. Cases of fat degeneration of the liver, even necrosis, kidney damage, haemorrhage in the lungs, liver, kidneys, heart muscle and intestine mucous membrane have been reported. Chlordane affects the metabolism and toxicity of a number of simultaneously applied substances by inducing the activity of hepatic microsome enzymes (WHO, 1984).
Symptoms such as disorientation, ataxia, tremors, convulsions and respiratory failure and cyanosis have been reported. In humans neurological symptoms, including headache, dizziness, vision problems, incoordination, irritability, excitability, weakness, muscle twitching and convulsions have been described in case reports on accidental acute exposures (WHO, 1984).

Repeated toxicity
Upon low-level longer-term exposure, the liver is the target for toxicity of chlordane with induction of hepatic microsomal enzyme activity as one of the most sensitive biochemical parameters. At higher levels, liver hypertrophy with histopathological (from liver cell hyperplasia to cell necrosis, fatty infiltration and particularly in mice, nodule formation) and functional changes may occur (WHO, 1984).

In experimental animals, the toxic effects of chlordane on the liver include lipid peroxidation and cell proliferation secondary to cytotoxicity. In the thyroid, chlordane has been shown to decrease thyroxine concentrations in rats. Chlordane induces hepatic and gonadal microsomal oxidative enzymes and also steroid hormone metabolism (IARC, 2001).

The NOAELs were 0.075 mg/kg bw/d in dogs (2-year study), 0.05 mg/kg bw/day in rats (2-year study) and 0.12 mg/kg bw/day in mice 2-year study).

Carcinogenicity
Case reports of leukaemia and other blood dyscrasias have been associated with exposure to chlordane/heptachlor, primarily in domestic situations (WHO, 2000).
Mortality from lung cancer was slightly elevated in two cohort studies of pesticide applicators; and one of chlordane/heptachlor manufacturers.

Small excess risks for other cancers, including leukaemia, non-Hodgkin's lymphoma and soft tissue sarcoma and cancers of the brain, skin, bladder and stomach were observed, with little consistency among studies (WHO, 2000).

Chlordane, technical-grade chlordane, heptachlor, technical-grade heptachlor, heptachlor epoxide and a mixture of heptachlor and heptachlor epoxide have been tested for carcinogenicity by oral administration in several strains of mice and rats. In the studies in mice, increased incidences of hepatocellular neoplasms (including carcinomas) were seen in both males and females. Increased incidences of thyroid follicular-cell adenomas and carcinomas were seen in one study each with chlordane and technical-grade heptachlor in rats. In a third study in rats, technical-grade chlordane marginally increased the incidence of liver adenomas in male rats. In initiation–promotion studies in mice, administration of chlordane or heptachlor after *N*itrosodiethylamine resulted in increased incidences of hepatocellular tumours (IARC, 2001).

Multiple studies have shown that high, daily dietary exposure to chlordane is associated with the development of hepatocellular carcinomas in mice (ATSDR, 1997).

Chlordane is not mutagenic in vivo and not or only weakly mutagenic in a few tests in vitro. It is a promoter of liver tumours in vivo and exhibit biochemical properties shared by many promoters of liver tumours (EFSA 2007).

Chlordane has been evaluated by the International Agency for Research on Cancer (IARC, 2001). It was concluded that there is *inadequate evidence* in humans for the carcinogenicity of chlordane. There is *sufficient evidence* in experimental animals for the carcinogenicity of chlordane. Chlordane is *possibly carcinogenic to humans (Group 2B)*.

In the European Union, chlordane was classified as Carc. Cat.3; R40 according to Dir 67/548/EEC and as Carc.2 H351 according to CLP Regulation (EC) No. 1272/2008.

The U.S. EPA categorizes chlordane as a probable human carcinogen (group 2B). This means that chlordane has been shown to cause cancer in laboratory animals, but there is inadequate or no evidence that it may cause cancer in humans.

Genotoxicity
No data were available on the genetic and related effects of chlordane or heptachlor in humans. Both compounds inhibited gap-junctional intercellular communication and induced gene mutations in rodent cells. Likewise, both compounds induced unscheduled DNA synthesis in human fibroblasts but not in rodent hepatocytes. Chlordane induced DNA damage in liver cells of rats treated *in vivo*, but heptachlor did not induce mutations in hepatocytes of *lacI* transgenic mice treated *in vivo*. Neither chlordane nor heptachlor caused dominant lethal mutation in mice. Neither chlordane nor heptachlor was mutagenic to bacteria, and only chlordane damaged bacterial or plasmid DNA (IARC, 2001).
Chlordane is not mutagenic *in vivo* and not or only weakly mutagenic in a few tests *in vitro* (EFSA, 2007).

Reproductive toxicity
There were no indications of teratogenicity.
Chlordane and heptachlor are toxic to reproduction and development in mice, rats and mink. Pre- and postnatal exposures to chlordane affected the development of the immune system in rodents.

Neurotoxicity
Accidental or intentional exposure to chlordane has resulted in signs of neurotoxicity and, in some cases, death.

Immunotoxicity
Impaired cell mediated immunity after prenatal exposure to chlordane has been observed in female BALB/c mice (IARC, 2001).
In a 28-day oral study in rats, exposure levels of 2.5 and 25 mg/kg bw/d caused significant effects on a number of immunologic endpoints, i.a. immunoglobulin levels, increased number of lymphocytes and reduced resistance to *Listeria monocytogenes*. The NOAEL for immune effects was 0.25 mg/kg

bw/day.

Establishment of Health Based Reference Values
JMPR re-evaluated its earlier assessments on chlordane in 1986 (FAO/WHO, 1987) and established an ADI of 0.5 µg/kg bw by applying an uncertainty factor of 100 to a NOAEL of 50 µg/kg bw/day for liver toxicity in a long-term study in rats. In 1994, JMPR converted the ADI into a provisional tolerable daily intake (PTDI) with the same value (FAO/WHO,1995).

Occurrence in food

Food is considered to be the major source of exposure of the general population to chlordane (WHO, 1984).
During the period when Chlordane and heptachlor were being used as pesticides, a number of studies were carried out to determine the concentrations of chlordane, heptachlor and related compounds in foods. Most foods were found to contain low or undetectable concentrations of these chemicals, with the exception of meat, poultry and dairy products, in which significant concentrations were found.
In Belgium a total of 870 single and compound feed samples were collected and analyzed between 2000 and 2004. The analytical methods covered *cis*- and *trans*-chlordane, each at a given limit of quantification of 2 µg/kg. One sample of (SM: simple-mat.Prem./S) contained chlordane at 9 µg/kg. All other samples were negative.
Chlordane and other pesticides were analyzed in free range eggs in the autumn 2006 (40 egg samples) and in the spring 2007 (58 egg samples; same location than in spring+18 other locations) in Belgium. For the sum of chlordane (α-chlordane, γ-chlordane, oxychlordane, transnanochlor), two samples were above the Belgian norm of 50 ng/g fat, but these compounds were not detected in 64% of the samples. α- and γ-chlordane were detected in only one sample of 59, oxychlordane and transnanochlor being responsible for the more elevated values measured (Windal et al., 2009).

Concentration in organochlorinated pesticides measured is fish in Belgium in 2005-2006 are generally under the reporting limit (Vromman et al., 2008).

Chlordane has been found in meat, eggs and milk. Some chlordane metabolites have been found in human milk. Content of chlordane in food samples taken by Fromberg et al. (2011) from 1998 to 2003 in Denmark are presented in table 2.

Table 2: Content of chlordane in food samples taken by Fromberg et al. (2011) from 1998 to 2003 in Denmark

Foodstuff	Mean sum chlordane (µg/kg fish and egg and µg/kg fat for other foods)
Chicken fat	1.1
Beef fat	1.1
Pork fat	1.2
Cheese, Danish	1.7
Cheese, foreign	1.2
Eel, farmed, raw	9.3
Greenland halibut, raw	13.6
Herring, raw	2.1
Herring, pickled	6.7
Herring, smoked	2.7
Lumpsucker, raw	9.4
Mackerel, raw	1.6
Mackerel, smoked	2.7
Mackerel, tinned in tomato	1.2
Rainbow trout, farmed, raw	2
Salmon, raw	4.8
Swordfish, raw	1
Trout, marine farmed, raw	3.4
Fish oil	24.8
Cod liver oil	93

The most significant source of exposure of infants to chlordane, heptachlor and their metabolites appears to be breast milk, in which the concentrations can be much higher than those in dairy milk. The concentrations of *cis*- and *trans*-chlordane in breast milk were higher in Inuit mothers from northern Quebec (3.7 ng/g of fat) than in southern Canadian residents (0.37 ng/g of fat) in 1989–92 (EFSA, 2007). The mean concentration of chlordane, measured as the sum of *cis*- and *trans*-chlordane, in 12 samples of breast milk from Arctic Canada in 1996 was 1.27 ng/g of fat, the values being 59 ng/g for oxychlordane, 4.29 ng/g for *cis*-nonachlor and 78 ng/g for *trans*-nonachlor (EFSA, 2007).

Chlordane were not detected in samples of Belgian human milk collected in 2006 during the fourth World Health Organization survey (Colles et al., 2008).

Dietary exposure assessment

Today food, particularly of animal origin is the primary source of chlordane exposure in the general population. This is because chlordane is no longer in use and because of the persistence and bioaccumulation of chlordane constituents and metabolites in the food chain (EFSA, 2007).

Dietary exposure of the Belgian adult population to chlordane has been estimated to 0.2 ng/kg bw/day (mean) and 1.13 ng/kg bw/day (P97.5) on basis on data from the belgian control plan 2010 and 2011. Fromberg et al. (2011) have estimated the dietary intake of chlordane for Danish adults to 1.5 ng/kg bw/day (mean), 2.6 ng/kg bw/day (P90) and 3.2 ng/kg bw/day (P95); Which is the same level as the estimate made by Darnerud et al. (2006) for Sweden (1.6 ng/kg bw/day). Calculated estimation for children was 2.5 ng/kg bw/day (mean), 4.6 ng/kg bw/day (P90) and 5.7 ng/kg bw/day (P95).

A total of 220 characteristic composite samples (representing an average food consumption basket) covering 205 food types in the form of 3696 individual samples were analyzed in the Czech Republic in 2004/2005. Based on the results of these investigations the daily dietary chlordane exposure was calculated as 7 ng/kg bw for 4 - 6 year old children and 1.5 – 2.5 ng/kg bw for adults (Ruprich, 2006).
Assessments of dietary chlordane intake between 1970 and 1996 were performed in Poland by multiplying the mean annual consumption rates of food commodities by the residue concentration in the respective food items. Estimated dietary intakes of chlordane constituents were between 6 and 7 ng/kg bw/day for a 60 kg adult. The highest contribution was found to be from fish with dietary exposures of 1.5 – 2 ng/kg bw/day for a 60 kg adult. A significant reduction in dietary chlordane exposure could not be observed between 1970 and 1996 (Falandysz, 2000, 2003).
Based on a market basket study performed in 1999, the dietary intake of chlordane and other organohalogen contaminants was assessed in Sweden. The estimated mean intake of chlordane (calculated as the sum of *cis*-chlordane, *trans*-chlordane, oxychlordane and *trans* nonachlor) was found to be 1.6 ng/kg bw per day based on the mean weight of 73.7 kg for the participants in the Swedish consumption study. Consumption of fish contributed 76% to this intake followed by fats/oils, meat, dairy products, pastries, and eggs with contributions of 10, 5.4, 4.0, 2.6 and 1.6%, respectively. This dietary chlordane exposure is considerably lower than in 1994 when a comparable assessment revealed a chlordane intake of 4.3 ng/kg bw/day for a 60 kg adult (Darnerud *et al*., 2006).

Median exposure to oxychlordane and *trans*-nonachlor of exclusively breastfed infants in the EU was recently estimated to be around 36 and 28 ng/kg bw, respectively (EFSA, 2007).

Risk characterization

The current human dietary exposure to chlordane is in the low ng/kg bw/day range, which is two to three orders of magnitude below the provisional tolerable daily intake (PTDI) of 500 ng/kg bw established by the WHO in 1995 (EFSA, 2007) (see table 3).

Table 3: Chlordane dietary exposure for adult and children and percentage of the PTDI

Population	Dietary exposure (ng/kg bw/day)	%PTDI (=500 ng/kg bw) (WHO, 1995)
Belgian, adults - Mean	0.2	0.04
Belgian, adults - P97,5	1.1	0.23
Belgian consumer, adults - Mean	2.3	0.47
Belgian consumer, adults - P97,5	5.9	1.17
Danish adults – Mean (Fromberg et al., 2011)	1.5	0.3
Danish adults - P90 (Fromberg et al., 2011)	2.6	0.52
Danish adults - P95 (Fromberg et al., 2011)	3.2	0.64
Danish children – Mean (Fromberg et al., 2011)	2.5	0.5
Danish children - P90 (Fromberg et al., 2011)	4.6	0.92
Danish children - P95 (Fromberg et al., 2011)	5.7	1,14
Adults Czech Republic (Ruprich, 2006)	1.5 – 2.5	0.3 – 0.5
Children Czech Republic (Ruprich, 2006)	7	1.4
Adults Poland (Falandysz, 2000, 2003)	6 - 7	1.2 – 1.4
Adult Sweeden – Mean (Darnerud *et al*., 2006)	1.6	0.32

The dietary exposure for the average Czech population (considering food consumption and the culinary factor) did not even reach one per cent of provisional tolerable daily intake of 500 ng/kg bw (Janouskova et al., 2005) (table 3).

Nougadère et al. (2011) have calculated an estimated daily intake (EDI) (mean % of TDI) between 1.4 and 37.5% for the French children and between 0.7 and 27.2% for the French adult on basis of monitoring results of 2005 and 2006.

Legislation

Regulation (EC) No 396/2005 of the European Parliament and of the Council of 23 February 2005 on maximum residue levels of pesticides in or on food and feed of plant and animal origin and amending Council Directive 91/414/EEC which will repeal the four Council Directives

Recommendations

The CONTAM Panel of EFSA made the following recommendations for chlordane in animal feed (EFSA, 2007):

- Besides *cis*- and *trans*-chlordane and oxychlordane, the analyses of feed samples, especially of marine origin, should also include the determination of *cis*- and *trans* nonachlor. Since MC5 and U82 are frequently found at the top of the marine food web and in human adipose tissues, they should also be included in the analytical scheme. Hence, standards for the latter two compounds need to be made commercially available.
- At low concentration of chlordane in biological samples, laboratories show large discrepancies in the performance in inter-comparison studies. Therefore improvement of the analytical methods is needed for environmental monitoring.
- The Members States are requested by the Commission to report the results of their monitoring programmes on undesirable substances in animal feed as compliant or non compliant only without information of concentrations determined. The availability of detailed

occurrence data concerning compounds and corresponding concentrations rather than condensed summary reports would be one prerequisite for an exposure assessment and identification of areas with an unusual high level of contamination. A European reporting system that facilitates these tasks should be set up.

- There are few toxicological data on target animal species. However, taking into consideration the long lasting ban of chlordane use and the low levels detected in animal feed, the Panel does not consider such data urgently needed.

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