

Rotterdam Convention - Operation of the Prior Informed Consent procedure for banned or severely restricted chemicals in international trade

Decision Guidance Document

Toxaphene



**Secretariat for the Rotterdam Convention
on the Prior Informed Consent Procedure for
Certain Hazardous Chemicals and Pesticides in
International Trade**



Mandate

The Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade was adopted at the Conference of Plenipotentiaries held in Rotterdam on 10 and 11 of September 1998. The Rotterdam Convention entered into force on 24 February 2004.

At its 6th session, held in Rome on 12-16 July 1999, the Intergovernmental Negotiating Committee adopted a decision guidance document for toxaphene (Decision INC-6/3) with the effect that this chemical became subject to the interim PIC procedure.

At its first meeting, held in Geneva 20 to 24 September 2004, the Conference of the Parties agreed to include toxaphene in Annex III of the Rotterdam Convention, with the effect that this chemical became subject to the PIC procedure.

The present decision guidance document for this chemical was communicated to the Designated National Authorities on 1 February 2005 with the request that they submit a response concerning the future import of these two chemicals to the Secretariat in accordance with Articles 7 and 10 of the Rotterdam Convention.

Purpose of the Decision Guidance Document

For each chemical included in Annex III of the Rotterdam Convention a decision guidance document has been approved by the Conference of the Parties. Decision guidance documents are sent to all Parties with a request that they provide a decision regarding future import of the chemical.

The decision guidance document is prepared by the Chemical Review Committee (CRC). The CRC is a group of government designated experts established in line with Article 18 of the Convention, that evaluates candidate chemicals for possible inclusion in the Convention. The decision guidance document reflects the information provided by two or more Parties in support of the national regulatory actions to ban or severely restrict the chemical. It is not intended as the only source of information on a chemical nor is it updated or revised following its adoption by the Conference of the Parties.

There may be additional Parties that have taken regulatory actions to ban or severely restrict the chemical as well as others that have not banned or severely restricted it. Such risk evaluations or information on alternative risk mitigation measures submitted by Parties may be found on the Rotterdam Convention web-site (www.pic.int).

Under Article 14 of the Convention, Parties can exchange scientific, technical, economic and legal information concerning the chemicals under the scope of the Convention including toxicological, ecotoxicological and safety information. This information may be provided directly to other Parties or through the Secretariat. Information provided to the Secretariat will be posted on the Rotterdam Convention website.

Information on the chemical may also be available from other sources.

Disclaimer

The use of trade names in this document is primarily intended to facilitate the correct identification of the chemical. It is not intended to imply any approval or disapproval of any

particular company. As it is not possible to include all trade names presently in use, only a number of commonly used and published trade names have been included in this document.

While the information provided is believed to be accurate according to data available at the time of preparation of this Decision Guidance Document, the Food and Agriculture Organization of the United Nations (FAO) and the United Nations Environment Programme (UNEP) disclaim any responsibility for omissions or any consequences that may flow therefrom. Neither FAO or UNEP shall be liable for any injury, loss, damage or prejudice of any kind that may be suffered as a result of importing or prohibiting the import of this chemical.

The designations employed and the presentation of material in this publication do not imply the expression of any opinion whatsoever on the part of FAO or UNEP concerning the legal status of any country, territory, city or area or of its authorities or concerning the delimitation of its frontiers or boundaries.

ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT

(N.B. Chemical elements and pesticides are not included in this list)

<	less than
≤	less than or equal to
<<	much less than
>	greater than
≥	greater than or equal to
µg	Microgram
a.i.	active ingredient
ACGIH	American Conference of Governmental Industrial Hygienists
ADI	acceptable daily intake
ADP	adenosine diphosphate
ATP	adenosine triphosphate
BBA	Biologische Bundesanstalt für Land- und Forstwirtschaft
b.p.	boiling point
Bw	body weight
°C	degree Celsius (centigrade)
CA	Chemicals Association
CCPR	Codex Committee on Pesticide Residues
CHO	Chinese hamster ovary
D	Dust
EC	Emulsifiable concentrates
EC50	Effect concentration, 50%
ED50	Effect dose, 50%
EHC	Environmental Health Criteria
ERL	Extraneous residue limit
EU	European Union
FAO	Food and Agriculture Organization of the United Nations
g	Gram
GAP	Good agricultural practice
GL	Guideline level
GR	Granules
ha	Hectare
i.m.	Intramuscular
i.p.	Intraperitoneal
IARC	International Agency for Research on Cancer

ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT

IC ₅₀	Inhibition concentration, 50%;
IPCS	International Programme on Chemical Safety
IRPTC	International Register of Potentially Toxic Chemicals
IUPAC	International Union of Pure and Applied Chemistry
JMPR	Joint FAO/WHO Meeting on Pesticide Residues (Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and a WHO Expert Group on Pesticide Residues)
k	Kilo- (x 1000)
kg	Kilogram
K _{oc}	Organic carbon-water partition coefficient
l	Litre
LC ₅₀	Lethal concentration, 50%
LD ₅₀	Lethal dose, 50%
LOAEL	Lowest observed adverse effect level
LD _{LO}	Lowest lethal dose
LOEL	lowest observed effect level
m	Metre
m.p.	melting point
mg	Milligram
ml	Millilitre
mPa	MilliPascal
MRL	maximum residue limit
MTD	maximum tolerated dose
NCI	National Cancer Institute
ng	Nanogram
NIOSH	National Institute of Occupational Safety and Health
NOAEL	no-observed-adverse-effect level
NOEL	no-observed-effect level
OP	organophosphorus pesticide
PHI	pre-harvest interval
PIC	prior informed consent
P _{ow}	octanol-water partition coefficient
POP	persistent organic pollutant
ppm	parts per million (used only with reference to the concentration of a pesticide in an experimental diet. In all other contexts the terms mg/kg or mg/l are used).
RfD	reference dose for chronic oral exposure
SBC	secretariat for the Basel Convention
SC	Soluble concentrate

ABBREVIATIONS WHICH MAY BE USED IN THIS DOCUMENT

SG	water soluble granules
SL	soluble concentrate
SMR	standardized mortality ratio
STEL	short term exposure limit
TADI	temporary acceptable daily intake
TLV	threshold limit value
TMDI	theoretical maximum daily intake
TMRL	temporary maximum residue limit
TWA	time weighted average
UNEP	United Nations Environment Programme
USEPA	United States Environmental Protection Agency
UV	Ultraviolet
VOC	volatile organic compound
WHO	World Health Organization
WP	wettable powder
Wt	Weight

PIC - Decision guidance document for a banned or severely restricted chemical

Toxaphene

Published: 1 September 1999

Reissued: 1 February 2005

Common name	Toxaphene (CA)
Other names/ synonyms	Camphechlor (ISO); chlorinated camphene; camphechlor; kamfochlor; octachloraphene; octachlorocamphene; polychlorinated camphenes; polychlorocamphene.
CAS No.	8001-35-2
Use category	Pesticide
Use	Toxaphene is a non-systemic contact and stomach insecticide with some acaricidal action. It has often been used in combination with other pesticides. It has been used as an insecticide to control armyworms, boll weevils, bollworms, cotton aphids, cotton fleahoppers, cotton leafworms, grasshoppers and others.
Trade names	Agricide maggot killer (f); alltex; Alttox; attac 4-2; attac 4-4; attac 6; attac 6-3; attac 8; camphochlor; camphofène huileux; chem-phène; Chem-Phène M5055; Chlor Chem T-590; Chlorocamphene; compound 3956; Crestoxo; cristoxo; cristoxo 90; Estonox; Fasco-Terpene; Geniphene; Gy-Phene; Hercules 3956; hercules toxaphene; Huilex; kamfochlor; m 5055; melipax; Motox; octachlorocamphene; Penphene; Phenacide; Phenatox; Polychlor-camphen; Strobane-T; toxadust; Toxakil; Toxaphene; Toxon 63; toxyphen; vertac toxaphene 90.
Formulation types	Dust formulations (D); emulsifiable concentrates (EC), granules (GR); wettable powders (WP).
Basic manufacturers	Hercules Inc, Boots, Drexel, Fahlberg-List.

Reasons for inclusion in the PIC procedure

Toxaphene is included in the PIC procedure as a pesticide. Inclusion was recommended at the eighth meeting of the FAO/UNEP Joint Group of Experts on Prior Informed Consent following detailed discussions during the sixth and seventh meetings. It is included in the procedure on the basis of the control actions reported by a number of Governments.

Summary of control actions (see Annex 2 for details)

Control actions have been reported by 18 countries and the European Union. In 16 countries (Austria, Canada, Cuba, Finland, Germany, India, Indonesia, Kuwait, Morocco, Pakistan, Republic of Korea, Slovenia, Switzerland, Thailand, United Kingdom, the United States of America) and the European Union toxaphene was reported as banned. Colombia and Belize reported that the use of toxaphene is severely restricted. Remaining uses were the control of froghoppers in rice (Belize) and aerial spraying of cotton (Colombia). The reasons for the control actions were concerns about the risk to human health related to the use of toxaphene as well as concern about environmental pollution.

Hazard classification by organization

WHO (WHO, 1996)	Technical product: Class II (moderately hazardous), classification based on an oral LD ₅₀ of 80 mg/kg bw.				
	Classification of formulations				
		Oral toxicity		dermal toxicity	
		LD ₅₀ : 80 mg/kg bw (see Annex 1)		LD ₅₀ : 780 mg/kg bw (see Annex 1)	
	Formulation	a.i. (%)	Hazard class	a.i. (%)	Hazard class
	Solid	>15 <15	II III	All conc.	III
USEPA	B2; probable human carcinogen (USEPA, 1994).				
EU	Toxic, carcinogen Cat.3 (T: R25, R40 (Carc. Cat. 3); Xn: R21-R37/38; N: R50-53) (classification in accordance with Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances).				
IARC	Group 2B (possibly carcinogenic to humans) (IARC, 1987).				

Protective measures that have been applied concerning the chemical

Measures to reduce exposure

For the health and welfare of workers and the general public, the handling and application of the substance should be entrusted only to competently supervised and well-trained applicators who must follow adequate safety measures and use the chemical according to good application practices. Regularly exposed workers should receive appropriate monitoring and health evaluations. Protective clothing as indicated in the *FAO Guidelines for Personal Protection when Working with Pesticides in Tropical Climates* (1990) is required.

Toxaphene is one of twelve persistent organic pollutants (POPs) which are currently being considered for international action to reduce/eliminate their releases under a global POPs convention, that should be completed by the year 2000 (UNEP, 1997 ; Ritter et al., 1995).

Packaging and labelling

Follow the *FAO Revised Guidelines on Good Labelling Practice for Pesticides* (1995).

The United Nations Committee of Experts on the Transportation of Dangerous Goods classifies the chemical in:

Hazard class	6.1	Poisonous substance.
Packing group	3	Substance presenting relatively low risk of poisoning in transport (toxaphene concentrations 40% solid or 15 % liquid). (IPCS/CEC, 1993)

Alternatives

Several countries which reported control actions (Austria, Germany, India, Indonesia, Morocco, Thailand, United States of America) indicated alternatives (see Annex 2).

It is essential that, before a country considers substituting any of the reported alternatives, it ensures that the use is relevant to its national needs. A first step may be to contact the designated national authority in the country where the alternative has been reported (see addresses of designated national authorities in Annex 3). It would then be necessary to determine the compatibility with national crop protection practices.

Waste disposal

Waste should be disposed of in accordance with the provisions of the Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and Their Disposal and any guidelines thereunder (SBC, 1994).

See the FAO Guidelines on Prevention of Accumulation of Obsolete Pesticide Stocks (1995) and The Pesticide Storage and Stock Control Manual (1996).

Wear protective clothing and respiratory equipment suitable for toxic materials. Sweep, scoop or pick up spilled material. Vacuuming or wet sweeping may be used to avoid dust dispersal. Do not flush to surface water or sanitary sewer system. Dispose of empty containers in a sanitary landfill or by incineration.

Waste material should be burned only in multiple chamber incinerators designed for organochlorine waste disposal (1000°C and 30-min residence time, with effluent gas scrubbing). Not all incinerators are suitable for carcinogens.

It should be noted that the methods recommended in the literature are often not suitable in a specific country. High temperature incinerators may not be available. Consideration should be given to the use of alternative destruction technologies.

Exposure limits

	Type of limit	Value
Food	MRLs (Maximum Residue Limits in mg/kg) in specified products (FAO/WHO, 1974).	No MRLs allocated.
	JMPR ADI (Acceptable Daily Intake) in mg/kg diet (FAO/WHO, 1974).	No ADI allocated.
Workplace	USA (ACGIH, 1998) TLV-TWA (Threshold Limit Value, Time-Weighted Average in mg/m ³) (ACGIH, 1998).	0.5 mg/m ³ .

First aid

Persons who have been poisoned (accidentally or otherwise) should be transported immediately to a hospital and placed under the surveillance of properly trained medical staff.

Eyes: Immediately flush eyes with plenty of water for at least 15 minutes, occasionally lifting the upper and lower lids. Seek medical attention immediately.

Skin: Flush skin with plenty of soap and water for at least 15 minutes before removing contaminated clothing and shoes.

Ingestion: Do not induce vomiting. Have the victim rinse his or her mouth and then drink 2-4 cupfuls of water, and seek medical advice.

Inhalation: Remove from exposure into fresh air immediately. Begin rescue breathing if breathing has stopped and cardio-pulmonary resuscitation if heart action has stopped. Transfer promptly to a medical facility. Medical observation is recommended for 24 to 48 hours after breathing overexposure, as pulmonary oedema may be delayed (*USEPA, 1987*).

Annexes

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| Annex 1 | Further information on the substance |
| Annex 2 | Details on reported control actions |
| Annex 3 | List of designated national authorities |
| Annex 4 | References |

Annex 1 - Further information on the substance

1 Chemical and physical properties

1.1	Identity	Toxaphene is an amber, waxy solid consisting of a complex mixture of polychlorinated bicyclic terpenes.
1.2	Formula	C ₁₀ H ₁₀ Cl ₈ (the chlorination grade may vary).
	Chemical name	Toxaphene (CA)
	Chemical type	Chlorinated hydrocarbons
1.3	Solubility	Water: 3 mg/L at 25 °C; soluble in organic solvents (<i>Worthing and Walker, 1987</i>).
	log P _{ow}	3.3 (<i>Callahan et al., 1979</i>)
1.4	Vapour pressure	0.2-0.4 mm Hg at 25 °C (<i>Hayes, 1982</i>).
1.5	Melting point	65-90 °C (<i>Budvari, 1989</i>)
1.6	Reactivity	This chemical is decomposed in the presence of an alkali (<i>Budvari, 1989; Sax, 1987; IARC, 1979; IARC, 1987</i>). It is corrosive to iron (<i>Budvari, 1989</i>). Toxaphene is incompatible with strong oxidizers (<i>Sittig, 1985</i>) and is non-corrosive in the absence of moisture (<i>IARC, 1979</i>).

2 Toxicity

2.1 General

2.1.1	Mode of action	Toxaphene is an insecticide with stomach and contact action and a rodenticide acting as stomach poison. The mechanism of action is relatively unknown. It has little initial but a persistent effect against insects. At temperatures below 16 °C a reduced effect is expected because of the pests' lower feeding activity (<i>EHC, 1984</i>).
2.1.2	Uptake	Toxaphene is absorbed following ingestion and inhalation, as well as through the skin (<i>EHC, 1984</i>).
2.1.3	Metabolism	When absorbed, toxaphene is rapidly distributed to all organs of the body and tends to concentrate in fatty tissues and muscle from which it is slowly released. Circulating toxaphene is primarily metabolized by hepatic mixed function oxidases. Toxaphene and its metabolites are excreted in faeces and urine. Both hydroxylation and dechlorination products have been found as metabolites (<i>EHC, 1984</i>).

2.2 Known effects on human health

2.2.1	Acute toxicity	
	Symptoms of poisoning	Toxaphene is an irritant. It is a central and peripheral neurotoxin. It can cause liver and kidney damage. (<i>EHC, 1984</i>) Symptoms of mild poisoning: dizziness, nausea, abdominal pain and vomiting.

In chronic poisoning, loss of weight and appetite, temporary deafness and disorientation may occur.

Moderate or severe poisoning: mild signs followed by severe irritability, convulsive seizures and coma. Seizures may be epileptiform in character with frothing at the mouth, facial congestion, violent convulsive movements or stiffness of the limbs associated with stupor or coma. In severe cases, the convulsions may be continuous, with elevated body temperatures, unconsciousness, laboured breathing with vigorous, rapid heart beat and eventually death.

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| 2.2.2 | Short- and long-term exposure | <p>Two workers, while harvesting cotton sprayed with toxaphene, developed dyspnoea and reduced pulmonary function and were found to have miliary opacities distributed over their lung fields (<i>Warraki, 1963</i>).</p> <p>Eight women working in fields sprayed with toxaphene at a rate of 2 kg/ha were reported to have a higher incidence of chromosome aberrations in cultured lymphocytes than in control individuals (<i>Samosh, 1974</i>).</p> <p>Two cases of acute aplastic anaemia associated with dermal exposure to toxaphene-lindane mixtures, with one death due to myelomonocytic leukaemia, have been reported (<i>IARC, 1979</i>).</p> |
| 2.2.3 | Epidemiological studies | <p>Twenty-five human volunteers were exposed to an aerosol of toxaphene at a maximal nominal concentration of 500 mg/m³ in a closed chamber for 30 minutes per day on 10 consecutive days (<i>Shelansky, 1947</i>). After 3 weeks, they received the same exposure on 3 consecutive days. Assuming a retention of 50% of the inhaled toxaphene, each individual absorbed 75 mg toxaphene per day or approximately 1 mg/kg body weight per day. Physical examinations and blood and urine tests did not reveal any abnormalities (<i>EHC, 1984</i>).</p> |

2.3 Toxicity studies with laboratory animals and *in vitro* systems

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| 2.3.1 | Acute toxicity | |
| | oral | The LD ₅₀ , if administered orally to mice and hamsters, was in the range of 112 – 200 mg/kg bw (<i>Richardson, 1993</i>). |
| | Dermal | The dermal LD ₅₀ for different species was determined as 300 – 1000 (a.i.; mg/kg bw) (<i>Gaines 1969</i>); (<i>USEPA 1976</i>). |
| | Inhalation | After inhalation, the LC _{Lo} (2hr) was 2000 mg/m ³ for mice (<i>Richardson, 1993</i>). |
| | Irritation | 500 mg toxaphene applied to the skin of rabbits caused erythema and edema (<i>Johnston and Eden, 1953</i>). |
| 2.3.2 | Short- and long-term exposure | <p>In a 90-day feeding trial with growing rats, the NOEL for toxaphene was 10 mg per rat per day (<i>BBA, 1990</i>).</p> <p>5.0 mg/kg bw/day was an effect level for decreased hepato-biliary function in rats treated with toxaphene (<i>Richardson, 1993</i>).</p> <p>0.35 mg/kg bw/day was a NOAEL for thyroid cytoarchitectural changes in rats. This value can be used as starting point for risk assessment after short-term exposure (<i>Chu et al., 1986</i>).</p> |

- 2 Long-term
 . exposure
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- In a nine-month study with two groups of 12 rats, histological changes in the liver were observed in three rats of the 2.5 mg/kg-dose group and in six rats of the 10 mg/kg-dose group (*WHO/FAO, 1975*).
- In feeding studies with four groups each of 40 rats fed 10, 100, 1000 and 1500 ppm, the liver weight and liver-to-body weight ratio were significantly increased only in the 1000 and 1500 ppm groups. Liver changes consisted of swelling and homogeneity of the cytoplasm with a peripheral arrangement of the granules in the cytoplasm of the centrilobular hepatic cells (*Treon et al., 1952*).
- Fitzhugh and Nelson (1951) and Lehman (1952) found centrilobular hepatic cell enlargement with increased oxyphilia, peripheral margination of basophilic granules and a tendency to hyalinization of the remainder of the cytoplasm in the livers of rats fed 400 ppm in the diet. Minor tissue damage at 100 ppm, no effect at 25 ppm.
- Kock and Wagner (1989) reported that 50 to 200 mg toxaphene/kg included with the diet caused hypertrophy in the liver cells of rats after two to nine months. The NOEL is given with 25 mg/kg diet as a result of a two-year feeding study (*RSC, 1988; Worthing and Walker, 1987*).
- 2.3.4 Effects on reproduction Adverse developmental effects have been observed in laboratory animals following toxaphene ingestion at doses below those required to induce maternal toxicity. The most sensitive endpoints of foetal toxicity appear to be behavioural effects and immunosuppression (*Kennedy et al., 1973*).
- 2.3.5 Mutagenicity Toxaphene induces sister chromatid exchanges in human lymphoid cells with and without metabolic activation (*IARC, 1979*).
- Chromosome aberrations were not found *in vivo* in human leukocytes (*Saleh, 1977*).
- Toxaphene did not induce dominant lethal mutation in mice (*Epstein et al., 1972*).
- In an *in vitro* test to determine breakage of DNA in bacteria, toxaphene did not induce breaks at a significantly higher rate than that occurring in controls (*Griffin and Hill, 1978*).
- Toxaphene was mutagenic in a test using *S. typhimurium* without requiring activation by liver homogenate (*Hooper et al., 1979*).
- Toxaphene was positive in *S. typhimurium* TA 98 and TA 100, both with and without metabolic activation, in TA 1537 without activation and it was negative in TA 1535 with and without activation and TA 1537 with activation (*IARC, 1979*).
- Toxaphene induces sister chromatid exchanges in Chinese hamster ovary (CHO) cells with and without metabolic activation (*Sobti et al., 1983*).
- 2.3.6 Carcinogenicity It is noted that hepatocellular carcinomas were found in mice and follicular cell carcinomas in the thyroid were increased in rats (*IARC, 1987*).
- A bioassay of technical grade toxaphene was conducted in groups of 50 mice and rats of both sexes by administering the test chemical in the feed. The incidence of hepatocellular carcinoma was dose-related in both males and

females (NCI, 1979).

Fifty rats of both sexes were administered toxaphene in their diets at time-weighted average doses of 556 and 1112 mg/kg for males, and 540 and 1080 mg/kg for females. Follicular carcinoma or thyroid adenoma was observed in 26% of the high dose group and 17% of the low dose group of male rats. Thyroid tumours were observed in 17% of the female high dose group and in 2% of the female low dose group. On the basis of this study, it was concluded that the results “suggest carcinogenicity of toxaphene for the thyroid of male and female Osborne-Mendel rats” (NCI, 1979).

3 Exposure		
3.1	Food	Consumption of contaminated food appears to be the major source of exposure for the general population (USEPA, 1987; Howard, 1991).
3.2	Occupational	Dyspnoea and reduced pulmonary function in two workers involved in harvesting cotton; miliary opacities distributed over lung fields were also observed (Warraki, 1963). Higher incidence of chromosome aberration was found in cultured lymphocytes of eight women working in an area that had been sprayed with the substance than in control individuals (Samosh, 1974). In a survey of 199 employees who worked or had worked with toxaphene between 1949 and 1977, with exposure ranging from 6 months to 26 years (mean 5.23 years), 20 employees died, 1 from cancer of the colon. None of these deaths appeared to be related to exposure to toxaphene (USEPA, 1979).
3.3	Environment	Toxaphene has not been found in drinking water supplies at detectable levels. Studies of surface and groundwaters generally have not found detectable levels. A few instances of surface water contamination at levels around 1 ppb have been reported (USEPA, 1987; Howard, 1991).
3.4	Accidental poisoning	In 1952, 10 cases of poisoning with 3 deaths were described. All had ingested toxaphene accidentally either in the form of an insecticide in the case of the children, or the adults as toxaphene-polluted food. Four of the cases were children aged between 17 months and 4 years old, three of whom died. All the adults survived. The 3 deaths occurred within several hours and those who recovered did so in 24 hours (McGee et al., 1952). A 16-month-old child died after ingesting a substance presumed to be toxaphene (Pollock, 1953). Another 9-month-old child's death was reported due to ingestion of dust containing toxaphene and DDT (Pollock, 1953).

4 Effects on the environment		
4.1 Fate		
4.1.1	Persistence	Toxaphene in the atmosphere has been shown to be transported over long distances. Reaction with hydroxyl radicals is expected to degrade toxaphene

in the air.

Toxaphene released to surface waters adsorbs strongly to sediment. It is also subject to evaporation. Hydrolysis, photolysis and biodegradation are not significant (*Paris, 1973*).

Toxaphene binds strongly to soils and is very resistant to degradation. Biodegradation is enhanced under flooded or anaerobic conditions. Migration to groundwater is unlikely (*Parr, 1976*).

Biodegradation of toxaphene occurs slowly in soil under anaerobic conditions. Toxaphene is a highly persistent pesticide. It is not degraded in surface water (*USEPA, 1987; Howard, 1991*).

- 4.1.2 Bioconcentration Toxaphene is a highly bioaccumulating compound. It bioaccumulates readily in aquatic organisms. The bioconcentration factor for fish varies from 3100 to 33300. (*USEPA, 1987; Howard, 1991*).

4.2 Ecotoxicity

- 4.2.1 Fish The 96-hour LC₅₀ is in the range of 1-20 µg/l; Toxaphene is highly toxic to fish (*Burke and Ferguson, 1969; Macek and McAllister, 1970*).

- 4.2.2 Aquatic invertebrates Toxaphene is highly toxic to aquatic invertebrates (*Courtenay and Roberts, 1973; Schimmel et al., 1977; Sanders and Cope, 1966*).

- 4.2.3 Birds Five-day dietary toxicity studies produce LC₅₀ values between 538 and 828 mg/kg feed depending on the bird species (*Hill et al., 1975*).

300 mg/kg toxaphene in the feed of pheasants reduced egg-laying and hatching, and 100 mg/kg significantly increased the mortality of chickens (*Korte et al., 1979*).

A number of experiments with birds substantiate the suspicion that toxaphene is a potentially toxic substance for wild birds under certain conditions in the field (*BBA, 1990; Pollock and Kilgore, 1978; Kuhnert, 1991*).

- 4.2.4 Bees Toxaphene is of low toxicity to bees. Atkins et al. (1975) found the LD₅₀ of the substance was 50.4 µg per bee.

German authorization documents (*BBA, 1990*) give the following values for the toxicity of toxaphene to bees:

LD₅₀ per os = 521.0 ± 62.79 ppm

LD₅₀ topical = 145.3 ± 6.68 ppm

LD₅₀ deposit = 3193.0 ± 141 µg/100 cm².

Jumar and Sieber (1967) found that bees fed with a sugar solution containing ⁸²bromo toxaphene first stored toxaphene to 95% in the body and then excreted it in the next few days as a chlorine-containing, water-soluble product. Obviously, bees are able to decompose and detoxify the substance.

Annex 2 - Details on reported control actions

AUSTRIA

Effective:	1992
Control action:	All uses banned.
Reasons:	A number of studies suggest that this pesticide and related products are carcinogenic in mice and rats, especially for the liver and thyroid. Toxaphene is suspected to have a foetal toxicity and effects on reproduction. A further problem is its high persistence in the environment and its bioaccumulation in the food chain.
Alternatives:	Many alternatives for designated purposes.

BELIZE

Control action:	Severely restricted.
Uses still allowed:	Temporary conditional registration granted for the control of froghoppers in the current rice crop only.
Reasons:	Environmental pollutant.

CANADA

Effective:	1982
Control action:	Most uses phased out between 1970 and 1980. Registration of last remaining product (for livestock use) was inactivated by the registrant in 1982.
Reasons:	Environmental persistence and bioaccumulation of residues. Difficulty in quantifying residues.

COLOMBIA

Effective:	1988
Control action:	Only mixtures with methyl parathion are allowed for use on cotton. (Sólo se permiten mezclas con metil paration en el algodónero.)
Uses still allowed:	Application by aerial spraying on cotton still allowed. (Aplicaciones por vía aérea en el algodónero.)
Reasons:	Its high toxicity requires careful technical management. (Su alta toxicidad requiere un manejo muy técnico y cuidadoso.)

CUBA

Effective:	1990
Control action:	The substance is banned for use, production and import as a pesticide (La sustancia está prohibida para su uso, producción e importación como plaguicida).
Reasons:	It is a highly toxic and dangerous substance. Known to induce cancer in experimental animals. Proven to be persistent in children and adult population. (Es una sustancia de alta toxicidad y gran peligrosidad. Se conoce la inducción al cáncer en especies experimentales. Su persistencia se ha demostrado en población infantil y adulta).

EUROPEAN UNION

Effective:	1984
Control action:	It is prohibited to use or place on the market all plant protection products containing toxaphene (camphechlor) as an active ingredient. No remaining uses allowed.
Reasons:	Toxaphene (camphechlor) is persistent in the environment. It is likely to bioaccumulate and produce food-chain effect on terrestrial and aquatic organisms. Toxaphene (camphechlor) has been classified by the European Community as a category 3 carcinogen (possibly carcinogenic to humans). It is extremely toxic to fish.

(Member States of the European Union are: Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Luxembourg, Netherlands, Portugal, Spain, Sweden, United Kingdom.)

FINLAND

Effective:	1970
Control action:	Total ban to use as pesticide.
Reasons:	High risk to human health.

GERMANY

Effective:	1981
Control action:	Totally banned for use as plant protection product.
Reasons:	High persistence; accumulation in food chain; carcinogenic effect in animal experiments; intolerable residues in foodstuffs; drinking water protection; game protection.
Alternatives:	Carbamate and organic phosphorous insecticides, pyrethroids; plant protection products containing chlorphacinon and zinc phosphide for the control of field rodents.

INDIA

Effective: 1989
Control action: Banned.
Reasons: Action taken owing to persistence in environment, food and agricultural products. It is also reported to be a possible cause of cancer.
Alternatives: Malathion, monocrotophos, chlorpyrifos.

INDONESIA

Effective: 1980
Control action: Prohibited to be used for all purposes.
Uses still allowed: No remaining uses allowed.
Reasons: The substance causes harmful effect to humans and environment.
Alternatives: Agrothion 50 EC (Phenitrotrion). Curacron 500 EC (Prophenophos). Decis 2,5 EC (Deltamethrin).

KUWAIT

Effective: 1980
Control action: The substance is banned for use as a pesticide.
Uses still allowed: No remaining uses allowed.
Reasons: Action was taken for health and environmental reasons.

MOROCCO

Effective: 1984
Control action: Banned for use in agriculture since 1984. (Interdit en agriculture depuis 1984.)
Uses still allowed: No remaining uses allowed. (Toute utilisation dudit pesticide est interdite.)
Reasons: High toxicity, persistence in the environment and bioaccumulation of residues in the food chain. (Toxicité très élevée, persistance dans l'environnement et bioaccumulation des résidus dans la chaîne alimentaire).
Alternatives: Alternative products are insecticides belonging to other groups of chemicals such as synthetic pyrethrinoides, organo-phosphorated, carbamates. (Les produits de remplacement sont des insecticides appartenant à d'autres familles chimiques - les organo-phosphorés, les pyrethrinoid des synthèse, les carbamates ...).

PAKISTAN

Effective: 1982
Control action: Prohibited. No remaining uses allowed.

REPUBLIC OF KOREA

Effective: 1991
Control Action: Banned for production, import, use and sale of both this substance and preparations containing it.
Reasons: Action taken because of mutagenic and carcinogenic effects; bioaccumulation of residue.

SLOVENIA

Effective: 1997
Control action: Banned for use in agriculture.
Reasons: This chemical was banned from the use in agriculture due to the effect of its toxic properties to human health and the environment according to the opinion given by the Commission on Poisons.

SWITZERLAND

Effective: 1986
Control action: Toxaphene(or Camphechlor) is a totally banned chemical: Manufacture, supply, import and use of the substance and of products which contain the substance is prohibited. (Applies to reactive mixtures of chlorinated camphenes containing 67-69% chlorine).
Reasons: Long persistence, bioaccumulation.

THAILAND

Effective: 1983
Control action: All use categories have been banned.
Reasons: Possibly carcinogenic to humans. Persistence.
Alternatives: Chlorpyrifos, fenitrothion, phosalone and synthetic pyrethroids.

UNITED KINGDOM

Effective: 1984
Control action: All uses revoked.
Uses still allowed: No remaining uses allowed.
Reasons: Action taken due to environmental hazard (persistent organochlorine).

UNITED STATES OF AMERICA

Effective:	1987
Control action:	Campechlor (Toxaphene) is banned for use. In 1977, EPA initiated a special investigation into the health effects of campechlor. In October 1982, most uses were cancelled because of potential effects on man and non-target species. A small number of uses were retained under specific restrictions and limited conditions. These uses were all subsequently cancelled in July 1987. No remaining uses allowed.
Reasons:	Toxaphene was linked to acute oral toxicity and carcinogenicity in humans, population reductions of non-target species, acute toxicology to aquatic organisms, and chronic and/or delayed effects to aquatic, avian, and mammalian species. In addition, toxaphene bioaccumulates.
Alternatives:	Agricultural crops: carbaryl, methyl parathion, esfenvalerate, permethrin, malathion, endosulfan, chlorpyrifos; Other uses: malathion, permethrin, coumaphos, stirofos, methoxychlor, pyrethrins; For cattle and hog only: amitraz.

Annex 3 - List of designated national authorities

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MOROCCO

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SWITZERLAND

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