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A REVIEW OF SELECTED PERSISTENT ORGANIC POLLUTANTS

DDT-Aldrin-Dieldrin-Endrin-Chlordane

Heptachlor-Hexachlorobenzene-Mïrex-Toxaphene

Polychlorinated biphenyls

Dioxins and Furans

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For:

The International Programme on Chemical Safety (IPCS)
within the framework of the
Inter-Organization Programme for
the Sound Management of Chemicals (IOMC)

This Review is produced for the International Programme on Chemical Safety (IPCS). The work is carried out within the framework of the Inter-Organization: Programme for the Sound Management of Chemicals (IOMC).

The Review does not necessarily represent the decisions or the stated policy of the United Nations Environment Programme, the International Labour Organisation, or the World Health Organization.

The International Programme on Chemical Safety (IPCS) is a joint venture of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. The main objective of the IPCS is to carry out and disseminate evaluations of the effects of chemicals on human health and the quality of the environment. Supporting activities include the development of epidemiological, experimental laboratory, and risk-assessment methods that could produce internationally comparable results, and the development of human resources in the field of chemical safety. Other activities carried out by the IPCS include the development of know-how for coping with chemical accidents, strengthening capabilities for prevention of an response to chemical accidents and their follow-up, coordination of laboratory testing and epidemiological studies, and promotion of research on the mechanisms of the biological action of chemicals.

The Inter-Organization Programme for the Sound Management of Chemicals (IOMC), was established in 1995 by UNEP, ILO, FAO, WHO, UNIDO, and OECD (Participating Institutions), following recommendations made by the 1992 UN Conference on Environment and Development to strengthen cooperation and increase international coordination in the field of chemical safety. The purpose of the IOMC is to promote coordination of the policies and activities pursued by the Participating Organizations, jointly or separately, to achieve the sound management of chemicals in relation to human health and the environment.

PREFACE

At its Ninth meeting in May 1995, the UNEP Governing Council adopted Decision 18/32 concerning Persistent Organic Pollutants. The decision invites the Inter-Organization Programme for the Sound Management of Chemicals (IOMC), working with the International Programme on Chemical Safety (IPCS) and the Intergovernmental Forum on Chemical Safety (IFCS) to undertake an expeditious assessment process addressing persistent organic pollutants (POPs). This process is to initially begin with 12 specific compounds[1] and should consolidate existing information on the relevant chemistry and toxicology, transport and disposition, as well as the availability and costs of substitutes to these substances. The effort will also assess realistic response strategies, policies, and mechanisms for reducing and/or eliminating emissions, discharges, and other losses of these substances. This information will serve as the basis for recommendations to be developed by the IFCS on potential international actions to be considered at the session of the UNEP Governing Council and the World Health Assembly in 1997.

IPCS, in consultation with the organizations participating in the IOMC, has proceeded with the initial phase of the work. The initial effort aims to compile the existing information on the chemistry, toxicology, relevant transport pathways and the origin, transport and disposition of the substances concerned and additionally, reference briefly what information is available on the costs and benefits associated with substitutes, and the socio-economic aspects of the issue. The effort builds on ongoing activities including the substantial work in progress under the Long-range Transboundary Air Pollution Convention and the 1995 International Expert Meeting on POPs sponsored by Canada and the Philippines.

This Review document is the full text of a companion document "Persistent Organic Pollutants: An Assessment Report on DDT, Aldrin, Dieldrin, Endrin, Chlordane, Heptachlor, Hexachlorobenzene, Mirex, Toxaphene, Polychlorinated Biphenyls, Dioxins and Furans (PCS 95.38)". The reader who desires a shortened version should consult the Assessment Report cited above. These documents will serve as a basis for development of a workplan to complete the assessment process called for in the UNEP Governing Council Decision.

Readers of this Review are reminded that definitions used herein are not the result of any international discussion or agreement, but rather are solely for the use of this paper.

[1] Substances identified in the UNEP Governing Council Decision on Persistent Organic Pollutants include PCBs, dioxins and furans, aldrin, dieldrin, DDT, endrin, chlordane, hexachlorobenzene, mirex, toxaphene, and heptachlor.

1. SUMMARY

Persistent organic pollutants (POPs) are organic compounds that, to a varying degree, resist photolytic, biological and chemical degradation. They are characterized by low water solubility and high water solubility, leading to their bioaccumulation in fatty tissues. They are also semi-volatile, enabling them to move long distances in the atmosphere before deposition occurs. Although many different forms of persistent organic pollutants may exist, both natural and anthropogenic, persistent organic pollutants which are noted for their persistence and bioaccumulative characteristics include many of the first generation organochlorine insecticides such as dieldrin, DDT, toxaphene and chlordane and several industrial chemical products or byproducts including polychlorinated biphenyls (PCBs), dibenzo-p-dioxans (dioxins) and dibenzo-p-furans (furans). Many of these compounds have been or continue to be used in large quantities and, due to their environmental persistence, have the ability to bioaccumulate and biomagnify. Some of these compounds such as PCBs, may persist in the environment for periods of years and may bioconcentrate by factors of up to 70,000 fold.

Persistent organic pollutants are also noted for their semi-volatility; that property of their physico-chemical characteristics that permit these compounds to occur either in the vapour phase or adsorbed on atmospheric particles, thereby facilitating their long range transport through the atmosphere. The properties of unusual persistence, when coupled with other characteristics such as semi-volatility, have resulted in the presence of compounds such as PCBs all over the world, even in regions where they have never been used. POPs are ubiquitous. They have been detected in both industrialized and non-industrialized, in urban and rural localities, in densely populated areas and in those that are sparsely inhabited. POPs have been measured in every continent at sites representing every major climatic zone and geographic sector throughout the world. These include remote regions such as the open oceans, the deserts, the Arctic and the Antarctic, where no significant local sources exist and the only reasonable explanation for their presence is long-range transport from other parts of the globe. PCBs have been reported in air, in all areas of the world, at concentrations up to 15ng/m^3; in industrialized areas, concentrations may be several orders of magnitude greater. PCBs have also been reported in rain and snow.

The group of persistent organic pollutants includes two types of important compounds: polycyclic aromatic hydrocarbons and halogenated hydrocarbons. This latter group includes the organochlorines which, historically, have proven to be most resistant to degradation and which have had the widest production, use and release characteristics. Organochlorines are also generally the most persistent of all the halogenated hydrocarbons. In general, it is known that the more highly chlorinated biphenyls tend to accumulate to a greater extent than the less chlorinated PCBs; similarly, metabolism and excretion are also more rapid for the less chlorinated PCBs than for the highly chlorinated biphenyls.

Humans can be exposed to POPs through the direct exposure, occupational accidents and the environment (including indoor). Short-term exposures to high concentrations of POPs may result in illness and death. Chronic exposure to POPs may also be associated with a wide range of adverse health and environmental effects.

Laboratory investigations and environmental impact studies in wildlife have provided evidence that persistent organic pollutants may be involved with endocrine disruption, reproductive and immune dysfunction, neurobehavioral and developmental disorders and cancer. More recently some authors have implicated persistent organic pollutants in reduced immunity in infants and children, and the concomitant increase in infection, also with developmental abnormalities, neurobehavioural impairment and cancer and tumour induction or promotion. Some POPs are being considered as a potentially important risk factor in. the etiology of human breast cancer.

2. INTRODUCTION

The behaviour and fate of chemicals in the environment is determined by their chemical and physical properties and by the nature of their environment. The chemical and physical properties are determined by the structure of the molecule and the nature of the atoms present in the molecule. Depending on the structure of the molecule, these physical and chemical properties span a large range of values. Compounds may be of very low persistence, of low toxicity and be immobile. At low levels of exposure, these compounds are unlikely to present a risk to the environment or to human health. At the other end of the scale are those compounds that are persistent, mobile and toxic and it is this range of the distribution where the persistent toxic and lipophilic organic pollutants are found. It must be recognized that relatively few substances possess the necessary properties to make them persistent organic pollutants. In fact, if the range of these properties were presented as a distribution, only those compounds at the extreme ends of the distribution would express the degree of persistence, mobility and toxicity to rank them as persistent organic pollutants (Figure 1).

2.1 PERSISTENCE, MOBILITY AND BIOAVAILABILITY

Some substances may be very persistent in the environment (i. e. with half-lives ($t_{1/2}$) greater than 6 months). The nature of this persistence needs to be clarified - it is the length of time the compound will remain in the environment before being broken down or degraded into other and less hazardous substances. Dissipation is the disappearance of a substance and is a combination of at least two processes, degradation and mobility. It is not an appropriate measure of persistence as mobility may merely result in the substance being transported to other locations where, if critical concentrations are achieved, harmful effects may occur.

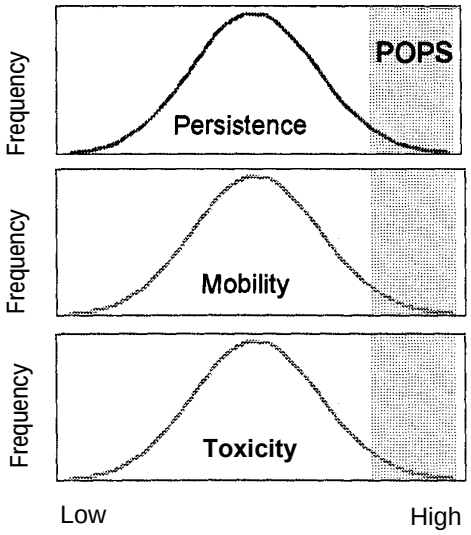


Figure 1 Illustration showing the combination of properties needed for a substance to be a persistent organic pollutant.

One important property of persistent organic pollutants is that of semi-volatility. This property confers a degree of mobility through the atmosphere that is sufficient to allow relatively great amounts to enter the atmosphere and be transported over long distances. This moderate volatility does not result in the substance remaining permanently in the atmosphere where it would present little direct risk to humans and organisms in the environment. Thus, these substances may volatilize from hot regions but will condense and tend to remain in colder regions. Substances with this property are usually highly halogenated, have a molecular weight of 200 to 500 and a vapor pressure lower than 1000 Pa.

In order to concentrate in organisms in the environment, persistent organic pollutants must also possess a property that results in their movement into organisms. This property is lipophilicity or a tendency to preferentially dissolve in fats and lipids, rather than water. High lipophilicity results in the substance bioconcentrating from the surrounding medium into the organism. Combined with persistence and a resistance to biological degradation, lipophilicity also results in biomagnification

through the food chain. Biomagnification results in much greater exposures in organisms at the top of the food chain.

2.2 LONG-RANGE TRANSPORT

Persistent organic pollutants must therefore, by definition, be more persistent, more mobile A and more bioavailable than other substances. These properites are conferred by the structural makeup of the moleculas and are often associated with greater degrees of halogenation. Included in this group of substances are some older chlorinated pesticides like DDT and the chlordanes, polychlorinated biphenyls, polychlorinated benzenes, and polychlorinated dioxins (PCDDs) and furans (PCDFs). The physico-chemical properties of these compounds are such that they favour sufficiently high atmospheric concentrations that result in global redistribution by evaporation and atmospheric transport.

3. ENVIRONMENTAL FATE AND TRANSPORT

3.1 INTRODUCTION

Aknowledge of the factors that affect the fate and transportation of persistent organic pollutants is critical to understanding how and why these substances have become a worldwide problem. It is not our intention to describe in detail the wide range of environmental processes that result in exposure of humans and the environment to persistent organic pollutants. The number of substances and processes is large, and, even for well-known substances, the information available is often incomplete. This chapter gives an overview of the most important processes that determine transport and fate of persistent organic pollutants.

The past decades have brought substantial new knowledge about the environmental fate of different types of substances (SETAC, 1996). This has shown that simple physical and chemical characteristics of the substances can be useful to predict its distribution among environmental compartments and between water, soil, sediments, air, and organisms (Mackay *et al.*, 1992; Meylan *et al.*, 1993). Even for those substances where physical and chemical data are not yet available, models have been developed and used to predict these characteristics (Meylan and Howard, 1991; 1993, Meylan *et al.*, 1992; Boethling *et al.*, 1994). In addition, the availability of information on the sources and ambient concentrations of persistent organic pollutants is rapidly increasing. Taken together, these data and models have allowed an understanding of the environmental fate and transport of a large group of persistent organic pollutants.

3.2 PHYSICAL AND CHEMICAL PROPERTIES THAT DETERMINE ENVIRONMENTAL FATE

Substances possess physical and chemical properties which determine their transport pathways and distribution in the environment. The physical properties of greatest importance are water solubility, vapour pressure (P), Henry's law constant (H), octanol-water partition coefficient (K_{ow}), and the organic carbon-water partition coefficient (K_{oc}). Some of these properties are interrelated. For example, Henry's law constant can be calculated from [vapour pressure/water solubility] and K_{oc} is correlated to K_{ow}. Environmental distributions can be estimated. Using relatively simple models such as those of Mackay et al., (1992; 1993), the environmental distribution of persistent organic pollutants can be estimated from P, H, K_{oc} and K_{ow}, which determine partitioning among air, water, soil, sediment and biota. Biota include soil organic carbon, plant waxes, and lipids in organisms. Persistence in the environment is the other important property of a substance since transport can extend the range of exposure to such substances far beyond the immediate area of use and/or release.

3.2.1 Environmental influences on persistence and transport

Environmental transformations of persistent organic pollutants can be subdivided into three processes: biotransformation; abiotic oxidation and hydrolysis; and photolysis. The relative importance of these processes depends on the rates at which they occur under natural environmental conditions. These rates are, in turn, depend on the chemical structure and properties of the substance and its distribution in the various compartments of the environment. Factors that affect these rates have been extensively reviewed (SETAC, 1996).

Factors controlling rates of biodegradation have been reviewed by Battersby (1990) and Banerjee *et al.* (1984). In the environment, where growth of microorganisms is dependent on the availability of substrates and concentrations of persistent organic pollutants are low compared to other potential growth substrates, the biodegradation rate is dependent on both substrate concentration and biomass of microorganisms (Baughman *et al.*, 1980; Paris *et al.*, 1981). Factors influencing microbiological biomass are correlated with effects on biodegradation rates.

A number of environmental factors can alter hydrolysis rates. These include; temperature, pH, ionic strength, the presence of metal ion catalyses and, the presence of sediments. If the processes that control rate are catalysed by acid or base, pH will have a strong effect on halflife. Many persistent organic pollutants are halogenated (mainly chlorinated) and the C-Cl bond in chlorinated aromatics is not readily hydrolysed. As a result, hydrolysis is a relatively unimportant process for these substances.

Photodegradation of persistent organic pollutants is a potentially important pathway for degradation. Photodegradation in the atmosphere is relatively unimportant process because of the nature of the persistent organic pollutants. Photodegradation on particulate surfaces is highly variable and is dependent on the surface type and the wavelength and intensity of light (SETAC, 1996). For example, Koester and Hites (1992b) found large differences between the rates of photolysis of PCDDs and PCDFs adsorbed on silica gel and fly ash. Half-lives of these substances adsorbed on silica gel and irradiated under laboratory conditions ranged from 3-14 h for PCDFs to 88000 h for PCDDs. Loss of PCDDs and PCDFs was found to be negligible on fly ash after 200 h. By comparison, the half-life of 2,3,7,8-TCDD adsorbed to the surface of vegetation was 44 h in natural sunlight (McCrady and Maggard, 1993).

As would be expected, environmental factors have little effect on the breakdown and transformation of persistent organic pollutants. In addition, those that might have some effect are less effective in polar regions. Given the continued use and release of persistent organic pollutants in other parts of the globe, the result of this is a net accumulation of persistent organic pollutants in the polar regions (Figure 2).

3.2.2 Influence of environment on movement

Some of the above properties of persistent organic pollutants are strongly dependent on environmental conditions (SETAC, 1996). For example, temperature strongly affects vapour pressure, water solubility, and, therefore, Henrys law constant. The effect of temperature on the partitioning of substances is well known. For example, the direction and magnitude of air-water gas exchange for polychlorinated biphenyls (PCBs) and hexachlorobenzene (HCB) in the Great Lakes changes seasonally with temperature (Hornbuckle *et al.*, 1994; McConnell *et al.*, 1993). Greater volatilization occurs in summer as a result of warming of the surface water. The net exchange direction for substances in the open ocean also reflects differences in surface water temperature and atmospheric concentration. For example, net movement of persistent organic pollutants in the warm waters Bay of Bengal in the Indian Ocean is from the ocean to the atmosphere (Iwata *et al.*, 1993;

Jantunen and Bidleman, 1995) while that in cooler polar regions is the reverse (Bidleman *et al.,* 1995).

3.2.3 Environmental effects on deposition

Temperature may also affect deposition in locations away from the source. The distribution of PCBs, organochlorine pesticides, PCDDs and PCDFs is inversely related to vapour pressure, and thus to temperature. Lower temperatures favour greater partitioning of these compounds from the vapour phase to particles suspended in the atmosphere. This increases the likelihood of their removal and transport to the surface of the earth by rain and snow (Falconer and Bidleman, 1994; Koester and Hites, 1992a).

3.3 TRANSPORT

Countries in the tropics experience higher year-round temperatures than countries in the temperate and polar regions of the globe. Use of some pesticides in tropical agricultural production during the warmer, welter growing season may facilitate the rapid dissipation of persistent organic pollutants through air and water. For example, in the Vellar River and its watershed in South India, the flux of hexachloro-cyclohexane (HCH or BHC) residues into the atmosphere was estimated at about 99.6% of the applied HCH in the rite-growing paddy areas of this watershed. Only about 0.4% was transported by welter to the estuary over the year and about 75 % of the water-borne flux to the estuary was estimated to be lost by volatilization to the air. Thus only about 0.1 % of the applied HCH was estimated to ultimately be drained to the sea via the water in the Vellar river (Takeoka et al., 1991). Similar observations have been made by other workers (Tanabe et al., 1991).

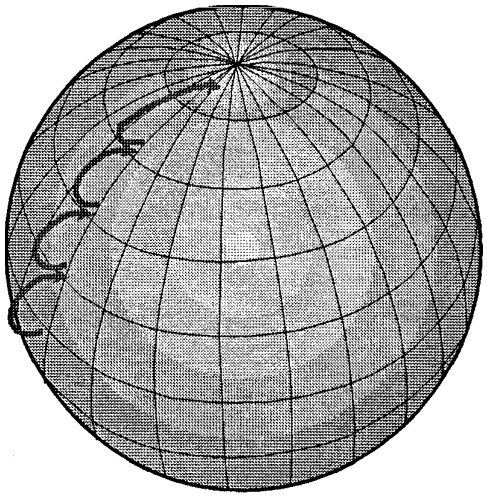


Figure 2 Net global deposition of persistent organic pollutants. It is recognized that POPs may originate throughout the latitudes where they are used.

These and other observations suggest that inputs of persistent organic pollutants to tropical oceans through discharge of river water are less significant than in temperate zones. In addition, the residence time in the tropical aquatic environment is quite short and transfer to the atmosphere is greater in these areas. The relatively short residence time of persistent organic pollutants in the tropical water bodies might be viewed as favourable for local organisms and environments, however, it does have more far-reaching implications for the global environment because these volatilized residues then disperse through the global atmosphere to deposit elsewhere.

Several monitoring studies have confirmed this. In a global monitoring survey of air and surface seawater from 1989-1990, Iwata et al. (1993) found HCH to be in the greatest concentration among the persistent organic pollutants. Concentrations were greatest in the Northern hemisphere. Concentrations were greater in the tropical source regions and in the cold welter deposition areas near the Arctic. On the other hand, DDT concentrations were higher only in the seas around tropical Asia. Other persistent organic pollutants such as PCBs and chlordanes showed a more uniform global distribution.

The present-day distribution of persistent organic pollutants in the oceans is indicative of a major change in distribution pattern during the last decades (SETAC, 1996). Until the early 1980s, there were higher concentrations of persistent organic pollutants (HCHs, DDT, and PCBs) in the mid-latitude oceans of the northern hemisphere, probably reflecting the large usage in developed countries such as Japan, Europe, and North America (Tanabe *et al.*, 1982, 1983; Tatsukawa and Tanabe, 1990). This distribution has not been seen in die most recent samples, an observation that is consistent with the changing use patterns of these substances (Goldberg, 1975). Other persistent organic pollutants have also been observed in higher concentrations in polar environments. PCBs

and chlordanes have also been detected in samples from the arctic (Kawano *et al.,* 1988; Muir *et al.,* 1992; Thomas *et al.,* 1992). The smaller geographical variations of PCB and chlordane concentrations in open ocean samples may be the result of their global distribution and use (SETAC, 1996). These findings support the assumption that a large proportion of persistent organic pollutants used in the tropics are released into the atmosphere and disperse through long-range global transport, most often to the polar regions.

3.4 DEPOSITION

Atmospheric transport and accumulation of persistent organic pollutants (PCBs, DDT, HCHs, and chlordanes) in the Arctic has been extensively documented (Cotham and Bidleman, 1991; Barrie *et al.,* 1992; Muir *et al.,* 1992; Lockhart *et al.,* 1992; Thomas *et al.,* 1992; Iwata *et al.,* 1993). Analyses of recent air samples from Antarctica also show continued transport of DDT, chlordanes, and HCHs to the southern polar regions (Larsson *et al.,* 1993; Bidleman, 1992). Accumulation in polar regions is partly the result of global distillation followed by cold condensation of compounds within the volatility range of PCBs and pesticides (Wania and Mackay, 1993; Mackay and Wania, 1995). It appears that, as these contaminants travel from tropical regions to the poles, they are continually deposited and re-evaporated and fractionate according to their volatilities (Figure 3). The final result is relatively rapid transport and deposition of persistent organic pollutants having intermediate volatility, such as HCB, and slower migration of less volatile substances such as DDT. The characteristics of polar ecosystems intensify the problems of contamination with persistent organic pollutants. The colder climate, reduced biological activity, and relatively small incidence of sunlight would be expected to increase the persistence of these substances. Poor vertical mixing of the surface layer of the Arctic Ocean may increase the availability of

organochlorines to the food chain and, during snowmelt, the sudden release of trapped persistent organic pollutants may occur, coinciding with the more active summer phase of the life cycle of polar organisms. The significant concentrations of persistent organic pollutants in arctic fish (Lockhart *et al.,* 1992), terrestrial (Thomas *et al.,* 1988) and aquatic mammals (Muir *et al.,* 1992) underlines the significance of long-range atmospheric transport from equatorial regions in the exposure of organisms from polar regions to persistent organic pollutants.

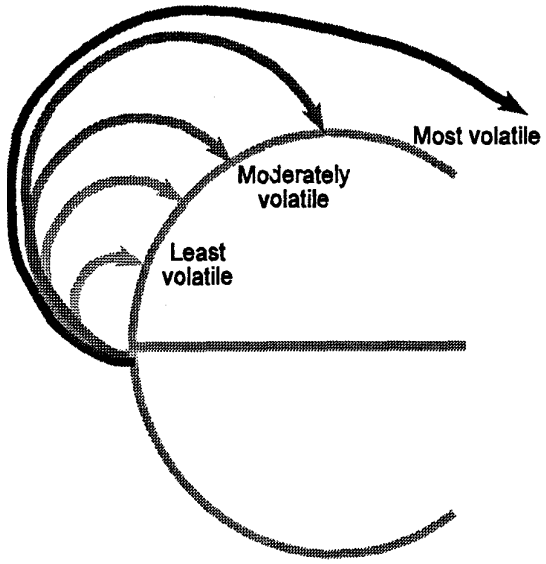


Figure 3 The effect of volatility on transportation distance of POPs. It is recognized that POPs may originate throughout the latitudes where they are used.

3.5 CONCENTRATIONS IN RELATION TO DEPOSITION

Considerable data on concentrations of persistent organic pollutants in samples from the Arctic are available and are summarized below. Most of these data are published in summary form as means or means with ranges. It was not possible to access the raw data from which these means were calculated, however, typical mean concentrations are presented in Tables 3.5-1 to 3.5-8 for information. Noteworthy is that inspection of this data showed indications of declines in concentrations since some of these persistent organic pollutants were banned or restricted.

The data in the literature are presented in several ways without standardization. The maintenance of a central database of all analytical data on the persistent organic pollutants would greatly aid in determining spatial and temporal trends in the data and linking these to changes in use pattern of these substances.

Table 3.5-1 Organochlorines in turbot liver from remote lakes and rivers in Canada. Concentrations are expressed as geometric means (ng/g of lipid) ± 95% confidence intervals, from Muir *et al. (1990).***

Locations	HCB	ΣChlordane	ΣDDT	Mirex	Toxaphene	Dieldrin	ΣPCB
Lake Winnepeg Man.	29 ± 4.5	142 ± 51	621 ± 218	10 ± 3	807 ± 285	41 ± 13	1941 ± 682
ELA Lake 625 Ont	22 ± 5	372 ± 125	1490 ± 601	14 ± 4	1723 ± 541	60 ± 9	1290 ± 386
Trout Lake Ont.	34 ± 8	377 ± 101	1029 ± 523	16 ± 7	2338 ± 769	70 ± 21	873 ± 467
S. Indian Lake Man.	66 ± 12	284 ± 67	461 ± 131	17 ± 4	1467 ± 323	34 ± 8	944 ± 281
Mackenzie R., Fort Simpson N.W.T.	34 ± 18	207 ± 105	162 ± 117	8 ± 2	1132 ± 683	14 ± 11	556 ± 389
Mackenzie R., Fort Good Hope N.W.T.	43 ± 22	172 ± 88	95 ± 57	7 ± 3	1570 ± 999	13 ± 6	343 ± 172
Mackenzie R., Arctic Red River N.W.T.	42 ± 37	229 ± 160	100 ± 67	5 ± 4	1700 ±1346	16 ± 11	301 ± 220
Peel River, Fort McPherson N.W.T.	23 ± 22	86 ± 79	50 ± 46	3 ± 3	930 ± 904	7 ± 6	344 ± 284

*Adapted from Lockhart *et al*., (1992).

Table 3.5-2 Organochlorine compounds detected in extracts of 200-litre samples of filtered river water (ng/L) and extracts of ground whole fish (µg/g wet weight) from five rivers in northern Ontario, June, 1981, from McCrea *et al.* (1984).*

Location	Species	HCB	Heptachlor	Heptachlor epoxide	α-Chlordane	*p,p'*-DDE-0.005	Dieldrin	ΣPCBs
Moose	Pike	ND-0.001	ND	ND	ND	ND-0.005	ND	0.02-0.05
	Sucker	ND	ND	ND-0.001	ND	ND-0.004	ND-0.002	0.02-0.21
	Water	0.0072	ND	ND	ND	ND	0.025	0.25
Albany	Pike	ND-0.002	ND	ND-0.001	ND	ND	ND	0.02-0.09
	Sucker	ND-0.002	ND	ND-0.001	ND	ND	ND	0.02-0.09
	Water	0.014	ND	ND	ND	ND	0.029	0.21
Attawapiskat	Pike	ND	ND	ND	ND	ND	ND	ND-0.02
	Sucker	ND-0.001	ND	ND-0.001	ND	ND-0.002	ND	ND-0.03
	Water	0.0037	ND	ND	ND	ND	ND	0.24
Winisk	Pike	ND-0.001	ND	ND	ND	0.003-0.007	ND	ND-0.03
	Sucker	ND-0.001	ND-0.001	ND	ND	ND-0.001	ND	ND-0.01
	Water	0.0088	ND	0.0012	ND	0.0050	ND	0.43
Severn	Pike	ND-0.002	ND	ND	ND-0.002	0.001-0.010	ND	ND-0.02
	Sucker	ND-0.001	ND	ND	ND	ND-0.001	ND	ND
	Water	0.003	ND	ND	0.0059	ND	ND	0.24

* Adapted from Lockhart *et al*., (1992).

Table 3.5-3 Organochlorine compounds (ng/g, wet weight) identified in muscle of lake whitefish from several communities in the Northwest Territories, 1986, from Lockhart *et al*. (1988, 1989).*					
Location	**ΣChlorobenzenes**	**ΣChlordanes**	**ΣDDT**	**Toxaphenes**	**Dieldrin**
Arctic Red River (N=4)	0.1-0.3	0.2-2.1	0.2-1.7	1.9-26.6	0.1-0.3
Fort Franklin (N=2)	0.8-1.8	5.7-9.8	2.8-3.5	50.5-59.9	0.5-13.3
Fort Good Hope (N=6[a])	0.1-1.4	0.2-4.8	0.2-3.6	6.7-85.8	0.1-0.6
Fort Simpson (N=2)	ND-0.9	1.0	0.4-0.8	3.0-10.0	0.2
Fisherman Lake (N=4)	0.1-0.4	0.4-2.7	0.4-1.3	6.9-11.6	0.1-0.3

*Adapted from Lockhart et al., (1992).
[a] Four lake whitefish, two broad whitefish

Table 3.5-4 Summary of DDT, PCBs and Dieldrin reported in freshwater fish from northern North America (ng/g, wet weight)*

Source of fish	Date	Species	No.	Tissue	ΣDDT	ΣPCB	Dieldrin	Reference
Hjalmar L., N.W.T	1970	Lake trout	≥3	HD[a]	3	-	Trace	1
Rutledge L., N.W.T.	1970	Lake trout	≥3	HD	20	-	Trace	1
Gordon L., N.W.T.	1970	Trout	≥3	HD	110	-	10	1
Kaminak L., N.W.T.	1970	Lake trout	≥3	HD	ND	-	ND	1
Kenai R., Alaska	1969	Lake trout	5	Whole	90	2640	-	2
Minto L., Que.	1970	Lake trout	4	Whole	45-150	41-91	-	3
Minto L., Que.	1970	Lake trout	1	H&F	597	640	-	3
Churchill Falls, Labrador	1977	Lake trout		Fat	-	370-690	-	4
Kenai R., Alaska	1969	Rainbow trout	5	Whole	140	5480	-	2
Chena R., Alaska	1970	Artic grayling	5	Whole	620	1420	10	2
Kenai R., Alaska	1969	Longnose sucker	5	Whole	30	1530	-	2
Chena R., Alaska	1969	Longnose sucker	5	Whole	1160	3870	10	2
Chena R., Alaska	1969	Roune whitefish	5	Whole	920	2620	10	2
Great Slave L., N.W.T.	1970	Lake whitefish	≥3	HD	10	-	10	1
Hjalmar L., N.W.T.	1970	Lake whitefish	≥3	HD	30	-	10	1
Nonacho L., N.W.T.	1970	Lake whitefish	≥3	HD	Trace	-	Trace	1
Rutledge L., N.W.T.	1970	Lake whitefish	≥3	HD	20	-	10	1
Merkley L., N.W.T.	1970	Lake whitefish	≥3	HD	Trace	-	10	1
Gymer L., N.W.T.	1970	Lake whitefish	≥3	HD	20	-	10	1
Gordon L., N.W.T.	1970	Lake whitefish	≥3	HD	190	-	30	1
Mackay L., N.W.T.	1970	Lake whitefish	≥3	HD	160	-	20	1
Baker L., N.W.T.	1970	Lake whitefish	≥3	HD	ND	-	ND	1
Jackson L., N.W.T.	1970	Lake whitefish	≥3	HD	10	-	Trace	1
Hay R., N.W.T.	1984	Lake whitefish	15	Muscle	3-30	1-3	1	5
Tuktoyaktuk, N.W.T.	1984	Broad whitefish	2	Muscle	0.4	1.9	-	6
Tuktoyaktuk, N.W.T.	1984	Unidentified	1	Muscle	2.5	3.5	-	6
Minto L., Que.	1970	Arctic char[b]	1	Gonads	108	130	-	3
Minto L., Que.	1970	Arctic char	1	Liver	47	31	-	3
S. Baffin Island	1986	Arctic char	Pooled	Liver	14	205	8	7
S. Baffin Island	1986	Arctic char	Pooled	Muscle	2	55	1	7

* Adapted from Lockhart *et al.*, (1992)

[a] HD, headless dressed; H & F, head and foreparts. [b]Landlocked.

References: 1 Reinke *et a*l. (1972); 2, Henderson *et al.* (1971); 3, Risebrough and Berger (1971); 4, Musial *et al*. (1979); 5, Wong (1985); Muir *et al.* (1986b); 7, Thomas and Hamilton, unpublished.

Table 3.5-5 Concentrations of selected organochlorine residues in terrestrial animals of the Canadian North.*										
Species	**Sex**	**Capture Location**	**Tissue**	**ΣDDT[a]**	**ΣChlordane[b]**	**ΣPCB[c]**	**Toxaphene**	**Mirex**	**Dieldrin**	**HCB**
Data from Thomas and Hamilton (1988) (ng/g wet tissue)										
Caribou *(Rangifer tarandus)*	F	Pond Inlet	Fat	2	5	11	NM	0.2	0.8	30
	M	Lake Harbour	Fat	3	5	25	NM	0.1	1.5	47
	M	Iqualit	Fat	2	2	52	NM	0.3	0.9	84
	M	Iqualit	Liver	1	8	8	NM	0.7	2.0	1
	M	Iqualit	Muscle	2	0.2	2	NM	0.1	0.05	2
	F	Arctic Bay	Fat	2	5	12	NM	0.08	0.8	57
	F	Clyde River	Fat	1	3	23	NM	0.4	0.7	57
Arctic Hare *(Lepus arcticus)*	F	Arctic Bay	Fat	0.9	2	3	NM	0.1	0.9	11
	F	Arctic Bay	Liver	<0.4	0.6	1	NM	0.03	0.4	0.4
	F	Arctic Bay	Muscle	<0.2	4	0.4	NM	0.01	0.003	0.2
Ptarmigan *(Lagopus mutus)*	M	Broughton Island	Muscle	0.3	0.8	4	NM	<0.02	0.13	0.3
	F	Arctic Bay	Liver	1.6	0.4	0.2	NM	0.7	0.15	0.4
	F	Arctic Bay	Muscle	0.3	0.2	0.6	NM	0.01	0.07	0.3
	F	Lake Harbour	Fat	11	7.4	12	NM	0.02	2.2	5.8
Data from Muir et al. (1988) (ng/g wet tissue)										
Caribou *(Rangifer tarandus)*	-	Broughton Island	Fat	4	5	33	13	<0.1	1.7	25
	-	Broughton Island	Muscle	1	2	10	3	0.1	0.4	1.2
Ptarmigan *(Lagopus nutus)*	-	Broughton Island	Muscle	0.5	1.0	18	3	NM	0.1	0.3
Data adapted from Peakall et al. (1990) ng/g wet weight)[d]										
Peregrine falcon *(Falco peregrinus* [anatum])	-	NWT & Yukon 1966-72	Eggs	12600	160[e]	NM	NM	NM	690	16
	-	1973-79		11900	230[e]	8800[f]	NM	320	670	48
	-	1980-87		12500	500[e]	5600[f]	NM	280	340	47
Peregrine falcon *(Falco peregrinus [tundrius]*	-	NWT & Ungava 1966-72	Eggs	6400	120[e]	NM	NM	NM	40	120
	-	1973-79		12400	350[e]	12800[f]	NM	40	690	330
	-	1980-87		7000	560[e]	9800[f]	NM	200	570	390

Table 3.5-5 Concentrations of selected organochlorine residues in terrestrial animals of the Canadian North.*										
Species	**Sex**	**Capture Location**	**Tissue**	**ΣDDT[a]**	**ΣChlordane[b]**	**ΣPCB[c]**	**Toxaphene**	**Mirex**	**Dieldrin**	**HCB**
Data adapted from Noble and Elliot (1990) (ng/g wet tissue)[d]										
Gyrfalcon *(Falco rusticolus)*	-	NWT & Yukon 1965-72	Eggs	9100	60[g]	NM	NM	NM	170	20
	-	1973-79		330	20[g]	600[f]	NM	ND	16	14
	-	1980-87		120	30[g]	470[f]	NM	ND	20	70
Data from the National Registry of Toxic Chemical Residue Database, Canadian Wildlife Service, Hull, Quebec (ng/g wet weight)										
Caribou *(Rangifer tarandas)*	M	Prince of Wales Island 1978	Liver	<0.05	1.6	<0.15[j]	NM	<0.05	<0.05	0.93
Musk-ox *(Ovibos moschatus)*	M	Sachs Harbour 1985	Liver	<0.05	2.5	0..26[j]	NM	<0.05	<0.05	3.88
	F	Sachs Harbour 1985	Liver	<0.05	2.15	0.21[j]	NM	<0.05	<0.05	1.83

*Adapted from Thomas *et al.*, (1992)
NM, not measured.
[a]ΣDDT= Sum of *p,p'*-DDT and *p,p'*-DDE and *p,p'*-DDD
[b] ΣChlordane = Sum of *cis*, *trans*-chlordane, oxychlordane, heptachlor, heptachlor epoxide and *cis*, trans-nonachlor.
[c] ΣPCB = Sum of individual congeners.
[e] Concentrations shown are geometric means. Not detected was counted as part of the data set at half the detection limit and included in the mean.
[e] Sum of heptachlor epoxide and oxychlordane.
[f] As Aroclor 1254:1260 (1:1).
[g] Heptachlor epoxide only.
[h] Sum of congeners 138 and 180; other congeners not detected at <0.07 ng/g.

Table 3.5-6 Organochlorine compounds (ng/g wet weight; mean ± SD) in Arctic vertebrates, fish, marine mammals and seabirds.*										
Species	**Location**	**Year**	**Tissue**	**N**	**Sex[a]**	**ΣDDT**	**PCB[b]**	**ΣChlor**	**PCC[b]**	**Reference**
Invertebrates										
Zooplankton	Ice Island	1986	Lipid	Pool	-	6	13	10	24	Bidelman *et al., 1989*
	81°97°W	1987	Lipid	Pool	-	620	110	51	160	
Amphipods	Ice Island	1986	Lipid	Pool	-	640	1600	900	1500	
	81°97°W	1987	Lipid	Pool	-	3460	14000	2520	8300	
Fish										
Arctic cod *(Boreogadus saida)*	Arctic Bay	1984	Muscle	Pool	-	2.6	1.9[c]	2	23	Muir *et al*., 1987
	Resolute Bay	1984	Muscle	Pool	-	4.7	2.4[c]	3.1	14	
	Pnagnirtung	1984	Muscle	Pool	-	4.5	6.3[c]	3.4	46	
Greenland cod *(Gadus ogac)*	Frobisher Bay	1984	Whole	2	-	3.2	2.8[c]	1.0	6	
	Victoria Is.	1984	Muscle	Pool	-	3.7	1.8c	1.1	<2	
	Queen Maud Gulf	1984	Muscle	2	-	-	3	-	-	EPS, 1985
Atlantic cod *(Gadus morhua)*	N. Finland Vester-Tana	1985-89	Lipid	?	-	147	570	128	540	Paasivirta & Rantio, 1991
Pacific herring *(Clupea harengus P.)*	Tuktoyaktuk harbor	1984	Muscle	Pool	-	4.1	5[c]	5.6	74	Muir *et al*., 1987
Inconnu *(Stenodus leucichtys)*	Tuktoyaktuk harbor	1984	Muscle	1	-	3.5	2.5[c]	3.3	24	
Arctic char *(Salvelinus alpinus)*	Baffin Is.	1972	Liver	3	-	15±16	10±10	-	-	Bowes & Jonkel, 1975
	Prince Patric Is.	1972	Liver	4	M&F	4.3±3	5±4	-	-	
			Muscle	3	M&F	8±5	8±10	-	-	
	W. Davis strait	1985	Whole	6	-	9±3	45±14[c]	26±7	157±67	Muir *et al., 1986*
	Cumberland Sound	1986	Whole	10	-	4±1	10±1[c]	15±3	95±18	Hendzel & Reiger (unpublished)
	West Hudson Bay	1985	Whole	10	-	9±3	14±3[c]	19±6	79±23	
	Queen Maud Gulf	1985	Whole	10	-	21±6	16±4[c]	44±9	153±44	
	S. Beaufort Sea	1985	Whole	9	-	4±1	4±1[c]	12±4	44±14	
	N. Baffin Is.	1986	Fat	1	-	4.5	64[c]	32	-	Thomas & Hamilton, 1988
			Muscle	1	-	9	9[c8]	-		
	W. Baffin Bay	1986	Fat	1	-	2.6	2[c]	2	-	
			Muscle	1	-	0.6	1[c]	0.6	-	
	Queen Maud Gulf	1984	Muscle	4	F	-	35±24	-	-	EPS, 1985
Atlantic salmon *(Salmo salar)*	W. Greenland	1979	Whole	2	-	36	75.3	15	300	Anderson *et al., 1988*
	Labrador Sea	1979	Whole	2	-	10	35[c]	4	84	
	N Finland, Tana R.	1985-89	Fish Lipid	?	-	516	572	139	2870	Paasivirta & Rantinoe, 1991

Table 3.5-6 Organochlorine compounds (ng/g wet weight; mean ± SD) in Arctic vertebrates, fish, marine mammals and seabirds.* (Continued)										
Species	**Location**	**Year**	**Tissue**	**N**	**Sex[a]**	**ΣDDT**	**PCB[b]**	**ΣChlor**	**PCC[b]**	**Reference**
Cetaceans										
Beluga *(Delphinapterus leucas)*	S Beaufort Sea (MacKenzie delta)	1972	Liver	7	Adult	780±230	<500	-	-	Addison & Brodie, 1973
			Blubber	7	Adult	3900±890	<500	-	-	
	S. Beaufort Sea	1972	Blubber	7	Adult	2560±1460	<500	-	-	
	S. Beaufort Sea	1983-87	Blubber	10	M	2200±830	3330±850[c]	1750±410	3830±1160	Muir *et al., 1990a*
			Blubber	2	F	670	1230[c]	670	1380	
	Jones Sound	1984	Blubber	8	M	1960±320	2530±570[c]	1870±440	4250±1020	
			Blubber	7	F	2190±1690	2460±1980[c]	1840±1130	3740±2120	
	W. Hudson Bay	1986	Blubber	4	M	3130±200	3120±340[c]	2330±260	5100±420	
			Blubber	4	F	850±960	960±1000[c]	850±800	1770±1410	
	E. Hudson Bay	1986	Blubber	8	M	2270±680	2770±510[c]	1860±350	4130±820	
			Blubber	8	F	980±730	1230±840[c]	870±580	1990±1100	
	Cumberland Sound	1983	Blubber	6	M	6830±1890	4910±250[c]	2380±400	5780±5390	
			Blubber	6	F	930±550	1150±410[c]	620±150	1770±1760	
Narwhal *(Monodon monoceros)*	W. Baffin Bay	1979	Blubber	9	F	1980±2010	6730±2220[d-]	-	-	Wagemann & Muir, 1984
			Blubber	11	M	4840±2130	12850±6880[d]	-	-	
	W. Baffin Bay	1982-1983	Blubber	15	M	5920±1710	5180±1920[c]	1920±410	9160±2350	Muir *et al., 1992a*
			Blubber	6	F	2540±2020	2700±1790[c]	1400±1020	2440±2840	
Minke whale *(Balaenoptera acutorostrata)*	W Greenland	1972	Blubber	6	-	1400±940	610±380	-	-	Johansen *et al., 1980*
Fin whale *(Balaenoptera physalus)*	S.E. Greenland	1975	Blubber	3	-	2830	3600			Holden, 1975
Porpoise *(Phocaena phocaena)*	W: Greenland	1972	Blubber	2	-	320±390	6700±6700	30		Clausen *et al., 1974*
Pinnipeds										
Ringed seal *(Phoca hispida)*	Arctic Canada	1970	Blubber	3	Adult	2700±1500	3000±1200	-	-	Holden, 1975
	Amundsen Gulf (Holman Is.)	1972	Blubber	15	M	1310±310	4100±1400	-	-	Addison & Smith, 1974
			Blubber	13	F	610±270	2000±900	-	-	
	Amundsen Gulf (Holman Is.)	1972	Blubber	12	M	1100±510	3690±1340	-	-	Addison *et al., 1986*
			Blubber	9	F	550±210	1830±820	-	-	
		1981	Blubber	16	M	780±560	1280±750	-	-	
			Blubber	15	F	330±140	580±250	-	-	
	W. Greenland	1972	Blubber	5	-	150±100	900±270	-	-	Clausen *et al.,1974*
	E. Beaufort Sea (Sachs Harbour)	1972	Liver	3	-	22±13	40±60	-	-	Bowes &Jonkel, 1975
			Blubber	5	-	1538±876	920±770	-	-	

Table 3.5-6 Organochlorine compounds (ng/g wet weight; mean ± SD) in Arctic vertebrates, fish, marine mammals and seabirds.* (Continued)										
Species	**Location**	**Year**	**Tissue**	**N**	**Sex[a]**	**ΣDDT**	**PCB[b]**	**ΣChlor**	**PCC[b]**	**Reference**
***Pinnipeds**, continued*										
Ringed seal *(Phoca hispida)*, continued	Jones Sound	1972	Liver	3	F	78±89	40±40	-	-	
			Blubber	2	-	367±266	500±490	-	-	
	Spitzbergen	1980	Blubber	1	-	4500	9000	-	4000	Anderson *et al*, 1988
	Barrowstrait	1984	Blubber	19	M	710±400	570±290[c]	460±210	-	Muir *et al.*, 1988
			Blubber	14	F	480±280	380±1700 [c]	350±180	-	
			Liver	19	M	9±7	6±4[x]	6±5	-	
			Liver	14	F	6±5	4±3[x]	5±3	-	
	Admiralty Inlet	1983	Blubber	10	M	1330±1500	790±880[c]	460±310	-	
			Blubber	16	F	480±320	310±140[c]	280±140	-	
			Liver	8	M	6±7	5±5[c]	4±3	-	
			Liver	7	F	2±3	1±2[c]	2±3	-	
	Various Baffin Is. Locations	1986	Blubber	5	M	720±340	680±340[c]	160±70	-	Thomas & Hamilton, 1988
			Blubber	5	F	370±320	490±170[c]	130±100		
			Liver	2	M	9	23 [c]	7	-	
			Liver	4	F	10±8	24±15 [c]	7±4		
	Spitzbergen	1986	Blubber	7	M&F	1620±1020	840±480[c]	26±9	-	Oehme *et al*., 1988
	E. Beaufort Sea	1988	Blubber	11	M	380±160	580±230 [c]	420±150	190±80	Muir *et al*., 1992b
			Blubber	10	F	360±360	520±240 [c]	340±130	130±50	
	S. Beaufort Sea	1986	Blubber	7	M&F	290±140	630±300 [c]	410±260	340±480	
	Queen Maude Gulf	1986	Blubber	10	M	470±380	880±600 [c]	560±330	390±140	
			Blubber	6	F	300±230	670±550 [c]	490±410	310±210	
	Spence Bay	1986	Blubber	9	M	240±230	560±500 [c]	670±1220	290±170	
			Blubber	5	F	230±140	510±390 [c]	400±310	310±240	
	W. Hudson Bay (Rankin Inlet)	1986	Blubber	10	M	690±530	880±650 [c]	520±270	470±350	
			Blubber	10	F	450±290	570±310 [c]	390±200	290±140	
	N. Hudson Bay (Coral Harbour)	1987	Blubber	10	M	86±76	1160±800 [c]	710±490	480±270	
			Blubber	6	F	31±8	530±220 [c]	320±60	310±200	
	W. Davis Straight (Broughton Is.)	1985	Blubber	8	M	410±190	540±210 [c]	330±90	250±160	
			Blubber	9	F	410±200	510±200 [c]	290±140	440±330	
	Cumberland Sound	1986	Blubber	10	M	330±190	510±230 [c]	300±110	380±160	
			Blubber	8	F	240±70	350±70 [c]	250±100	180±100	
	Chukchi Sea	1988	Blubber	2	M	210	720 [c]	70[f]	-	Becker et al., 1989

Table 3.5-6 Organochlorine compounds (ng/g wet weight; mean ± SD) in Arctic vertebrates, fish, marine mammals and seabirds.* (Continued)										
Species	**Location**	**Year**	**Tissue**	**N**	**Sex[a]**	**ΣDDT**	**PCB[b]**	**ΣChlor**	**PCC[b]**	**Reference**
Pinnipeds, continued										
Bearded seal *(Erignathus barbatus)*	W. Greenland	1972	Blubber	5	-	470±260	1800±1000	48[g]	-	Clausen et al., 1974
	Baffin Is.	1986	Blubber	2	F	980	630[c]	150	-	Thomas & Hamilton, 1988
			Muscle	1	F	20	10[c]	5	-	
Hooded Seal *(Cystophora cristata)*	W. Greenland	1974	Blubber	4	-	3500±1500	3900±2000	-	-	Johansen *et al., 1980*
Harp seal *(Phoca groenlandica)*	W. Greenland	1972	Blubber	5	-	290±100	2740±1830	-	-	Clausen *et al.*,1974
	W. Greenland	1972	Blubber	8	-	4900±5600	1900±1100	-	-	Johansen *et al., 1980*
		1974	Blubber	3	-	1500±600	1700±500	-	-	
		1976	Blubber	3	-	2800±1400	1600±700	-	-	
	N. Baffin Bay & Cumberland Sound	1976-78	Blubber	4	F/pups	810±1030	1090±830	70±30	-	Ronald et al., 1984
			Blubber	10	F/juv.	980±480	1440±780	270±70	-	
			Blubber	6	F/adult	1120±990	1360±790	170±120		
			Blubber	2	M/pup	1270±700	1160±490	210±30	-	
			Blubber	11	M/juv.	1640±960	1700±1290	320±130	-	
			Blubber	9	M/adult	090±2130	3760±3650	230±100		
	N.W. Greenland	1976-78	Liver	1	M&F	30	-	-	-	
			Muscle	2	M&F	130	-	-	-	
Fur seal *(Callorhinus ursinus)*	Bering Sea (Pribilof Is.)	1969	Liver	5	-/pups	2210	-	-	-	Anas & Wilson, 1970a
			Blubber	5	-/pups	15910	-	-	-	
	Bering Sea (Pribilof Is.)	1968	Liver	13	M	800	-	-	-	Anas & Wilson, 1970b
			Liver	10	F	980	-	-	-	
	Bering Sea (St. Paul Is.)	1972	Blubber	5	-/pups	39900± 48200	12900±33500	-	-	Kurtz & Kim 1976
			Blubber	2	F/adult	5200±2600	5800±1500	-	-	
	Bering Sea (St. Paul Is.)	1975	Blubber	6	M	5210±1260	3680±1090	-	-	Kurtz 1984
		1978	Blubber	6	M	5470±2210	3190±1250	-	-	
		1981	Blubber	6	M	4640±1880	3210±1260	-	-	
	Bering Sea (St. Paul Is.)	1984	Blubber	29	M	2780±830	1780±630	-	-	Kurtz , 1987
			Liver	29	M	120±50	100±40	-	-	
	Bering Sea (St. Paul Is.)	1978-80	Blubber	2	M	7520[h]	2490	-	-	Calambokidis & Peard, 1985
	Bering Sea (St. Paul Is.)	1987	Blubber	2	M	1290	440	210[f]	-	Becker *et al.*, 1989
Walrus *(Odobenus rosmarus rosmarus)*	N.W. Greenland (Thule)	1975-76	Blubber	8	M	90±130	360±310	-	-	Born *et al.*, 1981
			Blubber	20	F	50±50	180±120	-	-	

Table 3.5-6 Organochlorine compounds (ng/g wet weight; mean ± SD) in Arctic vertebrates, fish, marine mammals and seabirds.* (Continued)										
Species	**Location**	**Year**	**Tissue**	**N**	**Sex[a]**	**ΣDDT**	**PCB[b]**	**ΣChlor**	**PCC[b]**	**Reference**
Walrus *(Odobenus rosmarus rosmarus)*	N. Baffin Is.	1986	Blubber	1	M	36	131	64	-	Thomas & Hamilton, 1988
Walrus *(Odobenus rosmarus divergens)*	N. Bering Sea	1972	Blubber	4	-	80	1800	-	-	Galster & Burns, 1972
	N. Bering Sea	1981-84	Blubber	53	M&F	<100	<100	40±80[i]	<100	Taylor *et al.*,, 1989
Polar Bears										
Polar bear *(Ursus maritimus)*	Beaufort Sea	1982	Fat	6	Pool	140	4250[c]	2310	-	Norstrom *et al.*, 1988
	Amundsen Gulf	1982	Fat	8	Pool	180	3950	1810	-	
	Melville Is.	1982	Fat	8	Pool	280	8250	3420	-	
	Hadley Bay	1982	Fat	6	Pool	120	6630	3810	-	
	M'Clintock Chan'l	1982	Fat	16	Pool	220	4430	3680	-	
	Cornwallis Is.	1982	Fat	18	Pool	300	5940	3720	-	
	N. Baffin Is.	1984	Fat	10	Pool	210	4220	2860	-	
	W. Davis Strait	1984	Fat	10	Pool	390	3240	2750	-	
	St. Baffin Is.	1984	Fat	20	Pool	410	4250	2730	-	
	Southampton Is.	1983	Fat	10	Pool	940	8110	6890	-	
	W.Hudson Bay	1983	Fat	9	Pool	1190	8020	7090	-	Bowes & Jonkel, 1975
	S. Hudson Bay	1968-71	Fat	2	M&F	580	5750	-	-	
			Muscle	7	M&F	10±10	190±230	-	-	
	Southampton Is.		Muscle	7	M&F	10±10	80±70	-	-	
	Baffin Bay		Muscle	5	M&F	10±10	250±500	-	-	
	Cornwallis Is.	1972	Fat	3	F	150±40	2940±140	-	-	
	S. Beaufort Sea	1971	Muscle	4	M&F	5±4	230±120	-	-	
Sea birds										
Northern fulmar *(Fulmarus glacialis)*	Prince Leopold Is.	1975	Liver	10	Pool	250	980	150	-	Nettleship & Peakall, 1987
		1976	Liver	10	Pool	500	1790	200	-	
		1975	Egg	10	Pool	760	1930	130	-	
	Prince Leopold Is.	1987	Liver	8	Pool	140	560	230	-	Peakall, unpublished, 1989
			Egg	6	Pool	230	800	260	-	
	Davis Strait	1972	Liver	1	-	2100	4200	-	-	Bourne & Bogan, 1976
	Bjornoya	1972	Liver	1	-	300	1600	-	-	
	Spitzbergen	1980	Liver	10	-	630	1600	-	-	Norhelm & Kjos-Hansen, 1984
			Fat	10	-	220	59000	-	-	

Table 3.5-6 Organochlorine compounds (ng/g wet weight; mean ± SD) in Arctic vertebrates, fish, marine mammals and seabirds.* (Continued)										
Species	**Location**	**Year**	**Tissue**	**N**	**Sex[a]**	**ΣDDT**	**PCB[b]**	**ΣChlor**	**PCC[b]**	**Reference**
Northern fulmar, cont'd	N. Baffin Is.	1986	Fat	1	-	2010	2110	250	-	Thomas & Hamilton, 1988
			Muscle	1	-	53	325	51	-	
Ivory gull *(Phagophilia eburnea)*	Seymour Is.	1976	Egg	10	Pool	500	1630	84	-	Noble & Elliott, 1986
Black legged kittiwake *(Rissa tridactyla)*	Prince Leopole Is.	1975	Liver	10	Pool	50	1190	<100	-	Nettleship & Peakall, 1987
		1976	Liver	5		80	2420	<100		
		1976	Egg	10	Pool	380	5210	<100		
		1987	Egg	3	Pool	130	1610	110	-	Peakall, 1989 unpublished
	Davis Strait	1972	Liver	1		130	3200	-	-	Bourne & Bogan, 1976
	Bjornoya	1972	Liver	1		80	1600	-	-	
	N. Bering Sea	1973-76	Egg	9		33	365	-	-	Ohlendorf *et al., 1982*
Thick-billed murre *(Uria lomvia)*	Davis Strait	1972	Liver	2		130	80	-	-	Bourne & Rogan, 1976
	Bjornoya	1972	Liver	2		100	200	-	-	
	Spitzbergen	1980	Liver	9		160	400	-	-	Norrheim & Kjos-Hanssen, 1984
	W. Greenland	1972	Fat	5		3500	12900	-	-	Braestrup *et al*., 1974
	N. Bering Sea	1973-76	Egg	10		166	307	-	-	Ohlendorf *et al., 1982*
	Prince Leopold Is.	1975	Liver	10		60	220	<100		Nettleship & Peakall, 1987
		1976	Liver	12		190	530	<100		
		1977	Liver	11		120	360	<100		
		1975	Egg	12		310	720	<100	_	
		1976	Egg	10	Pool	440	1010	<100		
		1977	Egg	10		390	910	<100	-	
	Lancaster Sound	1976	Fat	8	adult	10480±7540	1250±1340	-	-	Wong, 1985
		1987	Egg	10		180	540	190	-	Peakall, unpublished, 1989
		1987	Liver	10	Pool	210	680	40	-	
Black guillemot *(Cephyus grylle)*	Lancaster Sound	1976	Fat	5	adult	13150±7950	900±280	-	-	Wong, 1985
			Fat	5	juv.	400±160	<10	-	-	
	Spitzbergen	1980	Muscle	4	Pool	120	260	-	90	Andersson *et al.,1988*

Table 3.5-6 Organochlorine compounds (ng/g wet weight; mean ± SD) in Arctic vertebrates, fish, marine mammals and seabirds.* (Continued)										
Species	**Location**	**Year**	**Tissue**	**N**	**Sex[a]**	**ΣDDT**	**PCB[b]**	**ΣChlor**	**PCC[b]**	**Reference**
Oldsquaw *(Clangula hyemalis)*	W. Hudson Bay	1971	Carcass	10	M	6400±6900	25000±25000	-	-	Wong, 1985
			Carcass	10	F	6500±6200	18000±13000	-	-	
			Egg	11	-	7600±6100	48000±31000			
Eider duck *(Somaeria molissima)*	Broughton Is.	1985	Liver	6	-	3±2	24±14	7±2	10±14	Muir *et al., 1986a*
	Spitzbergen	1980	Muscle	5	Pool	27	72	-	18	Andersson *et al*., 1988

*Adapted from Muir *et al*., (1992)
[a]Dash indicates results not available or not determined
[b]Concentrations on a wet weight basis. ΣDDT = sum of 4,4'-DDE, -DDD and -DDT; ΣPCB = aroclor equivalent unless otherwise indicated; ΣChlor = sum of chlordane related isomers (nonachlors, chlordanes, oxychlordanes, etc.): PCC (Polychlorinated camphenes) = toxaphene
[c]PCBs as sum of individual congeners
[d]High PCBs (as Aroclor 1254 equivalents) probably due to interference from PCC peaks
[e]Only oxychlordane reported
[f]Sum of *trans*-nonachlor and heptachlor epoxide
[g]Only heptachlor epoxide reported
[h]Reported as 4,4'-DDE only
[i]Only oxychlordane detected.

Table 3.5-7 Chlorinated dioxins and dibenzofurans in Arctic marine biota (ng/g).*								
Species	**Location**	**N**	**2, 3, 7, 8-TCDD**	**1, 2, 3, 7, 8 - PnCDD**	**2, 3, 7, 8 - TCDF**	**1, 2, 3, 7,8-PnCDF**	**2, 3, 4, 7, 8-PnCDF**	**Reference**
Atlantic salmon	N. Finland Tana R.	?[a]	-	-	<0.0001	-		Muir & Ford, 1990
Ringed seal	S. Beaufort Sea	4[b]	0.004	<0.003	0.002	<0.003	<0.003	Norstrom et al., 1990
	Queen Maud Gulf	4[b]	0.012	<0.003	0.004	<0.003	<0.003	
	Larsen Sound	4[b]	0.015	<0.003	<0.003	<0.003	<0.003	
	Barrow Strait	10[b]	0.035	<0.003	0.004	<0.003	<0.003	
	Admiralty Inlet (N. Baffin Is.)	11[b]	0.037	<0.003	0.005	<0.003	<0.003	
	W. Davis Strait (Broughton Is.)	3[b]	0.011	<0.003	<0.003	<0.003	<0.003	
Ringed seal	N. Hudson Bay (Coral Harbour)	4[b]	0.003	<0.003	0.007	<0.003	<0.003	
	W. Hudson Bay (Rankin Inlet)	4[b]	0.002	<0.003	0.004	<0.003	<0.003	
	Baltic Sea (1986/87)	10	0.046	0.118	0.038	0.006	0.077	
	Barents Sea, 1987	5	0.012	0.016	0.012	<0.005	0.009	
	Spitzbergen	5	<0.007	0.007	0.009	<0.002	0.007	
	Spitzbergen	7	0.004	0.011	0.0013	0.007	0.011	
Harp seal	Barents Sea	3	<0.002	0.007	0.004	0.001	0.005	
Beluga whale	Cumberland Sound	5	<0.002	<0.003	<0.002	<0.003	<0.003	
Polar bear	Beaufort Sea	5L[c]	0.002	<0.003	<0.002	<0.003	<0.003	
	Amundsen Gulf	4L[c]	0.003	<0.003	<0.002	<0.003	<0.003	
	Hadley Bay	6F[c]	0.011	<0.003	<0.002	<0.003	<0.003	
	Melville Island	6F[c]	0.018	<0.003	<0.004	<0.003	<0.003	
	Larsen Sound	8F[c]	0.023	<0.003	<0.002	<0.003	<0.003	
	Barrow Strait	10F[c]	0.020	<0.003	<0.002	<0.003	<0.003	
	Pond Inlet	20F[c]	0.004	<0.003	<0.002	<0.003	<0.003	
	W. Baffin Bay (Clyde River)	10F[c]	0.005	<0.003	<0.002	<0.003	<0.003	
	W<. Davis Strait (Broughton Is.)	10F[c]	0.003	<0.003	<0.002	<0.003	<0.003	
	Cumberland Sound	10F[c]	<0.002	<0.003	<0.002	<0.003	<0.003	
	N. Hudson Bay (Coral Harbour)	10F[c]	0.002	<0.003	<0.002	<0.003	<0.003	
	W. Hudson Bay (Rankin Inlet)	10F[c]	0.002	<0.003	<0.002	<0.003	<0.003	

*Adapted from Muir *et al*., (1992)

[a] Mean results reported. All concentrations in this study reported on a lipid weight basis.

[b] Pooled examples of male ringed seal blubber.

[c] Pooled samples of Liver (L) or Fat (F), combined males and females.

Table 3.5-8 Organochlorines in surface seawater (ng/L), ice and suspended particles from the Arctic Ocean. *									
Sample type	**Date**	**N**	**HCB[a]**	**dieldrin[a]**	**ΣChlor[a]**	**ΣDDT[a]**	**ΣPCB[a]**	**PCC[a]**	**Reference**
Seawater									
Ice Island, 81°N, 97°W	May 1986	6	0.017± 0.006	0.015± 0.002	0.004± 0.002	<0.004-0.014	0.007± 0.005	0.360[b]	Hargrave et al., 1988; Bidelman et al., 1989
	Aug 1986	5	0.022 ±0.007	0.014± 0.003	0.005± 0.002	<0.004	>0.004-<0.016	0.032[c]	
Norwegian Sea, 73°30'N, 16°W	Aug 1985	1	<0.030	NA	NA	<0.00005	<0.0005	NA	Gaul 1992
Norwegian Sea, 77°N, 1°33'W	Aug 1985	1	<0.030	NA	NA	<0.00005	<0.0005	NA	
Ice									
Ice Island, 81°N, 97°W	May 1986	3	<0.002	0.006± 0.001	<0.002	<0.002-0.012	<0.023	NA	Hargrave et al., 1988; Bidelman et al., 1989
Norwegian Sea, 73°30'N, 16°W	Aug 1985	3	0.040± 0.020	NA	NA	0.250± 0.070	1.000± 0.100[d]	NA	Gaul 1992
Norwegian Sea, 77°N, 1°33'W	Aug 1985	3	0.050± 0.030	NA	NA	0.100	2.500+ 0.700[d]	NA	
Particles									
Ice Island, 81°N, 97°W	May 1986	6	<0.0001	<0.0003-0.003	<0.0002	<0.0003-0.001	<0.001-0.006	NA	B.T. Hargrave Unpublished 1988
	May 1986	4	<0.0003	<0.0003	<0.0004	<0.0009	<0.002	NA	

* Adapted from Muir *et al.*, (1992)

[a]ΣChlor = sum of *cis*- and *trans*-chlordane and *trans*-nonachlor; ΣDDT = sum of 4,4'-DDE, and -DDD and -DDT; ΣPCB = Aroclor 1254 equivalents except where noted; PCC (Polychlorinated camphenes) = toxaphene.

[b]Single sample from 10 m depth.

[c]Single sample from 270 m collected June 1987.

[d]Only congener 138 reported.

4. CHEMISTRY AND TOXICOLOGY

4.1 CHEMISTRY

Persistent organic pollutants are, by definition, organic compounds that are highly resistant to degradation by biological, photolytic or chemical means. Persistent organic pollutants are often halogenated and most often chlorinated. The carbon-chlorine bond is very stable towards hydrolysis and, the greater the number of chlorine substitutions and/or functional groups, the greater the resistance to biological and photolytic degradation (Kannan *et al.*,1988). Chlorine attached to an aromatic (benzene) ring is more stable to hydrolysis than chlorine in aliphatic structures. As a result, chlorinated persistent organic pollutants are typically ring structures with a chain or branched chain framework. By virtue of their high degree of halogenation, persistent organic pollutants have very low water solubility and high lipid solubility leading to their propensity to pass readily through the phospholipid structure of biological membranes and accumulate in fat deposits (Swain *et al.*, 1992). Zitko (1979) has suggested that bromobiphenyls with five or more substitutions will tend to accumulate to a lesser degree than chlorobiphenyls and that highly chlorinated biphenyl compounds tend to accumulate to a greater extent than the less chlorinated PCBs. Metabolism and disposition appear to be inversely related ,to the degree of chlorination with highly chlorinated biphenyls being far more resistant than the less chlorinated PCBs (Peterson and Gainey, 1979). .

Halogenated hydrocarbons ate a major group of persistent organic pollutants and of these, the organochlorines are by far the most important group. Included in this class of organohalogens are dioxins and furans, PCBs, hexachlorobenzene, Mirex, toxaphene, heptachlor, chlordane and DDT. These substances are characterized by their low water solubility and high lipid solubility, many persistent organic pollutants are noted for their environmental persistence, long half-lives and their potential to bioaccumulate and biomagnify in organisms once dispersed into the environment.

Although some natural sources of organochlorines are known to exist, most persistent organic pollutants originate almost entirely from anthropogenic sources associated largely with the manufacture, use and disposition of certain organic chemicals. In contrast, dioxins and furans are formed unintentionally in a wide range of manufacturing and combustion processes, from both anthropogenic and non-anthropogenic sources.

Persistent organic pollutants are typically semi-volatile compounds, a characteristic that favours the long-range transport of these chemicals (Barrie *et al.*, 1992). They can thus move over great distances utilizing the atmosphere as the primary transport medium. Volatilisation may occur from plant and soil surfaces following application of persistent organic pollutants used as pesticides (Scholtz *et al.*, 1993, Benjey, 1993). Compounds trapped in soil may remain available for eventual volatilisation almost indefinitely, or until otherwise chemically or microbially degraded. Such processes may be highly dependent on a number of meteorological characteristics (Scholtz and Voldner, 1992), including temperature dependence and vapour pressure.

Halogenated, and particularly chlorinated organic compounds have become entrenched in contemporary society, being utilized by the chemical industry in the production of a broad array of products ranging from polyvinylchloride (millions of tonnes per year) to solvents (several hundreds of thousands of tonnes) to pesticides (tens of thousands of tonnes) and speciality chemicals and pharmaceuticals (thousands of tonnes down to kilogram quantities). In addition, industrial chemical production has also lead, in some instances, to production of undesirable by-products and emissions often characterized by their persistence and resistance to breakdown (such as chlorinated dioxins).

As noted above, organochlorine compounds have a range of physico-chemical properties. In the environment, organochlorines can be transformed by a variety of microbial, chemical and photochemical processes. The efficiency of these environmental processes are largely dependent on the physico-chemical properties of the specific compound and characteristics of the receiving environment.

Cyclic, aromatic, cyclodiene-type and cyclobornane type chlorinated hydrocarbon compounds, such as some chlorinated pesticides, with molecular weights greater than 236 g/mol have been noted for their ability to accumulate in biological tissues, and to particularly concentrate in organisms that occupy positions in the upper trophic levels; not surprisingly, these compounds are also known for their persistence in the environment. Compounds included in this class often share many physico-chemical characteristics and include some of the earliest organochlorine pesticides such as DDT, chlordane, lindane, heptachlor, dieldrin, aldrin, toxaphene, mirex and chlordecone (Smith, 1991). Conversely, the lower molecular weight chlorinated hydrocarbons (less than 236 g/mol) may include a number of alkanes and alkenes (dichloromethane, chloropicrin, chloroform) and are often associated with little acute toxicity, reversible toxicological effects and relatively short environmental and biological half-lives (Hayes and Curley, 1968). Bioavailability, that proportion of the total concentration of a chemical that is available for uptake by a particular organism, is controlled by a combination of chemical properties of the compound including the ambient environment and the morphological, biochemical and physiological attributes of the organism itself.

Generally, excretion of organic pollutants is facilitated through the metabolic conversion to more polar forms. Because of their resistance to degradation and breakdown, the persistent organic pollutants are not easily excreted and those pollutants (e.g., toxaphene, PCBs etc.) most resistant to metabolism and disposition tend to accumulate in organisms and through the food chain. Notably, some organic pollutants may also be converted to more persistent metabolites than the parent compound, as is the case with the metabolic conversion of DDT to DDE. Similarly, the rapid metabolic conversion of aldrin to its extremely environmentally persistent metabolite dieldrin, is also noteworthy.

4.2 TOXICOLOGY

4.2.1 Environment

If analyzed for in tissues or environmental samples, some persistent organic pollutants will almost always be found. As is the case with many environmental pollutants, it is most difficult to establish causality of illness or disease that is directly attributable to exposure to a specific persistent organic pollutant or group of POPs. This difficulty is further underscored by the fact that persistent organic pollutants rarely occur as single compounds and individual field studies are frequently insufficient to provide compelling evidence of cause and effect in their own right. More to the point, however, is the fact that the significant lipophilicity of these compounds means that persistent organic pollutants are likely to accumulate, persist and bioconcentrate and could, thus, achieve toxicologically relevant concentrations even though discrete exposure may appear limited and acute exposure may appear of little relevance to chronic toxicity.

Experimentally, persistent organic pollutants have been associated with significant environmental impact in a wide range of species and at virtually all trophic levels (Hileman, 1993; Gilman, 1991). While acute effects of POPs intoxication have been well documented (Flickinger, 1979; Babcock and Flickinger, 1977), adverse effects associated with chronic low level exposure in the

environment is of particular concern. Noteworthy in this context is the long biological half life of persistent organic pollutants in biological organisms thereby facilitating accumulation of seemingly small unit concentrations over extended periods of time. For some POPs, there is some experimental evidence that such cumulative low level exposures may be associated with chronic non-lethal effects including potential immunotoxicity, dermal effects, impairment of reproductive performance and frank carcinogenicity (Parkinson and Safe, 1987; Allen *et al.*, 1979).

Immunotoxicity in association with exposure to different persistent organic pollutants has been reported by several authors (Hileman, 1993; Martineau *et al.*, 1987; Reijnders and Brasseur, 1992; Muir *et al.*, 1990a; Tryphonas *et al.*, 1991). Investigators have demonstrated immune dysfunction as a plausible cause for increased mortality among marine mammals and have also demonstrated that consumption of diets contaminated with persistent organic pollutants in seals may lead to vitamin and thyroid deficiencies and concomitant susceptibility to microbial infections and reproductive disorders. Investigators have also noted that immune deficiency has been induced in a variety of wildlife species by a number of prevalent persistent organic pollutants, including TCDD's, PCBs, chlordane, HCB, toxaphene and DDT.

Exposure to persistent organic pollutants has been correlated with population declines in a number of marine mammals including the common seal (Reijnders, 1986) the harbour porpoise, bottle-nosed dolphins (Duinker, 1985) and beluga whales from the St. Lawrence River (Martineau *et al.*, 1987). More notably, a clear cause and effect relationship has been established between reproductive failure in mink and exposure persistent organic pollutants. Wren (1991) has reported a direct cause and effect relationship in mink and ferrets between PCB exposure and immune dysfunction, reproductive failure, increased kit mortality, deformations and adult mortality. Similarly, investigators have also demonstrated a correlation between environmental concentrations of PCBs and dioxins with reduced viability of larvae in several species of fish (Hansen *et al.*, 1985). Noteworthy as well is a report by Colborn (1991) suggesting significant reproductive impairment in a number of Great Lakes species described as top level predators dependent on the Great Lakes aquatic food chain. Supporting this is the observation that wildlife, including stranded carcasses of St. Lawrence beluga whales, with reported high incidence of tumours have contained significantly elevated concentrations of PCBs mirex, chlordane and toxaphene (Swain *et al.*, 1992; Beland *et al.*, 1992, Muir *et al.*, 1990; Martineau *et al.*, 1988). Leatherland (1992) has also reported a 100 % incidence of thyroid lesions associated with increased body burdens of persistent organic pollutants in coho, pink and chinook salmon sampled in the Great Lakes over the last two decades.

4.2.2 Human health

As noted for environmental effects, it is also most difficult to establish cause and effect relationships for human exposure of persistent organic pollutants and incident disease. As with wildlife species, humans encounter a broad range of environmental exposures and frequently to a mixture of chemicals at anyone time. Much work remains to be done to elucidate human health impact of exposure to persistent organic pollutants.

The weight of scientific evidence suggests that some POPs have the potential to cause significant adverse effects to human health, at the local level, and at the regional and global levels through long-range transport. For some POPs, occupational and accidental high-level exposure is of concern for both acute and chronic worker exposure. The risk is greatest in developing countries where the

use of POPs in tropical agriculture has resulted in a large number of deaths and injuries. In addition to other exposure routes, worker exposure to POPs during waste management is a significant source of occupational risk in many countries. Short-term exposure to high concentrations of certain POPs has been shown to result in illness and death. For example, a study in the Philippines showed that in 1990 endosulfan became the number one cause of pesticide-related acute poisoning among subsistence rice farmers and mango sprayers. Occupation, bystander and near-field exposure to toxic chemicals is often difficult to minimize in developing countries. Obstacles in managing workplace exposure are in part due to poor or non-existent training, lack of safety equipment, and substandard working conditions. As well, concerns resulting from near-field and bystander exposure are difficult to identify due to inadequacies in monitoring of the ambient environment and inconsistencies in medical monitoring, diagnosis, reporting and treatment. These factors contribute to a lack of epidemiological data. Earliest reports of exposure to persistent organic pollutants related to human health impact include an episode of HCB poisoning of food in south-east Turkey, resulting in the death of 90 % of those affected and in other exposure related incidences of hepatic cirrhosis, porphyria and urinary , arthritic and neurological disorders (Peters, 1976). In another acute incident in Italy in 1976, release of 2,3,7,8-TCDD to the environment resulted in a purported increase of chloracne and an increased leukaemia and thyroid cancer related mortality (Pestaori *et al.*, 1993). More recently, the US EP A have been reviewing the dioxin related health effects, especially for the non-carcinogenic endpoints such as immunotoxicity, reproductive diseases and neurotoxicity.

Such frank expressions of effects are not as common in the case of exposure to lower concentrations derived from the environment and the food chain. Laboratory and field observations on animals, and studies on cell cultures collectively demonstrate that overexposure to certain POPs may be associated with a wide range of biological effects. These adverse effects may include immune dysfunction, neurological deficits, reproductive anomalies, behavioural abnormalities, and carcinogenesis. The scientific evidence demonstrating a link between chronic exposure to sublethal concentrations of POPs (such as that which would occur as a result of long-range transport) and human health impacts is more difficult to establish, but gives cause for serious concern. Swedish investigations have reported that dietary intake of PCBs, dioxins and furans may be linked to important reductions in the population of natural killer cells (lymphocytes) (Svenson *et al.*, 1993), while DeWailly *et al.*, (1993) have reported that children with high organochlorine dietary intake may experience rates of infection some 10-15 times higher than comparable children with much lower intake levels. The developing fetus and neonate are particularly vulnerable to POPs exposure, due to transplacental and lactational transfer of maternal burdens at critical periods. Moreover, Ayottee and co-workers (1995) have also reported that residents of the Canadian arctic who exist at the highest trophic level of the arctic aquatic food chain, have PCB intake levels in excess of the acceptable daily intake, and that may place this population at special risk for reproductive and developmental effects. In another report, (DeWailly *et al.*, 1993), children in the northern Quebec region of Canada who have had significant exposure to PCBs, dioxins and furans through breast milk also had a higher incidence of middle ear infections than children who had been bottle fed. Most authors, however, conclude that the benefits of breast feeding outweigh the risks.

Studies of carcinogenesis associated with occupational exposure to 2,3,7,8, TCDD also seem to indicate that extremely high level exposures of human populations do elevate overall cancer incidence. Laboratory studies provide supporting evidence that selected organochlorine chemicals may have carcinogenic effects and act as a strong tumour promoter.

More recently, some authors have suggested a possible relationship between exposure to persistent organic pollutants and human disease and reproductive dysfunction. Sharpe and Skakkebaek have

observed (1993) that the increasing incidence of reproductive abnormalities in the human male may be related to increased estrogen (or estrogenic type) compound exposure in vitro, and further suggest that a single maternal exposure during pregnancy of minute amounts of TCDD may increase the frequency of cryptorchidium in male offspring, with no apparent sign of intoxication in the mother. Thomas (1995) has, however, suggested that with respect to the purported link between presumed environmental estrogens and the increasing frequency of testicular cancers, the male gonad is an important source of endogenous estrogens. Colborn and Clement (1992) have reported that high concentrations of various persistent organic pollutants have been associated with reproductive abnormalities, including changes in the semen quality of adult rats exposed neonatally to PCBs via their mothers' milk. Similarly, Rogan and co-workers have speculated (1987) that DDE may interfere with a mother's ability to lactate, possibly due to the estrogenic properties of this chemical. Associations have been suggested between human exposure to certain chlorinated organic contaminants and cancers in human populations. Preliminary evidence suggests a possible association between breast cancer and elevated concentrations of DDE, although the role of phytoestrogens and alterations in lifestyle cannot be dismissed as important risk factors in the increase in estrogen dependent breast cancer incidence. Wolff' and co-workers (1993) have reported that levels of DDE and PCBs were higher for breast cancer case patients than for control subjects, noting that statistical significance was achieved only for DDE. While a causal relationship between organochlorine exposure and malignant breast disease remains far from proven (Krieger et at., 1994; Houghton and Ritter, 1995), the possibility of chronic low level exposure, when coupled with the known bioaccumulative properties of persistent organic pollutants suggests that important work in this area will undoubtedly continue for some time to come.

5. PERSISTENT ORGANIC POLLUTANTS

A number of persistent organic pollutants have been identified for inclusion in this report. These are reviewed in more detail in the following sections.

Information on countries that have taken action to ban or severely restrict compounds is derived from multiple sources dating back to 1987. This information needs to be verified and updated.

5.1 ALDRIN

5.1.1 Introduction

Aldrin was first synthesized in 1948, and commercially manufactured as a pesticide in 1950. Aldrin is the common name ;of the insecticide containing 95 % HHDN (an acronym for the chemical name 1,2,3,4,10, l0-hexachloro-1,4,4a,5,8,8a-hexahydro-*exo*-1 ,4-*endo*-5,8-dimethanonapthalene), and technical aldrin contains 90% aldrin as defined above (WHO, 1989a).

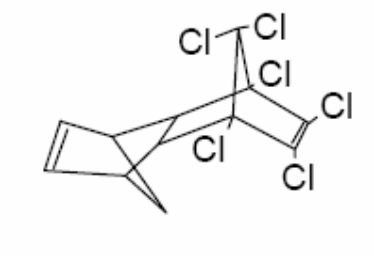
Technical aldrin contains no less than 85.5% of the main ingredient (HHDN), not less than 4.5 % of insecticidally active related compounds and not more than 10% other compounds (Smith, 1991). Aldrin is readily metabolized to dieldrin by both plants and animals. As a result, aldrin residues are rarely found in foods and animals, and then only in small amounts (WHO, 1989a). Aldrin binds strongly to soil particles and is very resistant to leaching into groundwater. Volatilization I is an important mechanism of loss from the soil. Due to its persistent nature and hydr9Phobicity, aldrin is known to bioconcentrate, mainly as its conversion products (WHO, 1989a).

Aldrin is produced by the Diels-Alder reaction of hexachlorocyclopentadiene with an excess of bicycloheptadiene at l00°C (WHO, 1989a). It is used to control soil insects such as tennites, com rootworm, wireworms, rice water weevil, and grasshoppers. It has been widely used to protect crops such as corn and potatoes, and has been effective to protect wooden structures from termites (WHO, 1989a).

Aldrin is banned in many countries, including but not restricted to Bulgaria, Ecuador, Finland, Hungary, Israel, Singapore, Switzerland and Turkey. Its use is severely restricted in many countries, including but not restricted to Argentina, Austria, Canada, Chile, the EU, Japan, New Zealand, the Philippines, USA, and Venezuela (Gips, 1987).

5.1.2 Chemical properties

CAS chemical name: 1,2,3,4,10,10-Hexachloro-1, 4, 41, 5, 8, 8a – hexahydro-1, 4:5, 8-dimethanonaphthalene.



Synonyms and Trade Names (partial list): Aldrec, Aldrex, Aldrex 30, Aldrite, Aldrosol, Altox, Compound 118, Drinox, Octalene, Seedrin.

CAS No.: 309-00-2; molecular formula: $C_{12}H_8Cl_6$; formula weight: 364.92

Appearance: White, odorless crystals when pure; technical grades are than to dark brown with a mild chemical odor.

Properties: Melting point: 104°C(pure), 49-60°C(technical); boiling point: 145°C at 2mm Hg; K_H:4,96 x 10^{-4} atm m^3/mol at 25°C; log K_{oc}: 2.61, 4.69; logK_{ow}:5.17-7.4; solubility in water: 17-180 μg/L at 25°C; vapor pressure: 2.31 x 10^{-5} mm Hg at 20°C. (source: Montgomery, 1993).

5.1.3 Toxicology

5.1.3.1 Studies in humans

Aldrin is toxic to humans; the lethal dose of aldrin for an adult man has been estimated to be about 5 g, equivalent to 83 mg/kg body weight (Hodge et at., 1967). Signs and symptoms of aldrin intoxication may include headache, dizziness, nausea, general malaise, and vomiting, followed by muscle twitchings, myoclonic jerks, and convulsions.

Brown (1992) has studied workers from a plant involved in the manufacture of aldrin, dieldrin and endrin. The study group insisted of white males who had been employed at the plant for at least six months prior to December 31, 1964. A statistically significant increase in liver and biliary tract cancers was observed. The author notes, however, that when interpreting the results, several factors must be considered; (1) there is a lack of quantitative information on exposure to the pesticide, (2) dibromochloropropane (DBCP), an animal carcinogen, which was manufactured at the plant between 1955 and 1976, and therefore is a potential confounding exposure, (3) the liver cancers were not homogenous, but were a mixture of extrahepatic and intrahepatic tumors, in contrast to studies in experimental animals which resulted in intrahepatic tumors and (4) there does not appear to be a dose-response relationship when dose is measured as length of employment.

Other organochlorines, such as dioxins, have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alterations in immune function (Holsapple, *et al.*, 1991). There is also limited evidence that cyclodienes such as aldrin may affect immune responses (Exon *et al.*, 1987). Some organochlorines, such as DDE, may have weak estrogenic properties, and some authors suggested a possible role in estrogen receptor positive breast cancer (Wolff *et al.*, 1993), while other authors (Krieger *et al.*, 1994) have been unable to demonstrate such a role for DDT or

its metabolites. Halogenated aromatic hydrocarbons are also known to affect endocrine function and reproductive systems (Peterson *et al.*, 1992; Gray, 1992; Thomas and Colborn, 1992). Aldrin itself has not been directly linked to these effects *per se*, the similarity of structure and chemical properties shared by halogenated aromatic hydrocarbons suggests a basis of concern for this chemical.

5.1.3.2 Studies in laboratory animals

Aldrin is highly toxic in laboratory animals (Table 5.1-1). Toxicity of the formulated product is highly dependant on the percentage of active ingredient in the formulation, the solvent used and the nature of the formulation. Acute oral LD_{50} values range from 100-4500 mg total aldrin formulation /kg body weight (Muir, 1970), while dermal LD50 values vary from 500-16000 mg total aldrin formulation/kg body weight (Rose 1982; 1984a).

Castro *et al.*, (1992) treated pregnant female rats with 1.0 mg/kg aldrin subcutaneously from the first day of pregnancy until delivery. The only changes reported included a decrease in the median effective time for incisor teeth eruption and an increase in the median effective time for testes descent. The authors also reported behavioral changes in rat pups (increased locomotion frequency), which persisted during the lifetime of the animal. WHO (1989a) was not able to establish a no-effect level for aldrin for reproductive toxicity, but did conclude that based on the available data, there is no evidence of a teratogenic potential for aldrin.

IARC (1987a) has concluded mat there is inadequate evidence for the carcinogenicity of aldrin in humans, and there is only limited evidence in experimental animals. Aldrin is therefore not classifiable as to its carcinogenicity in humans (IARC, Group 3).

Table 5.1-1 Acute toxicity of technical aldrin to mammals.*

Species	Route	Vehicle	LD_{50} (mg/kg body weight)	Reference
Mouse	oral	corn oil	44	Borgmann *et al.* (1952a)
Rat	oral	various	38-67	Lehman (1952); Borgmann *et al.* (1952a); Treon and Cleveland (1955); Gaines (1960); Worthing and Walker (1983)
	dermal	xylene	~ 100	Gaines (1960)
Hamster	oral	olive oil	320	Gak *et al.* (1976)
Guinea Pig	oral	corn oil	33	Borgmann *et al.* (1952a)
Rabbit	oral	corn oil	50-80	Borgmann *et al.* (1952a)
	dermal	dimthylphthalate	150	Lehman (1952)
Dog	oral	corn oil	65-95	Borgmann *et al.* (1952a)

* Taken from WHO (1989a).

5.1.3.3 Plants

Aldrin has low phytotoxicity, with tomatoes and cucumber affected only by extremely high application rates (Edwards, 1965). When applied at a rate of 16 kg active ingredient/ha to 2-3 week old tomato plants, cauliflower and Chinese cabbage seedlings, root development was inhibited and the growth rate of cabbage and cauliflower were inhibited (Hagley, 1965). Soybean emergence, growth, yield and chemical composition were unaffected by aldrin applied at 11 kg active ingredient/ ha (Probst and Everly, 1957).

5.1.3.4 Wildlife

The toxicity of aldrin to aquatic organisms is quite variable, with aquatic insects being the most sensitive group of invertebrates. The 96-h LC_{50} values range from 1-200 μg/L for insects, and from 2.2-53 μg /L for fish (WHO, 1989a). Long term and bioconcentration studies are performed primarily using dieldrin, the primary conversion product of aldrin. In a model ecosystem study, only 0.5 % of the original radioactive aldrin was stored as aldrin in the mosquitofish (*Gambusia affinis*), the organism at the top of the model food chain (Metcalf *et al.*, 1973).

Table 5.1-2 Acute toxicity of aldrin to selected aquatic organisms.*

Species	Developmental stage, body weight or length	Temperature (°C)	96 h LC_{50} (static) (μg/L)	Reference
Daphnia magna			29[a]	Anderson (1960)
Crangon septemspinosa (sand shrimp)	0.25 g 2.6 cm	20	8	Eisler (1969)
Pteronarcys californica (stonefly)	naiad, 3-3.5 cm	15.5	1.3	Sander and Cope (1968), Johnson and Finley (1980)
Salmo gairdneri (rainbow trout)	0.6 g	13	2.6	Johnson and Finley (1980)
Pimephales promelas (fathead minnow)	0.6 g	18	8.2	Johnson and Finley (1980)
Lepomis macrochirus (bluegill)	0.7 g	18	6.2	Johnson and Finley (1980)

[a] 48 h LC_{50} * Taken from WHO (1989a).

The acute toxicity of aldrin to avian species is given in Table 5.1-3. Eggs from chickens fed 1 mg aldrin/ kg diet for two years showed normal fertility and hatchability (Brown *et al.*,1965).

Table 5.1-3 Acute toxicity of aldrin for avian species.*		
Species	LD_{50} (mg/kg body weight)	Reference
Dendocygna bicolor (Fulvous whixtling duck)	29.2 (male)	Tucker and Crabtree (1970)
Anas piatyrhyndros (Mallard duck)	520 (female)	Tucker and Crabtree (1970)
Gallus domesticus (Domestic fowl)	25.5	Sherman and Rosenberg (1953)
Colinus virginianus (Bobwhite quail)	6.6 (female)	Tucker and Crabtree (1970)
Phasianus coldlicus (Ring-necked pheasant)	16.8 (female)	Tucker and Crabtree (1970)
Columbia livia (pigeon)	55	Turtle *et al.* (1963)

* Taken from WHO (1989a).

Aldrin treated rice is thought to be the cause of deaths of waterfowl, shorebirds and passerines along the Texas Gulf Coast, both by direct poisoning by ingestion of aldrin treated rice and indirectly by consuming organisms contaminated with aldrin (Flickinger and King, 1972). Residues of aldrin were detected in all samples of bird casualties, eggs, scavengers, predators, fish, frogs, invertebrates and soil. A total of 192 bird casualties were collected in the study areas during 1967-71, with Fulvous tree ducks having the highest number collected.

5.1.4 Persistence/fate

Aldrin is readily and rapidly converted to dieldrin in the environment. The half life of dieldrin in temperate soils is approximately 5 years (WHO, 1989a). This persistence, combined with high lipid solubility, provides the necessary conditions for dieldrin to bioconcentrate and biomagnify in organisms. Guppies (Poecilia reticulata) exposed to dieldrin concentrations ranging from 0.8 to 4.2 µg/L for 32 days had bioconcentration factors of up to 12,500 (Reinart, 1972). Similarly, sculpins (Cottus perplexus) exposed to dieldrin concentrations ranging from 0.017 to 0.86 µg/L for 32 days had bioconcentration factors of up to 13,300 (Chadwick and Brocksen, 1969). Diets containing dieldrin (or aldrin) administered concurrently with exposure to contaminated water did not have a significant effect on dieldrin accumulation, indicating that water is the principle source of dieldrin accumulation i.e. dieldrin is bioconcentrated rather than bioaccumulated (WHO, 1992a).

Aldrin's chemical properties (low water solubility, high stability, and semi-volatility) favor its long range transport, and dieldrin has been detected in arctic air, water and organisms (Barrie *et al.*, 1992; Lockhart *et al.*, 1992; Thomas *et al.*, 1992; Muir *et al.*, 1992). See ch. 3 for a more detailed explanation of this process and levels detected.

5.1.5 Exposure

Aldrin is readily metabolised to dieldrin in both animals and plants, and therefore aldrin residues are rarely present in animals and then only in very small amounts (WHO, 1989a). Residues of aldrin were not detected in a survey of Spanish meat and meat products conducted between January 1989 and December 1991 (Herrera *et al.*, 1994). Aldrin residues were detected in 21 of 100 samples (average concentration: 8.8 µg/kg, maximum concentration: 54.27 µg/kg) of Egyptian fish sampled between March 1986 and March 1988 (Abdallah *et al.*, 1990). Only 0.5 % of eggs of the domestic fowl (*Gallus domesticus*) from central Kenya contained residues of aldrin (Mugambi *et al.*, 1989)

The average daily intake of aldrin and dieldrin in India was calculated to be 19 µg/person (Kannan *et al.*, 1992b), exceeding the limit of 6.0 µg/60 kg of body weight recommended by the Joint FAO/WHO Meeting on Pesticide Residues (JMPR). Dairy products, such as milk and butter, and animal meats are the primary sources of exposure. This contrasts sharply with the average daily intake of 0.55 µg/person calculated for Vietnam (Kannan *et al.*, 1992a).

5.2 CHLORDANE

5.2.1 Introduction

Chlordane is a mixture of chlorinated hydrocarbons containing chlordane, heptachlor, nonachlor and related compounds. Technical chlordane typically contains 64-67 % chlorine (NRCC, 1974). Chlordane is highly insoluble in water, and is soluble in organic solvents. It is semi-volatile and can be expected to partition into the atmosphere as a result. It binds readily to aquatic sediments and bioconcentrates in the fat of organisms as a result of its high partition coefficient (log K_{ow} = 6.00).

Chlordane was first introduced as an insecticide in 1945. It is produced by reacting hexachlorocyclopentadiene with cyclopentadiene to form chlordene, which is then chlorinated to form chlordane (IARC, 1991a). Prior to 1951, the un reacted intermediate hexachlorocyclopentadiene was present in varying concentrations, resulting in a higher dermal and respiratory toxicity than later formulations, which contain less than 1 % of this contaminant (Smith, 1991).

Chlordane is a broad spectrum contact insecticide that has been used on agricultural crops including vegetables, small rains, maize, other oilseeds, potatoes, sugarcane, sugar beets, fruits, nuts, cotton and jute. It has also been used extensively in the control of termites (Smith, 1991).

Countries where chlordane is banned or severely restricted include, but not restricted to:

Banned		**Severely Restricted**	
Austria	Norway	Argentina	Indonesia
Belgium	Panama	Belize	Israel
Bolivia	Paraguay	Bulgaria	Mexico
Brazil	Philippines	Canada	New Zealand
Chile	Poland	China	South Africa
Columbia	Portugal	Cyprus	Sri Lanka
Costa Rica	Santa Lucia	Dominica	USA
Denmark	Singapore	Egypt	Venezuela
Dominican Republic	Spain	Honduras	
EU	Sweden		
Kenya	Switzerland		
Korea	Tonga		
Lebanon	Turkey		
Lichtenstein	United Kingdom		
Mozambique	Yemen		
Netherlands	Yugoslavia		

5.2.2 Chemical properties

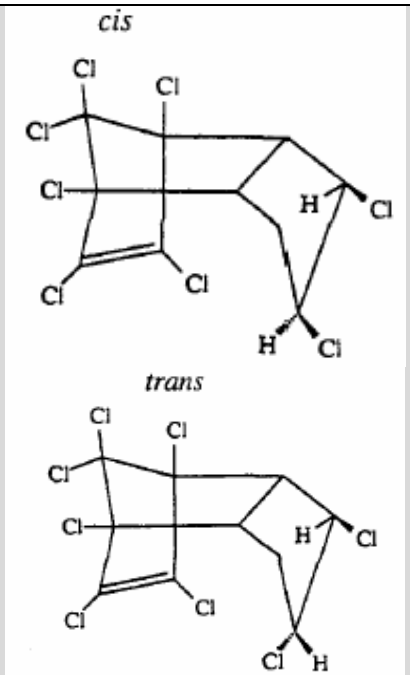
CAS Chemical Name: 1,2,3,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4, 7-methano-1H-indene

Trade names: (partial list): Aspon, Belt, Chloriandrin, Chlorkil, Chlordane, Corodan, Cortilan-neu, Dowchlor, HCS 3260, Kypchlor, M140, Niran, Octachlor, Octaterr, Ortho-Klor, Synklor, Tat chlor 4, Topichlor, Toxichlor, Veliscol-1068

CAS No.: 57-74-9; molecular formula: $C_{10}H_6Cl_8$; formula weight: 409.78

Appearance: colourless to yellowish-brown viscous liquid with an aromatic, pungent odour similar to chlorine;

Properties: Melting point: <25°C; bp: 165°C at 2 mm Hg; K_H: 4.8 x 10^{-5} atm m^3/mol at 25°C; log KOC: 4.58-5.57; log K_{ow}: 6.00; solubility in water: 56 ppb at 25°C; vapor pressure: 10^{-6} mm Hg at 20°C. (source: Montgomery, 1993).



5.2.3 Toxicology

5.2.3.1 Studies in humans

Symptoms of chlordane exposure include blurred vision, confusion, ataxia, delirium, cough, abdominal pain, nausea, vomiting, diarrhea, irritability, tremor, convulsions and anuria (Montgomery, 1993). Early studies on occupational exposure found no toxic effects in workers involved in the production of chlordane for 2 months to 5 years (Alvarez and Hyman, 1953) and for those with up to 15 years of exposure (Fishbein *et al.*, 1964). Stein and Hayes (1964)surveyed 1105 workers associated with pest control, most of whom used chlordane, however, only three attributed illness to it (mild dizziness, headache, weakness).

In another study that followed workers involved in the production of chlordane over a 23 year period (1964-1987) increased cancer related mortality was not reported, but the authors did observe an increase in cerebrovascular disease (Brown, 1992). The lack of an increase in mortality due to cancer is supported by several other earlier reports as well (Smith 1991).

In another study that examined 27 individuals who complained of health effects which they associated with chlordane exposure, the authors reported statistically significant changes in the immune systems characterized by impaired proliferative responses to mitogens. It should be noted that three of the individuals were exposed to direct skin application through accidental exposure at work (McConnachie and Zahalsky, 1992).

Organochlorines have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alterations in immune function (Holsapple *et al.*, 1991). There is also 47 December 1995 limited evidence that cyclodienes such as chlordane may affect immune responses (Exon *et*

al., 1987). Some organochlorines, such as DDE, may have weak estrogenic properties, and some authors have suggested a possible role in estrogen receptor positive breast cancer (Wolff *et al.*, 1993), while other investigators have been unable to demonstrate such a role for DDT or its metabolites (Krieger et at., 1994). Although chlordane itself has not been directly linked to these effects per se, halogenated aromatic hydrocarbons have been reported to affect endocrine function and reproductive systems (Peterson et at., 1992; Gray, 1992; Thomas and Colborn, 1992).

5.2.3.2 Studies in laboratory animals

Acute oral toxicity for chlordane varies with the purity of the test compound and with the vehicle of administration. Reported values for rats range from 83 mg/kg for pure cis-chlordane (Podowski *et al.*, 1979) to 560 mg/kg for chlordane of unspecified purity (Ambrose *et al.*, 1953).

Table 5.2-1 Acute toxicity of chlordane in mammals[1]

Species	Sex	Route	Vehicle	LD_{50} (mg/kg)	Reference
Rat	M	oral	peanut oil	335	Gaines (1969)
Rat	F	oral	peanut oil	430	Gaines (1969)
	F	dermal	xylene	530	Gaines (1969)
Rabbit	NS	oral	NS	100-300	Stohlman *et al.* (1950)
	NS	dermal	NS	1100-1200	Ingle (1965)
Hamster	NS	oral	NS	1720	Truhaut *et al.* (1974)

NS- Not specified. Data compiled by WHO (1984a)

As noted elsewhere, the acute toxicity of chlordane is dependent on, among other things, the purity of the test material. Noteworthy is that acute dermal toxicity of chlordane ranged from 780 mg/kg for technical compound prior to 1951 to 1100-1200 mg/kg for more recent and purified technical chlordane (Ingle, 1965).

Subchronic (90 day) inhalation exposure in rats and monkeys at doses up to 10 μg/L (10 mg/m3) resulted in increases in the concentration of cytochrome P-450 and microsomal protein in rats (Kaswinah *et al.*, 1989). These effects were largely reversible during a 90 day recovery period following exposure to the test compound. No significant treatment related biological changes were observed in the monkeys at any dose level. The results of this study provide a no-effect level in the rat of approximately 0.1 μg/L (0.1 mg/m^3) and in excess of 10 μg/L (10 mg/m^3) in the monkey.

Mice were fed diets with 25-100 mg chlordane/kg for 6 generations. At the highest level, viability was decreased in the first and second generation, and no offspring were produced in the third generation. At 50 mg/kg, viability was decreased in the third and fourth generation, December 1995 48 and at 25 fig/kg no statistically significant effects were observed after 6 generations (Keplinger et al., 1968). Rabbits were administered chlordane orally at levels of 0, 1.0, 5.0, and 15 fig/kg body weight on the 6th -18th days of gestation. No changes in behaviour, appearance or body weight were observed, and no teratogenic effects were reported (IRDC, 1972).

IARC (1991a) has concluded that while there is inadequate evidence for the carcinogenicity of chlordane in humans, there is sufficient evidence in experimental animals. IARC has classified chlordane as a possible human carcinogen (Group 2B).

5.2.3.3 Plants

No data available.

5.2.3.4 Wildlife

Table 5.2-2 Toxicity of chlordane to selected aquatic organisms.*

Species	Size/Age	Temp (°C)	96-h LC_{50} (μg/L)	Reference
Penaeus duorarum (pink shrimp)	50-65 mm	28.4	0.4	Parrish *et al.* (1976)
Cancer magister (Dungeness crab)	zoeal	13	1.3 (immobil.)	Caldwell (1977)
Lepomis macrochirus (bluegill)	38-44	25	22	Henderson *et al.* (1959)
Pimphales promeias (fathead minnow)	38-84 mm	25	52	Henderson *et al.* (1959)
Salmo gairdneri (rainbow trout)	0.9 g	13	7.8	Cope (1965)

*Data compiled by WHO (1984a).

Selected studies on aquatic organisms using the post-1951 formulation of chlordane are summarised in Table 5.2-2. There are large differences within and between species, which may be attributed to differences in water temperature and sediment loadings; differences in age, condition and history of test organisms; and to different chlordane formulations utilized (Eisler, 1990).

The acute oral LD_{50} to 4-5 month old mallard ducklings was 1200 mg/kg body weight (Tucker and Crabtree, 1970). The LC_{50} for bobwhite quail fed chlordane in their diet for 10 weeks was 10 mg/kg diet (Ludke, 1976).

Chlordane is toxic to earthworms. Legg (1968) reported worm casts up to 13 months after a single application of chlordane at 9.0, 13.4, or 20.2 kg/ha on turf. After 19 days, reductions were 52, 72 and 98 %, compared to the control plots. After 13 months, the reductions were 89, 95, and 97% respectively.

There is growing evidence linking persistent halogenated aromatic hydrocarbons (especially PCBs and dioxins) to reproductive and immunotoxic effects in wildlife (Fox, 1992; Reijinders and Brasseur, 1992). Although chlordane has not been directly linked to these effects in wildlife, residues of chlordane have been detected in arctic organisms in conjunction with these compounds (refer to chapter 3 for levels detected).

5.2.4 Persistence/Fate

The persistence of chlordane combined with a high partition coefficient, provides the necessary conditions for chlordane to bioconcentrate in organisms. For example, fathead minnows exposed to 5.0 μg chlordane/L for 32 days had a concentration factor of 37800 for the whole body (Veith, 1979). A concentration factor of 16 000 was determined for sheepshead minnow exposed for 189 days (Parrish *et al.*, 1978).

Concentration factors in fish exposed to chlordane in their diet were lower than those for fish exposed to chlordane in the water. Goldfish exposed to chlordane in the diet had a concentration

factor of 162 (Moore *et al.*, 1977) compared to a concentration factor of 8258 for mosquito fish exposed to chlordane in both diet and water (Sanborn *et al.*, 1976). This suggests that chlordane is bioconcentrated (taken up directly from the water) as opposed to being bioaccumulated (taken ,up by water and in food).

The chemical properties of chlordane (low water solubility, high stability, and semi-volatility) favour its long range transport, and chlordane has been detected in arctic air, water and organisms (Barrie *et al.*, 1992; Lockhart *et al.*, 1992; Thomas *et al.*, 1992; Muir *et al.*, 1992). See ch 3 for a more detailed explanation of this process and levels detected.

5.2.5 Exposure

Chlordane exposure may occur through food, but due to its highly restricted uses, this route does not appear to be a major pathway of exposure. In an ongoing program to monitor organochlorine contaminants in the fat of domestic farm animals in Canada, residues of chlordane, and its metabolites heptachlor and heptachlor epoxide, were below the detection limit (0.001 mg/kg) in all samples from 1986 to 1988 (Frank *et al.*, 1990). Similarly, samples of Spanish meat and meat products collected between January 1989 and December 1991 did not contain any detectable residues of the same compounds (Herrera *et al.*, 1994). The isomer gamma-chlordane was detected in only 2 (8.00 and 36.17 μg/kg ww) of 92 samples of Egyptian fish sampled between March 1986 and March 1988 (Abdallah *al.*, 1990). Gans *et al.* (1994) detected gamma-chlordane in 2 of 9 samples of food products imported into Hawaii from Western Pacific Rim countries. Residues were detected in Oriental party beans (2.70 ppb) and in prepared seaweed (0.48 ppb).

Chlordane has been detected in indoor air of residences of both Japan and the USA. Dearth and Hites (1991) have suggested that exposure to chlordane in the air is an important source of exposure to the US population. Mean levels detected in the living areas of 12 homes in New Jersey prior to and after treatment for termites ranged from 0.14 to 0.22 μg/m3, respectively (Louis and Kisselbach, 1987). Mean levels in non-living areas (crawl spaces and unfinished basements) were higher; 0.97 μg/m3 before treatment and 0.91 $\mu g/m^3$ after treatment. The similarity between values before and after treatment in the non-living areas suggest the likelihood of another source of chlordane, possibly the building materials. Asakawa *et al.* (1994) found a significant correlation between the variation in the concentration of chlordane 'under the floor and the average monthly temperature. This observation further supports the hypothesis that chlordane concentrations in the air are associated with volatilization of chlordane residue, which is a temperature dependent process. The relationship was not as strong for the first floor, possibly due to greater ventilation. Fenske and Sternbach (1987) Compared levels in homes in New Jersey for two periods, 1976-1982 and 1983-1985, to determine the effect of regulations issued in December 1982 restricting sale and use of chlordane to certified applicators. Concentrations for the second period are significantly lower with an almost three-fold decrease in median values, from 2.6 to 0.9 $\mu g/m^3$.

5.3 DDT

5.3.1 Introduction

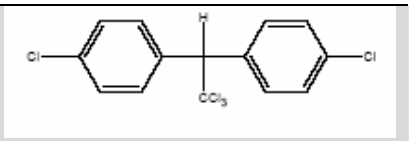
DDT was first synthesized by Othmar Zeidler in Germany in 1874, but its insecticidal properties were not discovered until 1939 by the Swiss chemist Paul Muller. It was widely used during the Second World War to protect the troops and civilians from the spread of malaria, typhus and other vector borne diseases (Smith, 1991). After the war, DDT was widely used on a variety of agricultural crops and for the control of disease vectors as well and is still being produced and used for vector control. Growing concern about adverse environmental effects, especially on wild birds, led to severe restrictions and bans in many countries in the early 1970s (Gips, 1987). DDT is highly insoluble in water and is soluble in most organic solvents. It is semi-volatile and can be expected to partition into the atmosphere as a result. Its presence is ubiquitous in the environment and residues have even been detected in the arctic. The persistence of DDT is a factor in both its suitability as a vector control agent and the problems in the environment related to it. It is lipophilic and partitions readily into the fat of all living organisms and has been demonstrated to bioconcentrate and biomagnify. The breakdown products of DDT, 1,1-dichloro-2,2-bis(4-chlorophenyl)ethane (DDD or TDE) and 1,1-dichloro-2,2bis (4-chlorophenyl)ethylene) (DDE), are also present virtually everywhere in the environment and are more persistent than the parent compound.

The largest agricultural use of DDT has been on cotton, which accounted for more than 80% of the USA's use before its ban there in 1972. DDT is still used to control mosquito vectors of malaria in a numerous countries (Gips, 1987).

The use of DDT has been banned in at least 34 countries and severely restricted in at least 34 other countries. The countries that have banned DDT include Argentina, Australia, Bulgaria, Colombia, Cyprus, Ethiopia, Finland, Hong Kong, Japan, Lebanon, Mozambique, Norway, Switzerland, and the USA Countries that have severely restricted its use include Belize, Ecuador, the European Union, India, Israel, Kenya, Mexico, Panama, and Thailand (Environment Canada, 1995). Other countries, not noted above, may also prohibit or severely restrict the use of DDT.

5.3.2 Chemical properties

CAS Chemical Name: 1, 1'-(2,2,2-Trichloroethylidene)bis(4-chlorobenzene).



Synonyms and Trade Names (partial list): Agritan, Anofex, Arkotine, Azotox, Bosan Supra, Bovidermol, Chlorophenothan, Chloropenothane, Clorophenotoxum, Citox, Clofenotane, Dedelo, Deoval, Detox, Detoxan, Dibovan, Dicophane, Didigam, Didimac, Dodat, Dykol, Estonate, Genitox, Gesafid, Gesapon, Gesarex, Gesarol, Guesapon, Gyron, Havero-extra, Ivotan, Ixodex, Kopsol, Mutoxin, Neocid, Parachlorocidum, Pentachlorin, Pentech, PPzeidan, Rudseam, Santobane, Zeidane, Zerdane

CAS No.: 50-29-3; molecular formula: $C_{14}H_9C_{15}$; formula weight: 354.49.

Appearance: Odourless to slightly fragrant colourless crystals or white powder.

Properties: Melting point: 108.5°C; boiling point: 185°C at 0.05 mm Hg (decomposes); K_H: 1.29 x 10^{-5} atm m^3/mol at 23°C; log K_{OC}: 5.146-6.26; log K_{ow}: 4.89-6.914; solubility in water: 1.2-5.μg/L at 25°C. (source: Montgomery, 1993).

5.3.3 Toxicology

5.3.3.1 Studies in humans

DDT has been widely used in large numbers of people who were sprayed directly in programs to combat typhus, and in tropical countries to combat malaria (WHO, 1979). Dermal exposure to DDT has not been associated with illness or irritation in a number of studies (Smith, 1991).

Studies were conducted using human volunteers to examine the storage and excretion of DDT and to determine possible effect of dosages considered to be safe. In the first study, DDT was administered at 0, 3.5 and 35 mg/person/day. These doses, combined with DDT in the food of the volunteers, resulted in dosage levels of 0.0021-0.0034, 0.038-0.63, and 0.36-0.61 mg/kg/day, respectively, depending on the weight of the individual. Six volunteers received the highest dosage for 12 months and 3 received it for 18 months. A smaller number of men ingested the lower dosage of technical DDT or *p,p'*-DDT for 12 or 18 months. No volunteer complained of any symptoms or showed, by the tests used, any sign of illness that did not have an easily recognizable cause clearly unrelated to the exposure to DDT (Hayes *et al.*, 1956). The same result was obtained in a second study in which the same dosages were given for 21 months and the volunteers observed for a minimum of an additional 27 months (Hayes *et al.*, 1971).

Brown (1992) performed a follow up study of mortality of workers involved in the production of DDT. The study groups comprised workers employed for a minimum of six months prior to December, 1964 and the follow up period was until December 1987. An excess of liver and biliary cancer was observed (expected deaths: 0.45; actual deaths 2). Although this increase was not statistically significant, the study is limited by lack of exposure data and a relatively small number of deaths. A significant increase in mortality from cerebrovascular disease was observed, although the role of DDT exposure and/or other factors related to cerebrovascular disease remains unclear.

Organochlorines have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alteration in immune function (Holsapple, *et al.*, 1991). Some organochlorines, such as

DDE, may have weak estrogenic properties, and some authors have suggested a possible role in estrogen receptor positive breast cancer (Wolff *et al.*, 1993), while other authors have been unable to demonstrate such an effect for DDT or its metabolites (Kreiger *et al.*, 1994; Ritter and Houghton, 1995). Halogenated aromatic hydrocarbons are also known to affect endocrine function and reproductive systems (Peterson *et al.*, 1992; Gray, 1992; Thomas and Colborn, 1992). Although DDT has not been directly linked to all of these effects *per se*, the similarity of structure and chemical properties shared by halogenated aromatic hydrocarbons suggests a possible adverse role for this chemical.

5.3.3.2 Studies in laboratory animals

Female rats reproduced normally .when fed DDT for two generations at dietary levels as high as 200 ppm (equivalent to approximately) 10 mg/kg/day) (Ottoboni, 1969). At a dietary level of 20 ppm, the dams had a significantly longer reproductive life span than their littermate controls. In a six generation reproduction study in mice, no effect on fertility, gestation, viability, lactation or survival were observed at a dietary level of 25 ppm (Keplinger *et al.*, 1970). A level of 100 ppm produced a slight reduction in lactation and survival in some generations, but not all, ant the effect was not progressive. A level of 250 ppm produced clear adverse reproductive effects. In both these and other studies, no evidence of teratogenicity has been observed (Smith, 1991).

Exon *et al.*, (1987) examined the available information regarding DDT effects on the immune system, and concluded that DDT is either suppressive or has no effect on the immune system. Generally, DDT appears to depress humoral immune responses.

Perinatal administration of weakly estrogenic pesticides such as DDT produces estrogen-like alterations of reproductive development (Gray, 1992). Similarly, Hunter and Kelsey (1993) have reported that the available data, although limited, do suggest a possible association between organochlorines, such as DDT and its metabolite DDE, and risk of breast cancer.

IARC (1991b) has concluded that while there is inadequate evidence for the carcinogenicity of DDT in humans, there is sufficient evidence in experimental animals. IARC has classified DDT as a possible human carcinogen (Group 2B).

Table 5.3-1 Acute toxicity of DDT to mammals.*

Species	Formulation	Oral (mg/kg)	Dermal (mg/kg)
Rat	oil solution	113-450	250-3000
Mouse	oil solution	100-800	250-500
Guinea Pig	oil solution	250-560	100
Rabbit	oil solution	300-1770	300-2820

*Taken from WHO (1989a)

5.3.3.3 Plants

No data available.

5.3.3.4 Wildlife

DDT is highly toxic to fish (Table 5.3-2). It also affects fish behaviour. Atlantic salmon eggs were exposed to DDT at concentrations of 5,10,50 or 100 μg/L, and the behavioural development of hatched fry was observed for 30 days following hatch (Dill and Saunders, 1974). The two highest dose levels of DDT impaired balance and delayed the appearance of normal behaviour patterns. DDT also affects temperature selection in fish. Gardner (1973) found that DDT induced selection of lower temperatures in brook trout (*Salvelinus fontinalis*) over a concentration range between 0 and 50 μg /L. Exposure of the same species of fish to concentrations as high as 8 mg/L caused fish to select higher temperatures (Peterson, 1973).

Table 5.3-2 Toxicity of DDT and its derivatives to selected aquatic organisms.*

Organism	Compound	Temp (°C)	96-h LC_{50} (μg/L)	Reference
Estuarine/Marine Organisms				
Crangon septemspinosa (shrimp)	DDT	20	0.4	McLeese and Metcalfe (1980)
Mysidopsis bahia (mysid shrimp)	DDT	25	0.45 (0.39-0.52)	Mayer (1987)
Cyprinodon variegatus (sheepshead minnow)	DDT	15	2.0 (48-h)	Mayer (1987)
Morone saxatillis (striped bass)	DDT (77%)	17	0.53 (0.38-0.84)	Korn and Earnest (1974)
	DDE	17	2.5 (1.6-4.0	Kornn and Earnest (1974)
Freshwater				
Daphnia magna (water flea)	DDT	20	1.1 (48-h) (1.0-1.3)	Randall *et al.* (1979)
Palemonetes kadiakensis (glass shrimp)	DDT	21	2.3 (1.3-4.9)	Sander (1972)
	TDE	21	0.68 (0.47-1.1)	Sander (1972)
Pteronarccys californica (stonefly naiad)	DDT	15.5	7 (4.9-9.9)	Sander and Cope (1968)
	TDE	15.5	380 (280-520)	Sander and Cope (1968)
Salmo gairdneri (rainbow trout)	DDT	20	42.0	Katz (1961)
	DDE	12	32.0 (26.0-40.0)	Mayer and Ellersieck (1986)
	TDE	12	70.0 (58.0-85.0)	Mayer and Ellersieck (1986)
Pimephales promelas (fathead minnow)	DDT	18	13.2 (10.1-17.3)	Mayer and Ellersieck (1986)

Table 5.3-2 Toxicity of DDT and its derivatives to selected aquatic organisms.*				
Organism	Compound	Temp (°C)	96-h LC_{50} (μg/L)	Reference
Lepomis macrochirus (bluegill)	DDT	18	6.3 (4.3-9.3)	Mayer and Ellersieck (1986)
	DDE	17	240 (201-286)	Mayer and Ellersieck (1986)
	TDE	24	42.0 (36.0-49.0)	Mayer and Ellersieck (1986)

* Taken from WHO (1989a).

DDT is not highly toxic to binds (Table 5.3-3), however it is best known for its adverse effects on reproduction, especially DDE, which causes egg shell thinning in birds with associated significant adverse impact reproductive success. There is considerable variation in the sensitivity of bird species t this effect, with birds of prey being the most susceptible and showing extensive egg shell thinning in the wild (WHO, 1989b).

American kestrels were fed day old cockerels injected with DDE, receiving doses of 0.3, .3, 6 or 10 mg DDE/kg diet (Lincer, 1975). Residues of DDE m the eggs correlated closely with the dietary DDE concentration. No egg shell thinning was associated with the lowest dose of 0.3 mg/kg. Higher doses showed 15.1%, 22.8% and 29.2% thinning, at the 3, 6, and 10 mg/kg dose levels respectively. There was a linear relationship between degree of egg shell thinning and the logarithm of the DDE residue in the egg. Data collected in the field has confirmed this trend.

Table 5.3-3 Acute toxicity of DDT and its derivatives to selected avian species.*				
Organism	Compound/ Purity %	Age	LD_{50} (mg/kg body weight)	Reference
Callipepla californica (California quail)	DDT (TG)	6 mo	595 (430-825)	Hudson *et al.* (1984)
	TDE (>95)	6mo	> 760	Hudson *et al.* (1984)
Anas platyrhynchos (mallard duck)	DDT (77.2)	3mo	> 2240	Hudson *et al.* (1984)
	TDE (>95)	3mo	> 2000	Hudson *et al.* (1984)
Phasianus colchicus (pheasant)	DDT (>99)	3-4 mo	1334 (864-1990)	Hudson *et al.* (1984)
	TDE (>95)	3-4 mo	386 (270-551)	Hudson *et al.* (1984)

* Taken from WHO (1989b).

There is growing evidence linking persistent halogenated aromatic hydrocarbons (especially PCBs and dioxins) to reproductive and immunotoxic effects in wildlife. (Fox, 1992; Reijinders and Brasseur, 1992). Specifically, DDT (in conjunction with other halogenated aromatic hydrocarbons) has been linked with feminization and altered sex-ratios of Western Gull populations off the coast of southern California, and Herring Gull populations in the Great Lakes (Fox, 1992).

5.3.4 Persistence/fate

DDT and related compounds are very persistent in the environment, as much as 50% can remain in the soil 10-15 years after application (Keller, 1970). This persistence, combined with a high partition coefficient (log K_{ow} = 4.89-6.91) provides the necessary conditions for DDT to bioconcentrate in organisms. Fathead minnows (*Pimephales promelas*) exposed to 2.0 µg/L for 14 and 112 1 days had bioconcentration factors of 69,100 and 154,100, respectively (Jarvinen *et al.*, 1977). Rainbow trout (*Salmo gairdneri*) exposed to DDT at 0.133 µg/L for 12 weeks had a bioconcentration factor of 51,335 (Reinert *et al.*, 1974). WHO (1989b) suggest that higher accumulations of DDT at higher trophic levels in aquatic systems results from a tendency for 'organisms at higher trophic levels to accumulate more DDT directly from the water, rather than by biomagnification.

The chemical properties of DDT (low water solubility, high stability and semi-volatility) favour its long range transport and DDT and its metabolites have been detected in arctic air, water and organisms (Barrie *et al.*, 1992; Lockhart *et al.*, 1992; Thomas *et al.*, 1992; Muir *et al.*, 1992). See ch 3 for al more detailed explanation of this process and levels detected. DDT has also been detected in virtually all organochlorine monitoring programs and is generally believed to be ubiquitous throughout the global environment.

5.3.5 Exposure

DDT and its metabolites have been detected in food from all over the world and this route is likely the greatest source of exposure for the general population (WHO, 1979). DDE was the second most frequently found residue (21 %) in a survey of domestic animal fats and eggs tested between 1986 and 1988 in Ontario, Canada (Frank *et al.*, 1990). The highest residue detected was 0.410 mg/kg in beef fat. Residues in domestic animals, however, have declined steadily over the past 20 years. In a survey of Spanish meat and meat products conducted between January, 1989 and December, 1991, 83% of lamb samples tested contained at least one of the DDT metabolites investigated with a mean level of 25 ppb (Herrera *et al.*, 1994). An average of 76.25 ppb *p,p'*-DDE was detected in fish samples from Egypt (Abdallah, *et al.*, 1990). DDT was the most common organochlorine detected in foodstuffs in Vietnam (Kannan *et al.*, 1992a). A higher proportion of *p,p'*-DDT than *p,p'*-DDE levels indicates the ongoing use of DDT in Vietnam. The highest concentrations of DDT and its metabolites were detected in meat and fish, with mean residue values of 3.2 and 2.0 µg/g fat, respectively. Residues as high as 6.2 and 5.0 µg/g fat were detected. The estimated daily intake of DDT and its metabolites in Vietnam was 9 µg/person/day, below the FAO/WHO acceptable daily intake of 600 µg/person/day. The estimated daily intake of DDT and its metabolites in India was somewhat higher, at 48 µg/person/day, but still below the acceptable daily intake (Kannan et at., 1992b). Average residues detected in meat and fish were 1.0 and 1.1 µg/g fat respectively, with maximums of 5.5 and 5.6 µg/g fat.

DDT has also been detected in human breast milk. Spicer and Kereu (1993) surveyed lactating mothers in four remote villages in Papua New Guinea. In a general survey of 16 separate compounds, DDT as detected in 100% of samples (41), and was one of only two organochlorines detected (the other was heptachlor epoxide). DDT has also been detected in the breast milk of Egyptian women (Dogheim *et al.*, 1991), with an estimated daily intake of total DDT for breast feeding infants of 6.90 µg/kg body weight /day. While lower than the acceptable daily intake of 20.0 µg/kg body weight recommended by the Joint FAO/WHO Meeting on Pesticide Residues (JMPR) , its continuing presence raises serious concerns regarding potential effects ~n developing infants. The average total DDT detected was 57.59 ppb.

5.4 DIELDRIN

5.4.1 Introduction

Dieldrin was first synthesized, together with aldrin, in 1948, and commercially manufactured in 1950. Dieldrin is the common name of the insecticide containing 85% HEOD (an acronym for the chemical name 1,2,3,4,10,10-hexachloro-6, 7-epoxy-l,4,4a,5,6, 7 ,8,8a-octahydro-endn-1 , 4-exo- 5, 8-dimethanonapthalene) , and technical dieldrin contains not less than 95 % of dieldrin as defined above (WHO, 1989a). To wit, technical dieldrin contains not less than 80.75% HEOD, not less than 14.25% insecticidally active related compounds and not more than 5% other compounds (Smith, 1991). Dieldrin binds strongly to soil particles and hence is very resistant to leaching into ground water. Volatilization is an important mechanism of loss from the soil, and because of its persistent nature and hydrophobicity, dieldrin is known to bioconcentrate (WHO, 1989a).

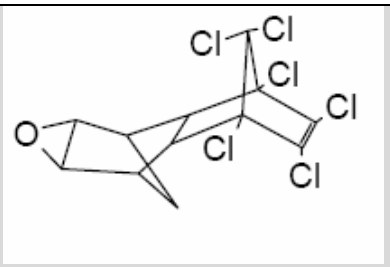
Dieldrin may be synthesized by the epoxidation of aldrin with a peracid such as peracetic acid, but may also be synthesized through the condensation of hexachlorocyclopentadiene with the epoxide of bicycloheptadiene (WHO, 1989a). Dieldrin has been used in agriculture for the control of soil insects and several insect vectors of disease (Smith, 1991) but this latter use has been banned in a number of countries due to environmental and human health concerns. Principle contemporary uses are restricted to control termites and wood borers and against textile pests (WHO, 1989a).

Action to ban dieldrin has been taken in many countries, including Bulgaria, Ecuador, the EU, Hungary, Israel, Portugal, Singapore, Sweden, and Turkey. Its use is severely restricted in numerous countries, includin9 Argentina, Austria, Canada, Colombia, Cyprus, India, Japan, New Zealand, Pakistan, USA and Venezuela (Gips, 1987). Countries other than those listed above may also prohibit or severely restrict the use of dieldrin.

5.4.2 Chemical properties

CAS Chemical Name: 3,4,5,6,9,9-Hexachloro-dimetanonapth-[2,3-*b*]oxirene.

Synonyms and Trade Names (partial list): Alvit, Dieldrite, Dieldrix, Illoxol, Panoram D-31, Quintox.



CAS No.: 60-57-1; molecular formula: $C_{12}H_8Cl_6O$; formula weight: 380.91.

Appearance: A stereo-isomer of endrin, dieldrin may be present as white crystals or pale tan flakes, ordourless to mild chemical odour.

Properties: Melting point: 175-176°C; boiling point: decomposes; K_H: 5.8 x 10^{-5} atm m^3/mol at 25°C; log K_{OC}: 4.08-4.55; log K_{ow}: 3.692-6.2; solubility in water: 140 μg/L at 20°C; vapour pressure: 1.78 x 10^{-7} mm Hg at 20°C. (source: Montgomery, 1993).

5.4.3 Toxicology

5.4.3.1 Studies in humans

Dieldrin is toxic to humans. Based on anecdotal evidence, the lethal dose of dieldrin has been estimated to be 10 mg/kg body weight (Hayes, 1982). Signs and symptoms of acute dieldrin intoxication are essentially the same as for aldrin, including headache, dizziness, nausea, general malaise and vomiting followed by muscle twitching, myoclonic jerks and convulsions.

Hunter *et al.* (1969) investigated the pharmacodynamics of dieldrin in a human volunteer study. The subjects, with no recent occupational exposure to dieldrin, received 0, 10, 50, or 211 μg dieldrin per day for 2 years. All the volunteers continued in excellent health, and clinical, physiological and laboratory findings remained essentially unchanged through the 24 month exposure period and an 8 month follow up.

Brown (1992) has studied workers from a plant involved in the manufacture of aldrin, dieldrin and endrin. The study group consisted of white males who had been employed at the plant for at least six months prior to December 31, 1964. The author reported a statistically significant increase in liver and biliary tract cancers. However, the author notes that when interpreting the results, several factors must be considered; (1) there is a lack of quantitative information on exposure to the pesticides, (2) a known animal carcinogen, dibromochloropropane (DBCP), was manufactured at the plant between 1955 and 1976, and therefore is a potential confounding exposure, (3) the liver cancers were not homogenous, but were a mixture of extrahepatic and intrahepatic tumours, in contrast to studies in experimental animals which resulted in intrahepatic tumours and (4)there does not appear to be a dose-response relationship when dose is measured as length of employment.

Organochlorines have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alterations in immune function (Holsapple, *et al.*, 1991). There is also limited evidence that cyclodienes such as dieldrin may also affect immune responses (Exon *et al*., 1987). Some organochlorines, such as DDE, may have weak estrogenic properties, and some authors have suggested a possible role in estrogen receptor positive breast cancer (Wolff *et al.*, 1993) while other

authors have been unable to demonstrate such an effect for DDT or its metabolites (Krieger *et al.*, 1994). Halogenated aromatic hydrocarbons are also known to affect endocrine function and reproductive systems (Peterson *et al.*, 1992; Gray, 1992; Thomas and Colborn, 1992). Although dieldrin itself has not been directly linked to these effects per se, the similarity of structure and chemical properties shared by halogenated aromatic hydrocarbons suggests a basis for concern for this chemical.

5.4.3.2 Studies in laboratory animals

Dieldrin is highly toxic in laboratory animals (Table 5.4-1). The toxicity of formulated products is lower than that of the pure compound, and depends largely on the concentration of active ingredient in the formulation, the solvent used and the nature of the formulation. Acute oral LD_{50} values range from 100-400 mg/kg body weight, depending on the formulation (Muir, 1970), while acute dermal LD_{50} values vary from 200-2700 mg formulation/kg body weight (Rose, 1984b). As with other organochlorine compounds, the liver is the major target organ in rats, with effects which included increased liver/body weight ratio, hypertrophy and histopathological changes. The no observed adverse effect level (NOAEL) in rats from available short and long term oral studies is 0.5 mg/kg diet, equal to 0.025 mg/kg body weight/day (WHO, 1989a).

When rats were fed 0, 0.1, 1 or 2 mg dieldrin/ kg diet over three generations, no changes in reproductive capacity were observed at any dose level tested (Eisenlord et al., 1967). WHO (1989a) has established 2 mg,dieldrin /kg diet as the NOAEL for reproduction in rats. There was no evidence for teratogenic potential in studies in rats, mice or rabbits using oral doses of up to 6 mg/kg body weight. Abnormal development and fetotoxicity were observed in hamsters and mice with single doses equal to half the LD_{50}, however these results are unlikely to be of significance in view of the maternal toxicity noted at the high dose levels (WHO, 1989a).

IARC (1987b) has concluded that there is inadequate evidence for the carcinogenicity of dieldrin in humans, and limited evidence in experimental animals. Dieldrin is not classifiable as to its carcinogenicity in humans (Group 3).

Table 5.4-1 Acute toxicity of technical dieldrin in mammals.*				
Species	Route	Vehicle	LD_{50} (mg/kg body weight)	Reference
Mouse	oral	corn oil	38	Borgmann *et al.* (1952b)
Rat	oral	various	37-87	Rat oral 37-87 various Lehman (1951); Borgmann *et al.* (1952b); Treon and Cleveland (1955); Gaines (1960); Lu *et al.* (1965); Worthing and Walker (1983)
	dermal	xylene	60-90	Gaines (1960)
Hamster	oral	olive oil	330	Gak *et al.* (1976)
Guinea-pig	oral	corn oil	49	Borgmann *et al.* (1952b)
Rabbit	oral	corn oil	45-50	Borgmann *et al.* (1952b)
	dermal	dimethyl phthalate	150	Lehamn (1952)
Dog		corn oil	65-80	Borgmann *et al.* (1952b)

* Taken from WHO (1989a).

5.4.3.3 Plants

Dieldrin has low phytotoxicity, with tomatoes and cucumber affected only by application rates of greater than 22 kg/ha (Edwards, 1965). Soybean emergence, growth, yield and chemical composition were unaffected by dieldrin applied at 11 kg active ingredient/ ha (Probst and Everly, 1957).

5.4.3.4 Wildlife

The acute toxicity of dieldrin is quite variable for aquatic invertebrates, with insects being the most sensitive group (values\range from 0.2-40 µg/L). It is highly toxic to most species of fish tested in the laboratory (values range from 1.1-41 µg/L).

Schuytema *et al.* (1991) examined the effects of dieldrin in three species of frogs at various growth stages. Acute toxicity (96-h LC_{50}) ranged from 8.7 µg/L for *Rana catesbeiana* tadpoles to 71.3 µg/L for the tadpoles of *Rana pipiens*. Spinal deformities in embryo-larval tests were observed at concentrations as low as 1.3 µg/L for *Xenopus laevis* after a 10 day exposure. *R. pipiens* tadpoles were exposed to 0.8.ug/L dieldrin, and achieved a mean steady state bioconcentration factor (BCF) of 1,130. Depuration in uncontaminated water was rapid with dieldrin concentrations in tissues reaching undetectable levels in 8 days. Bioconcentration factors observed in this study (ranging from 430 to 1,130) are much lower than those observed in freshwater fish exposed to dieldrin (2,385 to 68,286) (US EPA 1980).

Table 5.4-2 Acute toxicity of dieldrin to selected aquatic organisms.*				
Species	Developmental stage, body weight or length	Temp. (°C)	96-h LC_{50} *(µg/L)*	Reference
Daphnia magna	-	-	330[a]	Anderson (1960)
Crangon septemspinosa (Sand shrimp)	0.25 g, 2.6 cm	20	7	Eisler (1969)
Pteronarcys californica (stonefly)	naiad, 3-3.5 cm	15.5	0.5	Sanders and Cope (1968); Johnson and Finley (1980)
Salmo gairdneri (rainbow trout)	1.4 g	13	12	Johnson and Finley (1980)
Pimephales promelas (Fathead minnow)	0.6 g	18	3,8	Johnson and Finley (1980)
Lepomis macrochirus (bluegill)	1.3 g	18	3.1	Johnson and Finley (1980)

[a] 48-h LC_{50} * Taken from WHO (1989a).

The acute toxicity of dieldrin to avian species varies widely (Table 5.4-3). In a long term study, eggs from chickens fed 1 mg dieldrin/kg diet for 2 years showed normal fertility and hatchability, but these parameters were slightly decreased at a level of 10 mg/kg diet (Brown *et al.*, 1965). No significant effect on fertility or hatchability was observed in the eggs of pheasants fed 25 mg dieldrin/ kg diet, but at 50 mg/kg a clear effect was observed (Genelly and Rudd, 1956). Based on the available studies, WHO (1989a) determined that reproduction success was not consistently affected in the absence of maternal toxicity.

In another study, mallard ducklings were exposed to 0, 0.3, 16, 48, 155, 272, and 606 µg/g dieldrin/g diet for 24 days (Nebeker *et al.*, 1992). Complete mortality was observed within 5 days for groups receiving 155, 272 and 6O6 µg/g. A 24 d NOAEL of 0.3j.lg dieldrin/g diet, based on growth impairment, was determined. In the same study birds exposed to 0, 0.014, 0.052 and 0.118 mg dieldrin/L in water, with uncontaminated food, for 34 days did not exhibit toxic effects. BCFs were two orders of magnitude greater in birds exposed to dieldrin through contaminated water than those exposed in the feeding study (1325 vs. 18, respectively).

Data on mammalian species, other than laboratory animals, is limited. The acute LD_{50} of dieldrin to four species of voles range from 100 to 210 mg/kg body weight, suggesting that these microtine rodents are less susceptible than laboratory rodents to dieldrin (Cholakis *et al.*, 1981). In another study, white tailed deer (*Odocoileus virginianus*) were fed 0, 5, or 25 mg dieldrin /kg diet for up to 3 years. Adult survival was not affected, and fertility and *in utero* mortality was comparable for all groups. Fawns from treated does were smaller at birth, experienced greater postpartum mortality and weight gain was reduced (Murphy Korshgen, 1970). Blesbuck (*Damaliscuc dorcas phillipsi*) were fed diets of 5, 15, 25, 35, or 50 mg dieldrin/kg diet for 90 days (Wiese *et al.*, 1973). None of the animals fed 5 or 15 mg/kg diet died during the study period, but all animals at the higher dose levels died within 24 days.

Table 5.4-3 Acute oral toxicity of dieldrin for avian species. *[a]		
Species	LD_{50} (mg/kg body weight)	Reference
Dendocygna bicolor (Fulvous whistling duck)	100-200 (female)	Tucker and Crabtree (1970)
Anas platyrynchos (Mal1ard duck)	381 (female)	Tucker and Crabtree (1970)
Gallus domesticus (Domestic fowl)	43	Sherman and Rosenberg (1953)
Branta canadensis (Canada goose)	50-150	Tucker and Crabtree (1970)
Phasianus colchius (Ring-necked grouse)	79 (female)	Tucker and Crabtree (1970)
Columba livia (pigeon)	26.6	Tucker and Crabtree (1970)

[a].Details concerning age and weight of birds are not summarized here but can be found in the original publications.
* Taken from WHO (1989a).

There is growing evidence linking persistent halogenated aromatic hydrocarbons (especially PCBs and dioxins) to reproductive and immunotoxic effects in wildlife (Fox, 1992; Reijinders and Brasseur, 1992). Although dieldrin has not been directly linked to these effects in wildlife, residues of chlordane have been detected in arctic organisms in conjunction with these compounds (refer to chapter 3 for levels detected).

5.4.4 Persistence/Fate

The half life of dieldrin in temperate soils is approximately 5 years (WHO, 1989a). This persistence, combined with high lipid solubility, provides the necessary conditions for dieldrin to bioconcentrate and biomagnify in organisms. Guppies (*Poecilia reticulata*) exposed to dieldrin concentrations ranging from 0.8 to 4.2 µg/L for 32 days had bioconcentration factors of up to 12,500 (Reinart, 1972). Similarly, sculpins (*Cottus perplexus*) exposed to dieldrin concentrations ranging from 0.017 to 0.86 µg/L for 32 days had bioconcentration factors of up to 13,300 (Chadwick and Brocksen, 1969). Diets containing dieldrin (or aldrin) administered concurrently with exposure to contaminated water did not have a significant effect on dieldrin accumulation, indicating that water is the principle source of dieldrin accumulation i.e. dieldrin is bioconcentrated rather than bioaccumulated (WHO, 1992a).

Dieldrin's chemical properties (low water solubility, high stability, and semi-volatility) favour its long range transport, and dieldrin has been detected in arctic air, water and organisms (Barrie *et al.*, 1992; Lockhart *et al.*, 1992; Thomas *et al.*, 1992; Muir *et al.*, 1992). See ch 3 for a more detailed explanation of this process and levels detected.

5.4.5 Exposure

Dieldrin residues have been detected in air, water, soil, fish, birds and mammals, including humans and human breast milk. In Egypt, the estimated dietary intake of dieldrin by breast fed infants of 1.22 µg/kg body weight day exceeded the FAO/WHO acceptable daily intake of 0.1 µg/kg (Dogheim *et al.*, 1991).

Diet is the main source of exposure to the general public (WHO 1989a). Dieldrin was the second most common pesticide detected in a survey of US pasteurized milk, detected in 172 of the 806 composite samples tested (Trotter and Dickerson, 1993). The highest level detected was 0.003 ppm. Dieldrin residues were detected in 9 of 602 (1.5%) samples of domestic animal fats and eggs tested between 1986 and 1988 in Canada (Frank et at. 1990). The highest residue detected, 0.050 mg/kg in avian broiler fat is, however, lower than the Canadian maximum residue limit of 0.2 mg/kg. Dieldrin was also detected in Spanish meat sampled between January 1989 and December 1991 (Herrerra *et al.*, 1994). Residues of 20 to 40 ppb were detected in the fat of 8 to 15 % of pork products (meat, cured sausage, pork bologna) and in 28 % fresh poultry sausage. Dieldrin residues were detected in Oriental party beans at 3.45 ppb (Gans *et al.*, 1994). Dieldrin residues were detected in 95% of domestic fowl (*Gallus domesticus*) eggs sampled in central Kenya (Mugambi *et al.*, 1989). Although 12% of the eggs of free range birds were without detectable limits, the mean concentration was significantly higher (0.61 mg/kg for free range vs. 0.16 mg/kg for enclosed hens).

The average daily intake of aldrin and dieldrin in India was calculated to be 19 µg/person (Kannan *et al.*, 1992), exceeding the acceptable daily intake 6.0 µg//60 kg of body weight recommended by the Joint FAO/WHO Meeting on Pesticide Residues (JMPR). Dairy products, such as milk and butter, and animal meats were the primary sources of exposure. Exposure through food intake has been estimated at 0.55 µg/person in Vietnam (Kannan *et al.* , 1992a).

5.5 POLYCHLORINATED DIBENZO -P -DIOXINS AND FURANS

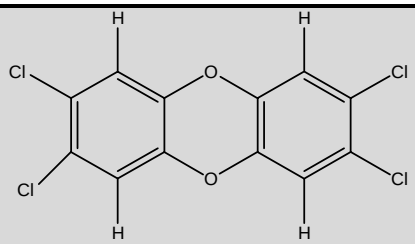
5.5.1 Introduction

Polychlorinated dibenzo-para-dioxins (dioxins) and polychlorinated dibenzofurans (furans) are two groups of planar tricyclic compounds that have very similar chemical structures and properties. They may contain between 1 and 8 chlorine atoms; dioxins have 75 possible positional isomers and furans have 135 positional isomers. They are generally very insoluble in water, are lipophilic and are very persistent. The chemical properties of each of the isomers has not been elucidated, further complicating a discussion of their properties which vary with the number of chlorine atoms present (WHO, 1989c). The most extensively studied dioxin is 2,3,7 ,8-tetrachlorodibenzo-p-dioxin (TCDD).

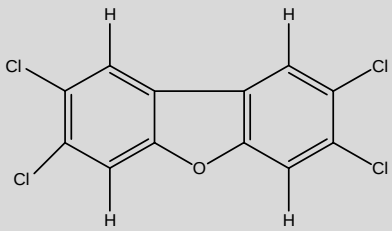
Neither dioxins nor furans are produced commercially, and they have no known use. They are by-products resulting from the production of some other chemicals and may be present as impurities in the end-product or released into the air. Dioxins may be released into the environment through the production of pesticides and other chlorinated substances. Furans are a major contaminant of PCBs. Both dioxins and furans are related to a variety of incineration reactions, combustion and the synthesis and use of a variety of chemical products. Dioxins and furans have been detected in emissions from the incineration of hospital waste, municipal waste, hazardous waste, car emissions, and the incineration of coal, peat and wood (WHO, 1989c).

Of the 210 dioxins and furans, 17 contribute most significantly to the toxicity of complex mixtures. In order to facilitate a comparison of mixtures, International Toxicity Equivalency Factors have been assigned to individual dioxins and furans based on a comparison of toxicity to 2,3,7 ,8-tetrachlorodibenzodioxin. For example, 2,3,7,8- TCDF has been shown to be approximately one-tenth as toxic as 2,3,7,8- TCDD in animal tests, and its toxic equivalent value is 0.1 (Environment Canada, 1993a). TEFs are regarded as risk management tools and they do not necessarily represent actual toxicity with respect to all end points. Rather, they tend to over-estimate the toxicity of mixtures.

5.5.2 Chemical properties



2,3,6,8-TCDD



2,3,7,8-TCDF

Dioxins				
Congener Group	Molecular weight (g/mol)	Vapour Pressure (Pa X 10^{-3})	Water Solubility (mg/m^3)	Log K_{ow}
M_1CDD	218.5	73-75	295-417	4.75-5.00
D_2CDD	253.0	2.47-9.24	3.75-16.7	5.60-5.75
T_3CDD	287.5	1.07	8.41	6.35
T_4CDD	322.0	0.00284-0.272	0.0193-0.55	6.60-7.10
P_5CDD	356.4	0.00423	0.118	7.40
H_6CDD	391.0	0.00145	0.00442	7.80
H_7CDD	425.2	0.000177	0.0024	8.00
O_8CDD	460.0	0.000953	0.000074	8.20

Mackay *et al.* (1992).

Furans				
Congener Group	Molecular weight (g/molecular)	Vapour Pressure (Pa X 10^{-3})	Water Solubility (mg/m^3)	Log K_{ow}
D_2CDF	237.1	14.6	14.5	5.44
T_3CDF	306.0	0.199	0.419	6.1
P_5CDF	340.42	0.0172	0.236	6.5
H_6CDF	374.87	0.0031-0.0036	0.0177-0.0083	7.0
H_7CDF	409.31	0.00054-0.00057	0.00135	7.4
O_8CDF	443.8	0.000101	0.00116	8.0

Mackay *et al.* (1992).

5.5.3 Toxicology

5.5.3.1 Studies in humans

At the present time, the only persistent effect associated with dioxin exposure in humans is chloracne (WHO, 1989c). Other health effects that have been reported include peripheral neuropathies, fatigue, depression, personality change~, hepatitis, enlarged liver, abnormal enzyme levels and porphyria cutanea tarda, though no causal relationships were established in every case (Fingerhut *et al.*, 1991a).

Fingerhut *et al.* (1991b) have studied a subcohort of 1520 workers, within a larger cohort of 5172 workers, known to have been exposed to 2,3,7,8- TCDD for a period of at least one year, and with a latency of at least twenty years between exposure and diagnosis of disease. While the authors did not observe increased mortality related to several cancers previously associated with TCDD exposure (stomach, liver and nasal, Hodgkin's disease and non-Hodgkin's lymphoma), the study did reveal a slightly, but significantly elevated mortality from soft tissue sarcoma and cancers of the respiratory system. As with other studies, interpretation of results was limited by the small number of deaths and by possible confounders including smoking and other occupational exposures.

In contrast, the US Ranch Hand Studies (Roegner *et al.*, 1991), conducted by the US Air Force on veterans who handled and sprayed Agent Orange during the Vietnam War did not demonstrate any association between elevated serum TCDD levels and peripheral neuropathies, fatigue, depression, hepatitis, enlarged liver or porphyria cutanea tarda. Similar results have also been reported in a US NIOSH cohort by Sweeney *et al.*, (1993) who have noted no significant differences in the prevalence of peripheral neuropathies between workers with medium TCDD levels of 220 ppt when compared to controls.

Two recent studies followed a young population from the area of the Seveso, Italy industrial accident. The first, a cancer study (Pesatori *et al.*, 1993), examined a cohort of people aged 0-19 years living in the accident area at the time of the accident, for the period 1977-1986. While a consistent tendency toward increased risk was apparent, none of the relative risks were significantly elevated. Two ovarian cancers were observed, versus one expected, myeloid leukemia showed a clear, but not statistically significant increase, and a nonsignificant increase in thyroid cancer was observed. The study is limited, however, by the relatively short latency periods, the definition of exposure based on place of residence and the limited number of events. The second study examined the mortality of the same cohort of people for the same time period (Bertazzi *et al.*, 1992). Among the exposed, mortality from all causes did not deviate from expectations; however, as noted above, this study provides only limited evidence.

Direct exposure of humans to furans has been reported in two incidents of rice oil, contaminated with very high doses of PCDFs, in Japan (Yusho) and Taiwan (Yucheng). While it is possible that the effects observed in these incidents (see section 4.9 on PCBs) may be due to the very high doses and/or presence of furans, the similarity of structure, effects and mode of action of PCBs and PCDFs (Poland and Knutson, 1982) precludes a definite conclusion on the causative agent.

5.5.3.2 Studies in laboratory animals

The acute oral toxicity in laboratory animals is highly variable (Table 5.5-1). Effects of dioxin exposure that are common to most, and sometimes all, species include wasting, lymphoid involution, hepatotoxicity, chloracne and epidermal changes, and gastric lesions. Other characteristic responses include edema, ascites and hypopericardium in chickens; fetal death and resorption in rats and fetal wastage, embryotoxicity and malformations in mice (Poland and Knutson, 1982).

Table 5.5-1 Single lethal dose values for TCDD*

Species/strain	Age/weight	Route/vehicle	Time to death (days)	LD_{50} (μg/kg)	Reference
Rat/Sherman	Not Reported	oral/corn oil: acetone (9:1)	9-27	22	Schwetz *et al.*(1973)
Mice/C57BL/6	3 mo/23.6-30.8 g	oral/corn oil: acetone (9:1)	15-30	114	Vos *et al.* (1973)
Guinea pig/Hartley	Not Reported	oral/corn oil	5-34	0.6	Schwetz *et al.*(1973)
Hamster/Golden Syrian	Not Reported /50-80 g	oral/olive oil	2-47	1157	Olson *et al.* (1980)

* Adapted from WHO (1989c).

A three-generation study was conducted in which rats were fed diets containing 2,3,7,8- TCDD that maintained dose levels of 0, 0.001, 0.01 or 0.1 μg TCDD/kg/day (Murray *et al.*, 1979). No significant toxicity was observed in the f_o rats during the 90 days prior to mating. Significant decreases in fertility and neonatal survival were observed in the f_o group receiving 0.1 μg TCDD/kg/day, effectively halting continuation of this dose level in subsequent generations. At 0.01 μg TCDD/kg/day, fertility was significantly reduced in the f_1 and f_2 generations. Decreases in litter size, gestation survival and neonatal survival and growth were also observed at this dose level. No effect on fertility, litter size at birth or post natal body weight was observed in any generation of the 0.001 μg TCDD/kg/day group.

Some teratogenic effects have been observed in mice in association with dioxin and furan exposure (Birnbaum *et al.*, 1987) including hydronephrosis and cleft palate. The most potent teratogenic isomer was 2,3,4,7 ,8-pentachlorodibenwfuran, with an ED_{50} of 36 μg/kg for cleft palate and 7 μg/kg for hydronephrosis. Teratogenic responses observed are similar to those seen with TCDD, but these compounds are only 1/10 to 1/100 as potent.

Dioxins, specifically 2,3,7,8- TCDD, have been reported to be associated with a variety of adverse effects on the reproductive systems of both male and female rats (Peterson *et al.*, 1992). Male reproductive toxicity has included altered regulation of luteinizing hormone secretion, reduced testicular steroidogenesis, reduced plasma androgen concentrations, reduced testis and accessory sex organ weights, abnormal testis morphology, decreased spermatogenesis, and reduced fertility. Signs of female reproductive toxicity included hormonal irregularities in the oestrous cycle, reduced litter size and reduced fertility.

The immunotoxicity of 2,3,7,8- TCDD has been extensively studied. A review of recent literature concerning 2,3,7,8- TCDD effects on immunocompetence suggests that 2,3,7,8- TCDD either indirectly (in the case of T -cells) or directly (in the case of B-cells) affects the maturational or differentiation processes of immunocompetent cells (Holsapple *et al.*, 1991). Studies in exposed human populations and in non-human primates have shown that halogenated aromatic hydrocarbons produce measurable alterations in both innate and acquired immunity, although significant deficits in immunocompetence have not been conclusively associated with these changes (Holsapple *et al.*, 1991).

IARC (1987c) has concluded that while there is inadequate evidence for the carcinogenicity of 2,3,7,8- TCDD in humans, there is sufficient evidence in experimental animals. IARC has classified 2,3,7,8- TCDD as a possible human carcinogen (Group 2B). Other chlorinated dibenzodioxins (other than 2,3,7,8- TCDD) are deemed not classifiable as to their carcinogenicity in humans (IARC, 1977). Similarly, Roegner and coworkers (1994) have been unable to detect TCDD related physiologic abnormalities of the immune system.

5.5.3.3 Plants

No data were available.

5.5.3.4 Wildlife

Exposure of fish to dioxins and furans results in a delayed mortality that can continue many days post-exposure. Mehrle *et al.* (1988) exposed rainbow trout to 2,3,7,8- TCDD at concentrations of 0, 38, 79, 176, 382 and 789 pg TCDD/L (parts per quadrillion) and to 2,3,7,8-TCDF at concentrations of 0, 0.41,0.90, 1.79,3.93 and 8.78 ng TCDF/L (parts per trillion) for 28 days, followed by a 28 day depuration period. A 56-day LC_{50} of 46 pg/L was calculated for TCDD, and the NOEC based on growth and mortality was below the lowest exposure concentration of 38 pg/L. The 56-day NOEC for TCDF was calculated to be 1.79 ng/L for mortality and 0.41 ng/L for growth. Mortality continued after the 28 day exposure period ended, and behaviour also continued to be affected. Changes in behaviour included lethargic swimming, feeding inhibition and lack of response to external stimuli.

Early life stages of fish are very sensitive to the effects of dioxins, furans, and PCBs (Walker and Peterson, 1992). Parts per trillion concentrations of these structurally related chemicals in lake trout and rainbow trout eggs exhibit toxicity through sac fry mortality associated with yolk sac edema and haemorrhages.

Hart *et al.* (1991) examined the relationship between concentrations of PCDDs and PCDFs in great blue heron eggs and the effects on chicks. Eggs were collected from sites of low, intermediate and high contamination. Levels of 2,3,7,8- TCDD in eggs were 10 ng/kg (wet weight), 135 ng/kg and 211 ng/kg, respectively. There was little difference in mortality of chicks from eggs collected from the various sites, suggesting no effect on survival at levels of PCDD/PCDFs seen in the eggs. Effects of contamination included decreased growth with increased TCDD level, depression of skeletal growth with increased TCDD levels and subcutaneous edema which increased with increasing PCDD and PCDF contamination. Also observed were shortened beaks and a scarcity of down follicles in the chicks from the more contaminated sites.

Mink were administered a single dose of 2,3,7,8- TCDD at concentrations of 0, 2.5, 5.0 and 7.5 μg/kg body weight and observed for 28 days (Hochstein *et al.*, 1988). Mink administered the high doses of TCDD experienced the wasting syndrome associated with TCDD intoxication, and gastric lesions were observed at these doses. The 28 day oral LD_{50} in mink administered a single dose of TCDD was calculated to be 4.2 μg TCDD/kg body weight.

5.5.4 Persistence/fate

Dioxins and furans are considered to be very stable and persistent, as illustrated by the half life of TCDD in soil of 10-12 years (WHO, 1989c). This persistence, combined with high partition coefficients (up to 7.10 for TCDD) provides the necessary conditions for these compounds to bioconcentrate in organisms. Rainbow trout (*Salmo gairdneri*) exposed to 2,3,7,8- TCDD for 28 days resulted in an average BCF of 26,707. Those fish exposed to 2,3,7,8- TCDF at a concentration of 0.41 ng/L for 28 days had an average BCF of 6,049 (Mehrle *et al.*, 1988).

The chemical properties of dioxins and furans (low water solubility, high stability and semivolatility) favour their long range transport and these compound have been detected in arctic organisms (Norstrom *et al.*, 1990). See Chapter 3 for a more detailed explanation of this process and levels detected.

5.5.5 Exposure

As with most other organochlorines, food is a major source of exposure to dioxins and furans in the general population, with food of animal origin contributing the most to human body burdens. In a survey of dioxins in US food, total PCDD/Fs ranged from 0.42 to 3.42 ppt (wet weight) (total TEQ range: 0.02 to 0.13 ppt) in fish; 0.8 to 61.8 ppt (total TEQ range: 0.3 to 1.5 ppt) in meats and 0.9 to 19 ppt (TEQ range: 0.04 to 0.7 ppt) in dairy products (Schecter *et al.*, 1994). The estimated daily intake for adults ranged from 0.3 to 3.0 pg TEQS/kg body weight, and for breast fed infants the range was 35.3 to 52.6 pg TEQS/kg body weight. A survey in Ontario, Canada (Birmingham *et al.*, 1989) found that many samples of foods had non-detectable levels of most PCDD/F congeners, however the average adult daily intake was estimated to be 1.52 pg TEQ/kg body weight. A 1992 survey from Germany estimated a daily intake of 2 pg TEQS/kg body weight/ day identified in a broad range of food samples including meat, fish, dairy products, fruits and vegetables (Beck *et al.*, 1992). A study in the Netherlands estimated a median daily intake of 1 pg TEQ/kg body weight (Theelen *et al.*, 1993). These are below the TDI of 10 pg/kg body weight for lifetime exposure estimated by WHO (Ahlborg *et al.*, 1992).

5.6 ENDRIN

5.6.1 Introduction'

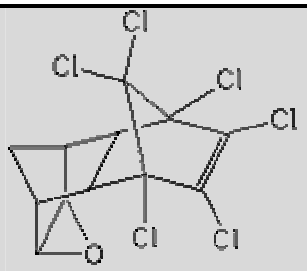
Endrin is the *endo, endo* stereoisomer of dieldrin and was first registered for use in the USA in 1952 (Smith, 1991). Technical endrin has a purity of at least 92% (WHO, 1992a). Endrin is rapidly metabolised by animals and does not accumulate in fat to the same extent as other compounds with similar structures. It can enter the atmosphere by volatilization, and can contaminate surface water from soil run-off (WHO, 1992a).

Endrin is produced by the condensation of vinyl chloride with hexachlorocyclopentadiene, dehydrochlorinating the adduct and subsequent reaction with cyclopentadiene to form isodrin, which is epoxidized by peracetic or perbenzoic acid (WHO, 1992a). Endrin is a foliar insecticide used mainly on field crops such as cotton and grains. It has also been used as a rodenticide to control mice and voles (Smith, 1991).

Endrin is banned in many countries, including Belgium, Cyprus, Ecuador, Finland, Israel, Philippines, Singapore, Thailand and Togo. Its use is severely restricted in many countries, including Argentina, Canada, Chile, Colombia, the EU, India, Japan, New Zealand, Pakistan, USA, and Venezuela (Gips, 1987).

5.6.2 Chemical properties

CAS Chemical Name: 3,4,5,6,9, 9-Hexachlorodimetanonapth[2,3-*b*]oxirene.



Synonyms and Trade Names (partial list): Compound 269, Endrex, Hexadrin, Isodrin Epoxide, Mendrin, Nendrin.

CAS No.: 72-20-8; molecular formula: $C_{12}H_8Cl_6O$; formula weight: 380.92

Appearance: White, Odourless, crystalline solid when pure; light tan colour with faint chemical odour for technical grade

Properties: Melting point: 200°C; boiling point: 245°C (decomposes); K_H: 5.0 x 10^{-7} atm m^3/moleculare; log K_{ow}: 3.209-5.339; solubility in water: 220-260 μg/L at 25°C; vapour pressure: 7 x 10^{-7} mm Hg at 25°C. (source: Montgomery, 1993).

5.6.3 Toxicology

5.6.3.1 Studies in humans

Endrin is toxic to humans; the estimated lethal dose is approximately 6 g (Reddy *et al.*, 1966), equivalent to approximately 100 mg/kg body weight. Symptoms of mild intoxication include dizziness, weakness of the legs, abdominal discomfort and nausea but usually not vomiting. More severe poisoning results in repeated, violent epileptiform convulsions lasting several minutes, followed by semiconsciousness or coma (Smith, 1991).

A study of 241 workers from a Dutch plant involved in the production of aldrin, dieldrin and endrin did not find endrin in the blood of workers, except in cases of accidental, acute over-exposure (Versteeg and Jager, 1973) These findings are in agreement with results of a study of 71 workers in an endrin plant in the USA (Hayes and Curley, 1968). Data on absenteeism, results of liver function tests, blood chemistry, blood morphology, urine analysis, occurrence of sensitization, the incidence and pattern of diseases including the occurrence of malignant growth showed no difference between workers exposed to endrin and other chemical plant operators.

Brown (1992) studied workers from a plant involved in the manufacture of aldrin, dieldrin and endrin. The study group consisted of white males who had been employed at the plant for at least six months prior to December 31, 1964. A statistically significant increase in liver and biliary tract cancers was observed. However, the author notes that when interpreting the results, several factors must be considered; (1) there is a lack of quantitative information on exposure to the pesticides, (2) a known animal carcinogen, dibromochloropropane (DBCP), was manufactured at the plant between 1955 and 1976, and therefore is a potential confounding exposure, (3) the liver cancers were not homogenous, but were a mixture of extrahepatic and intrahepatic tumours, in contrast to studies in experimental animals which resulted in intrahepatic tumours and (4)there does not appear to be a dose-response relationship when dose is measured as length of employment.

Some organochlorines have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alterations in immune function (Holsapple et al., 1991). There is also limited evidence that cyclodienes such as endrin may also affect immune responses (Exon et al., 1987). Some organochlorines, such as DDE, may have weak estrogenic properties, and some authors have suggested a possible role in estrogen receptor positive breast cancer (Wolff et al., 1993) while other authors have been unable to demonstrate such an effect for DDT or its metabolites (Krieger et al., 1994). Halogenated aromatic hydrocarbons are also known to affect endocrine function and reproductive systems (Peterson et al., 1992; Gray, 1992; Thomas and Colborn, 1992). Although endrin itself has not been directly linked these effects per se, the similarity of structure and chemical properties shared by halogenated aromatic hydrocarbons suggests a basis for further investigation.

5.6.3.2 Studies in laboratory animals

The acute toxicity for formulated endrin to rats as a 50% wettable powder was 7.6 mg/kg body weight (3.80 mg active material/kg body weight), and the dermal LD_{50} for the same formulation was 21.80 mg/kg body weight (dry) (10.90 mg active material/kg body weight) (Muir, 1970).

Male and female Long-Evans rats were fed endrin in the diet at 0, 0.1, 1, or 3 mg/kg diet over three generations. No difference in appearance, behaviour, body weight, or number or size of litters was observed. The weights of liver, kidneys and brain were normal, and no histopathological abnormalities were observed in third generation weanlings. Significant increased mortality of pups

in the second and third generations of rats fed 3 mg/kg was noted (Hine, 1965). Endrin was not teratogenic at levels that did not cause maternal toxicity (Smith, 1991).

Endrin is metabolised rapidly by animals, and very little is accumulated in fat compared to compounds of similar structure (including its stereoisomer dieldrin). The formation of *anti*-12-hydroxyendrin is considered to be the major route of metabolism of endrin (WHO, 1992a).

IARC (1974) has concluded that there is inadequate evidence for the carcinogenicity of endrin in humans, and there is only limited evidence in experimental animals. Endrin is therefore not classifiable as to its carcinogenicity in humans (Group 3).

Table 5.6-1 Acute toxicity of technical grade endrin in mammals.*

Species	Route	Vehicle	LD_{50} (mg/kg body weight) males	females	Reference
Mouse	oral	unknown	13	13	Gray *et al.* (1981)
Rat	oral	unknown	4	4	Gray *et al.* (1981)
	dermal	xylene	18	15	Gaines (1960,1969)
Guinea pig	oral	peanut oil	36.0	16.0	Treon *et al.* (1955)
Rabbit	oral	peanut oil	-	7-10	Treon *et al.* (1955)
	dermal	none	-	Min. lethal dose: 60-94	Treon *et al.* (1955)
Hamster	oral	unknown	18	18	Gray *et al.* (1981)
Monkey (*Macacus mulatta*)	oral	peanut oil	3	-	Treon *et al.* (1955)

* Taken from WHO (1992a).

5.6.3.3 Plants

No data were available.

5.6.3.4 Wildlife

The toxicity of endrin to selected aquatic organisms is summarized in table 4.6-2. Endrin is highly toxic to fish, with most LC_{50} values below 1.0 µg/L (WHO, 1992a). Sheepshead minnows were exposed to endrin concentrations of 0, 0.027, 0.077, 0.12, 0.31 and 0.72 µg/L for 23 weeks (Hansen *et al.*, 1977). Embryos exposed to 0.31 and 0.72 µg/L hatched early, and all those exposed to 0.72 µg/L died by the ninth day of their exposure, while those exposed at 0.31 µg/L were initially stunted and some died. The reproductive ability of the survivors of the 0.31 µg/L was impaired. No significant effects were observed at an exposure concentration of 0.12 µg/L The lowest observed adverse effect level (LOAEL) for aquatic organisms was 30 ng/L over 20 days for reproduction in mysid shrimp (*Mysidopsis bahia*) (McKenney, 1986).

Table 5.6-2 Acute toxicity of endrin to selected aquatic organisms.*

Organism	Size/age	Temp. (°C)	96-h LC_{50}	Reference
Freshwater				
Daphnia magna *(water flea)*		22.24	59 mg/L	Elnabaraway *et al.* (1986)
Palaemonetes kadiakensis (glass shrimp)	Adult	21	0.5 mg/L	Mayer and Ellersieck (1986)
Pteronarcys californica (stonefly)	Larvae	15	0.25 mg/L	Mayer and Ellersieck (1986)
Oncorhynchus mykiss (rainbow trout)	1.4 g	18	0.75 µg/L	Mayer and Ellersieck (1986)
Pimephales promelas (fathead minnow)	1.2 g	18	1.8 µg/L	Mayer and Ellersieck (1986)
Lepomis macrochirus (bluegill)	1.5 g	18	0.61 µg/L	Mayer and Ellersieck (1986)
Estuarine/Marine				
Penaeus duorarum (pink shrimp)	adult	17	0.037 µg/L	Mayer (1987)
Pagurus longicarpus (hermit crab)	-	-	1.2 µg/L	Eisler (1970a)
Cyprinodon variegatus (sheepshead minnow)	adult	18	0.38 µg/L	Mayer (1987)
Mugil cephalus (striped mullet)	83 mm	20	0.3 µg/L	Eisler (1970b)

* Taken from WHO (1992a).

The toxicity of endrin to some terrestrial species is given in table 4.6-3. Male and female mallard ducks were fed diets containing 0, 0.5 or 3.0 mg/kg during their oviposition period (Roylance *et al.*, 1985). Reproduction (*i.e.* fertility, embryo survival and hatchability) was not impaired by any of the dose levels tested.

Table 5.6-3 Acute oral toxicity of endrin to selected terrestrial species.*

Species	LD_{50} (mg/kg body weight)	Reference
Anas platyrhynchos (mallard)	5.6(2.7-11.7)	Hudson *et al.* (1984)
Columbia livia (pigeon)	2.0-5.0	Hudson *et al.* (1984)
Phasianus colchicus (pheasant)	1.0(1.1-2.8)	Hudson *et al.* (1984)
Eptesicus fuscus (big brown bat)	5-8	Luckens and Davis (1965)
Microtus pitymis pinetorum (pine mouse)	2.6 1.3	Petrella *et al.* (1975) Webb *et al.* (1973)

* Taken from WHO (l992a).

5.6.4 Persistence/fate

The half life of endrin in soil may be up to 12 years, depending on local conditions. This persistence, combined with a high partition coefficient (log K_{ow} = 3.21-5.340), provides the necessary conditions for endrin to bioconcentrate in organisms. Sheepshead minnows were exposed to endrin at levels of 0.027-0.72 µg/L from embryonic stage through adulthood. At adulthood, they had accumulated 6400 times the concentration in the water (Hansen *et al.*, 1977). Bluegill sunfish exposed to water containing ^{14}C-labelled endrin at 1 g/L took up 91 % of the radio-labelled endrin with in 48 hours (Sundershan and Kahn, 1980), with a half life of loss from the tissues of approximately four weeks. *Leiostomus xantharus* exposed to 0.05 µg/L for 5 months had a tissue residue level of 78 µg/kg tissue (Lowe, 1966). After 18 days in uncontaminated water, no residues were detected, suggesting that endrin disappears rapidly from this organism.

The chemical properties of endrin (low water solubility, high stability in the environment, and semi-volatility) favour its long range transport, and it has been detected in arctic freshwater (Lockhart *et al.*, 1992).

5.6.5 Exposure

The main source of endrin exposure to the general population is residues in food, contemporary intake is generally below the acceptable daily intake of 0.002 mg/kg body weight recommended by the Joint FAO/WHO Meeting on Pesticide Residues (JMPR). Recent food surveys have generally not included endrin, and hence recent monitoring data are not available.

5.7 HEXACHLOROBENZENE

5.7.1 Introduction

Hexachlorobenzene (HCB) is a fungicide that was first introduced in 1945 for seed treatment, especially for control of bunt of wheat (Edwards et at., 1991). HCB is also a byproduct of the manufacture of industrial chemicals including carbon tetrachloride, perchlorethylene, trichloroethylene and pentachlorobenzene. It is a known impurity in several pesticide formulations, including pentachlorophenol and dicloram and may be present as an impurity in others (Tobin, 1986).

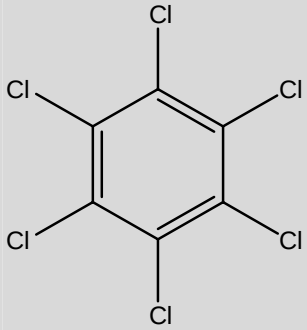
HCB is highly insoluble in water, and is soluble in organic solvents. It is quite volatile and can be expected to partition into the atmosphere as a result. It is very resistant to breakdown, has a high partition coefficient (log K_{ow} = 3.03-6.42), and is known to bioconcentrate in the fat of living organisms as a result.

HCB is formed during the liquid phase substitution reaction of chlorine and benzene, with a ferric oxide catalyst, at temperatures greater than 150°C. It can also be produced from the unwanted stereoisomer of hexachlorocyclohexane (those not needed in lindane manufacture) by treatment with metal chlorides or anhydrous sulfuric acid (Vancouver Proceedings).

HCB is banned in Austria, Belgium, Czechoslovakia, Denmark, the EU, Germany, Hungary, Liechtenstein, Netherlands, Panama, Switzerland, Turkey, United Kingdom and the USSR. It is severely restricted or has been voluntarily withdrawn in Argentina, New Zealand, Norway and Sweden (Environment Canada, 1995). Countries other than those listed above may also ba11, or severely restrict the use of hexachlorobenzene.

5.7.2 Chemical properties

CAS Chemical Name: Hexachlorobenzene



Trade Names (partial list): Amaticin, Anticarie, Buntcure, Bunt-no-more, Co-op hexa, Granox, No bunt, Sanocide, Smut-go, Sniecotox.

CAS No.: 118-74-1; molecular formula: C_6Cl_6; formula weight: 284.78.

Appearance: White monoclinic crystals or crystalline solid.

Properties: Melting point: 227-230°C; boiling point: 323-326°C (sublimes); K_H: 7.1 x 10^{-3} atm m^3/mol at 20°C; log K_{OC}: 2.56-4.54; log K_{ow}: 3.03-6.42; solubility in water: 40 μg/L at 20°C; vapour pressure: 1.089 x 10^{-5} mm Hg at 20°C.

5.7.3 Toxicology

5.7.3.1 Studies in humans

The most notable episode involving the effects of HCB on humans involved the ingestion of HCB treated seed grain in eastern Turkey between 1954 and 1959. Approximately 3000-4000 people who ingested the treated seed developed porphyria turcica, a disorder of haem biosynthesis (Peters *et al.*, 1986). In laboratory animals, HCB has been reported to induce porphyria cutanea tarda, resulting from a HCB mediated decrease in the activity of uroporphyrinogen decarboxylase in the liver. Although this decrease has not been observed in the human disorder, the presence of a decrease has been suggested by the occurrence of the typical porphyrin excretion pattern associated with this enzyme defect (Elder, 1986).

The patients who ingested the treated seed experienced a range of symptoms including photosensitive skin lesions, hyperpigmentation, hirsutism, colic, severe weakness, porphyrinuria, and debilitation. Mortality was up to 14%. Mothers who ingested the seeds passed the HCB to their children by placental transfer and through maternal milk (Peters *et al.*, 1986). Children born to these women developed "pembe yara" or pink sore, with a reported mortality rate of approximately 95% (Edwards *et al.*, 1991).

A study of 32 individuals twenty years after the outbreak showed that porphyria can persist years after the ingestion of HCB, although as individuals who were known to still have symptoms were chosen for re-examination, it was not possible to determine the frequency of the persistent effects (Edwards *et al.*, 1991).

A small cross-sectional study of workers exposed to HCB (Currier *et al.*, 1980) did not find any evidence of cutaneous porphyria or any other adverse effects associated with exposure of 1 to 4 years.

5.7.3.2 Studies in laboratory animals

HCB has a very low acute toxicity (Table 5.7-1). Porphyria, skin lesions, hyperexcitability and changes in weight, enzyme activities and morphology of the liver have been reported in association with subchronic toxicity of HCB (Strik, 1986). HCB has also been reported to stimulate the immune system in rats, and suppress the immune system of mice (Vos, 1986).

HCB has been reported to have adverse effects on reproduction and reproductive tissue. Female rats were fed 0, 60, 80, 100, 120 and 140 ppm HCB in the diet (Kitchin *et al.*, 1982). While no effects on fertility or fecundity were observed, treatment did cause mortality in the offspring, with a 21 day LD_{50} of 100 ppm. A four generation reproduction study in rats fed 0, 10,20,40, 80, 160,320 and 640 pp, HCB in the diet was conducted (Grant *et al.*, 1977). No gross abnormalities were observed, but HCB did affect reproduction by reducing the number of litters whelped, litter size and the number of pups surviving to weaning. HCB at a concentration of 100 mg/kg body weight/day was associated with cleft palate and some kidney malformations in CD-1 mice (Courtney *et al.*, 1976).

Oral exposure of female cynomolgus monkeys to 0.1 mg HCB/kg body weight/day for 90 days caused degenerative changes in the ovarian surface epithelium (Babineau *et al.*, 1991).

Foster *et al.* (1992a) found that HCB induced a dose dependent suppression of serum progesterone in cynomologus monkeys, and concluded that HCB interferes with mechanisms regulating ovarian steroidogenesis. Exposure of rhesus monkeys to HCB at concentrations of 8, 32, 64 and 128 mg/kg body weight/day for 60 days resulted in a dose dependent atrophy of thymic cortex and a reduction in the number of lymphocytes (Iatropoulos *et al.*, 1976). Dose dependent degenerative changes in the ovaries and kidney and degenerative changes in the liver compatible with porphyria tarda were also observed.

IARC (1987d) has concluded that while there is inadequate evidence for the carcinogenicity of HCB in humans, there is sufficient evidence in experimental animals. IARC has classified HCB as a possible human carcinogen (Group 2B).

Table 5.7-1 Acute toxicity of HCB to mammals.*

Species	Route	LD_{50} (mg/kg body weight)	Reference
Rat	Oral	3 500	Savitskii (1964)
Mouse	Oral	4 000	Savitskii (1964)
Rabbit	Oral	>2 600	Savitskii (1964)
	dermal	>2 000	unpublished

* Taken from Strik (1986).

5.7.3.3 Plants

No data were available.

5.7.3.4 Wildlife

HCB is unlikely to cause direct toxicological effects in aquatic animals at or below saturation concentrations (approximately 5 µg/L) in water (Carlson and Kosian, 1987). Earlier studies have observed effects at concentrations higher than saturation, by using co-solvents. At an exposure concentration of 4.8 µg HCB/L for 32 days, there was no observed effect on embryonic through juvenile stages in developing fathead minnows (*Pimephales promelas*) giving a NOEC of 4.8 µg/L (Carlson and Kosian, 1987).

Nebeker *et al.* (1989) exposed the caldoceran *Daphnia* magna, the amphipods *Hyalella* azeteca, and *Gammarus lacustris*, the annelid worm *Lumbricus variegatus* and the fathead minnow *Pimephales promelas* to HCB at saturation concentration (5 µg/L) for 68 days. No effects on survival, growth or reproduction were observed.

Calamari *et al.* (1983) examined the effects of HCB on two species of fish, *Salmo gairdneri* and *Brachydanio rerio* and a crustacean *Daphnia magna*. HCB was not acutely toxic to any of the organisms at the saturation concentration of 0.03 mg/L. A 14 day EC_{50} for reduced fertility in *Daphnia magna* was 0.016 mg/L.

Adult Japanese quail (*Coturnix japonica*) were fed diets containing HCB for 90 days, resulting in increased mortality at 100 µg/g diet and hatchability of eggs was significantly reduced at 20 µg/g. At 5 µg/g increased liver weight, slight liver damage and increased faecal excretion of coproporphyrin were observed (Vos *et al.*, 1971; 1972).

Bleavins *et al.* (1984) exposed mink (*Mustela vison*) and European ferrets (*Mustela putorius furo*) to diets containing 1, 5, 25, 125 or 625 mg HCB/kg diet. The two highest levels were lethal to adults of both species. Adverse reproductive effects observed at lower doses included decreased litter size, increased percentage of stillbirths, increased kit mortality and decreased kit growth. These effects were seen in both species, although usually at higher levels in the ferrets. A second experiment involved the cross-fostering of kits born to untreated dams to females fed a diet containing 2.5 mg HCB/kg diet, and *vice versa*. Results from this experiment indicated that *in utero* exposure to HCB resulted in higher kit mortality than exposure via the mothers milk.

There is also growing evidence linking persistent halogenated aromatic hydrocarbons (especially PCBs and dioxins) to immunotoxic effects in wildlife (Fox, 1992; Reijinders and Brasseur, 1992). Although HCB has not been directly linked to these effects in wildlife, residues of HCB have been detected in arctic organisms in conjunction with these compounds (refer to chapter 3 for levels detected).

5.7.4 Persistence/fate

HCB is very persistent. Estimated half lives in soil from aerobic and anaerobic degradation range from 2.7 to 22.9 years (Environment Canada, 1993). This persistence, combined with a high partition coefficient (log K_{ow} = 3.03-6.42), provides the necessary conditions for HCB to bioconcentrate in organisms. Fathead minnows (*Pimephales promelas*) exposed to HCB at 4.8 µg/L for 32 days had a bioconcentration factor of 22,000 (Carlson and Kosian, 1987). Worms (*Lumbricus variegatus*) exposed to HCB at 1.2 µg/L for 68 days had a BCF of 106,840 (Nebeker *et al.*, 1989).

The chemical properties of HCB (low water solubility, high stability, and semi-volatility) favour its long range transport, and HCB has been detected in arctic air, water and organisms (Barrie *et al.*, 1992; Lockhart *et al.*, 1992; Thomas *et al.*, 1992; Muir *et al.*, 1992). See Chapter 3 for a more detailed explanation of this process and levels detected.

5.7.5 Exposure

HCB is ubiquitous in the environment, and has been measured in foods of all types. HC$ as one of two organochlorines detected in all samples of Spanish meat and meat products surveyed between January 1989 and December 1991 (Herrera *et al.*, 1994). Mean levels ranged from 8 ppb (fat weight) in pork products (cured ham) to 49 ppb in lamb, with a maximum level of 178 ppb in lamb. HCB was detected in 13 of 241 serum samples from Colorado beef cattle in a monitoring program, with an average concentration of 3.1 ppb (Salman *et al.*, 1990). In a survey of fat from domestic farm animals in Ontario, Canada, HCB residues were below detection limits (0.1 mg/kg fat) in all samples analysed (Frank *et al.*, 1990). A survey of US pasteurized milk detected HCB in 8 of 806 composite milk samples (Trotter and Dickerson, 1993). A survey of foods from India found average concentrations of HCB ranging from 1.5 ng/g (fat weight) in both oils and milk to 9.1 ng/g in fish and prawns, with a maximum concentration of 28 ng/g in fish and prawns (Kannan *et al.*, 1992b). The average daily intake was calculated to be 0.13 µg/person. Average HCB residues in foods from Vietnam ranged from 0.28 ng/g (fat weight) in pulses to 27 ng/g in caviar (Kannan *et al.*, 1992a). The daily intake was estimated at 0.10 µg/person/day.

5.8 HEPTACHLOR

5.8.1 Introduction

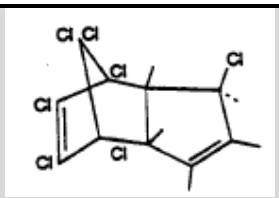
Heptachlor was isolated from technical chlordane in 1946, and introduced as a commercial insecticide in the USA in 1952 (WHO, 1984b). Technical-grade heptachlor contains approxin1ately 72% heptachlor and 28% related compounds, including about 20% chlordane (!ARC, 1991a). It is highly insoluble in water, and is soluble in organic solvents. It is quite volatile and can be expected to partition into the atmosphere as a result. It binds readily to aquatic sediments and bioconcentrates in the fat of living organisms. Heptachlor is metabolised in animals to heptachlor epoxide, the toxicity of which is similar to that of heptachlor, and which may also be stored in animal fat.

The synthesis of heptachlor is similar to that of chlordane; hexachlorocyclopentadiene is reacted with cyclopentadiene to form chlordane. Heptachlor is prepared by the free-radical chlorination of chlordane (IARC, 1991a).

Heptachlor is a non-systemic stomach and contact insecticide, used primarily against soil insects and termites (WHO, 1984b). It has been used against cotton insects, grasshoppers, and some crop pests (Smith, 1991). It has also been used to combat malaria (IARC, 1991a).

Heptachlor is banned in many countries, including Cyprus, Ecuador, EU, Portugal, Singapore, Sweden, Switzerland and Turkey. It is severely restricted in other countries including, Argentina, Austria, Canada, Czechoslovakia, Denmark, Finland, Israel, Japan, New Zealand, Philippines, USA and the USSR. Countries other than those listed above may prohibit or severely restrict the use of heptachlor.

5.8.2 Chemical properties



CAS Chemical Name: 1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7.mehtanol-1*H*-indene.

Synonyms and Trade Names (partial list): Aahepta, Agroceres, Baskalor, Drinox, Drinox H-34, Heptachlorane, Heptagran, Heptagranox, Heptamak, Heptamul, Heptasol, Heptox, Soleptax, Rhodiachlor, Veliscol 104, Veliscol heptachlor.

CAS No.: 76-44-8; molecular formula: $C_{10}H_5Cl_7$; formula weight: 373.32.

Appearance: White to light tan, waxy solid or crystals with a camphor-like odour.

Properties: Melting point: 95-96°C (pure); 46-74°C (technical); boiling point: 135-145°C at 1-1.5 mm Hg; decomposes at 760 mm Hg; K_H: 2.3 x 10^{-3} atm mm^3/mol; log K_{OC}: 4.38; log K_{ow}: 4.40-5.5; solubility in water: 180 ppb at 25°C; vapor pressure: 3 x 10^{-4} mm Hg at 20°C. (source: Montgomery, 1993).

5.8.3 Toxicology

5.8.3.1 Studies in humans

There is no information on accidental or suicidal intoxication by heptachlor in humans (WHO. 1984b). Symptoms in animals include tremors and convulsions (Montgomery, 1993).

A study of workers from a plant involved in the production of heptachlor and endrin found a significant increase in bladder cancer (Brown, 1992). This result was unexpected as no know bladder carcinogens were used at the plant, however, the small number of deaths (3) makes interpretation of these findings difficult. No deaths from liver or biliary tract cancer were observed, although mortality from cerebrovascular disease was higher than expected.

Some organochlorines such as dioxins have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alterations in immune function (Holsapple *et al.*, 1991). There is also limited evidence that cyclodienes such as heptachlor may also affect immune responses (Exon *et al.*, 1987). Some organochlorines, such as DDE, may have weak estrogenic properties, and some authors have suggested a possible role in estrogen receptor positive breast cancer (Wolff *et al.*, 1993) while others have been unable to demonstrate such a role for DDT or its metabolites (Krieger, 1994). Although heptachlor itself has not been directly linked to these effects *per se*, some halogenated aromatic hydrocarbons are also known to affect endocrine function and reproductive systems (Peterson *et al.*, 1992; Gray, 1992; Thomas and Colborn, 1992). This requires further investigation of such possible outcomes.

5.8.3.2 Studies in laboratory animals

Acute toxicity of heptachlor to mammals is given in Table 5.8-1

Groups of male and female rats were administered daily doses of heptach1or orally at 0, 5, 50 or 100 mg/kg body weight, beginning at 4 months of age, and continuing for 200 days (Pelikan *et al.*, 1968). All the animals in the 50 and 100 mg/kg groups died by the 10th day of exposure. Three animals in the 5 mg/kg group and 1 in the control died before the end of the study. Beginning on the 50th day of the study, hyper-reflexia, dyspnoea and convulsions were observed in the rats exposed to 5 mg/kg. Histological examination revealed fatty degeneration of the liver cells and moderate fatty infiltration of the epithelium of the renal tubules in the 5 mg/kg exposed group.

In a reproduction study, rats were fed diets containing heptachlor at 0, 0.3, 3, 6, or 110 mg/kg diet throughout three generations (Witherup *et al.*, 1976b). Mortality of pups in the 10 mg/kg group was slightly increased during the second and third weeks after birth in the second generation only. No adverse effects were observed in the lower dose levels. WHO (1984b) has reported no evidence of teratogenicity of heptachlor in rats and rabbits.

IARC (1991a) has concluded that while there is inadequate evidence for the carcinogenicity of heptachlor in humans, there is sufficient evidence in experimental animals. IARC ,as classified heptachlor as a possible human carcinogen (Group 2B).

Table 5.8-1 Acute toxicity of heptachlor to mammals.*				
Species	Route	Sex	LD_{50} (mg/kg body weight)	Reference
Rat	oral	M	40	NIOSH (1978)
	dermal	NS	119	NIOSH (1978)
Mouse	oral	NS	68	NIOSH (1978)
Guinea pig	oral	NS	80-90	NIOSH (1978)
Rabbit	oral	NS	116	NIOSH (1978)

*Taken from WHO (1984b).

5.8.3.3 Plants

No data were available.

5.8.3.4 Wildlife

The acute toxicity of heptachlor to selected aquatic organisms is given in table 5.8-2.

Table 5.8-2 Toxicity of heptachlor to selected aquatic organisms.*				
Organism	Grade	Temp (°C)	96-h LC_{50} (μg/ L)	Reference
Stonefly	technical (72%)	15.5	0.9-1.1	Sander and Cope (1968)
Penaeus duorarum (pink shrimp)	technical	27.5-30	0.11	Schimmel *et al.* (1976a)
Pimephales promelas (fathead minnow)	technical (72%)	25	130	Henderson *et al.* (1959)
Lepomis macrochirus (bluegill)	technical (72%)	25	26	Henderson *et al.* (1959)
Salmo gairdneri (rainbow trout)	technical (72%)	25	7.0	Macek *et al.* (1969)

Table 5.8-3 gives the acute toxicity of heptachlor to selected avian species. Chickens were fed heptachlor epoxide in their diet at 0, 0.02, 0.1 or 0.2 mg/kg diet for 25 weeks (Wovin *et al.*, 1969). Egg production and offspring viability were not affected, but hatchability was slightly decreased in groups fed 0.1 and 0.2 mg/kg.

Table 5.8-3 Acute toxicity of heptachlor to selected avian species.*		
Species	LD_{50} (mg/kg body weight)	Reference
Mallard	>2000	Tucker and Crabtree (1970)
Chicken	62.4	Sherman and Ross (1961)
Bobwhite quail	125	DeWitt and George (1960)
Ring-necked pheasant	150-400	DeWitt and George (1960)

*Taken from WHO (1984b).

Heptachlor has been strongly implicated in the decline of several wild bird populations including Canada geese and the American Kestrel in the Columbia Basin in the USA. A population of Canada geese at the Umatilla National Wildlife Refuge in Oregon experienced lowered reproductive success, and adult mortality (Blus *et al.*, 1984). Heptachlor epoxide residues in the brains of dead birds found in 1978 and 1979 were equal to or exceeded the experimentally determined lethal hazard zone of 8-9 μg/g. Low nest success was associated with egg residue levels of > 10 μg/g. The source of the exposure was thought to be heptachlor treated seeds, which were consumed by the geese from farmers fields. Following a partial ban of heptachlor for seed treatment in the area and its subsequent replacement with lindane, reproductive success increased, adult mortality decreased and the nesting population increased to 170 pairs in 1983, from a low of 102 pairs in 1979. The reproductive success of American Kestrels in the same area was also reduced (Henny *et al.*, 1983). Heptachlor epoxide residues in the eggs at concentrations> 1.5 ppm was associated with reduced productivity. The presence of residues in the eggs indicates that heptachlor is transferred through the food chain, as Kestrels are not seed eaters, which was the presumed route of exposure for the geese. Samples of treated seeds were analysed and concentrations in the seeds were lower than the recommended usage level (Blus *et al.*, 1984) which indicates that effects on wildlife may occur, even if heptachlor is used responsibly.

Mink were fed diets containing 0, 12.5, 25, 50 or 100 mg/kg heptachlor for 28 days, followed by a 7 day recovery period to determine the subacute toxicity of heptachlor to mink (Aulerich *et al.*, 1990). The NOEL for mortality was 50 mg/kg (5.67 kg/kg body weight/day). Signs of toxicity including reduced food consumption and loss of body weight were observed in mink fed the 25 mg/kg diet. During the recovery period, both food consumption and body weight increased for the groups fed 25, 50 and 100 mg/kg, relative to the values of the fourth week of exposure, however, food consumption in these groups was still less than control values. Adult male and female mink were fed diets containing 0, 6.25, 12.5 and 25 μg/g heptachlor for 181 days (before and during the reproductive period) to determine effects on reproduction (Crum *et al.*, 1993). All the mink in the 25 μg/g group (male and female) died, within 88 and 55 days respectively. The LOAEL, based on reduced kit growth, was 6.25 μg/g.

There is growing evidence linking persistent halogenated aromatic hydrocarbons (especially PCBs and dioxins) to reproductive and immunotoxic effects in wildlife (Fox, 1992; Reijinders and Brasseur, 1992). Although heptachlor has not been directly linked to these effects in December 1995 85wildlife, residues of heptachlor have been detected in arctic organisms in conjunction with these compounds (refer to chapter 3 for levels detected).

5.8.4 Persistence/fate

The half life of heptachlor in temperate soil is up to 2 years (WHO, 1984b). This persistence, combined with a high partition coefficient (log K_{ow} = 4.4-5.5), provides the necessary conditions for heptachlor to bioconcentrate in organisms. For example, the bioconcentration factors of heptachlor and heptachlor epoxide in fathead minnows (*Pimephales promelas*) were 9,500 and 14,400, respectively (Veith *et al.*, 1979). The chemical properties of heptachlor (low water solubility, high stability, and semi-volatility) favour its long range transport, and heptachlor and its epoxide have been detected in arctic air, water and organisms (Barrie *et al.*, 1992; Lockhart *et al.*, 1992; Thomas *et al.*, 1992). See chapter 3 for a detailed explanation of this process and levels detected.

5.8.5 Exposure

WHO (1984b) suggest that food is the major source of exposure of heptachlor to the general population. Heptachlor has been detected in the blood of cattle from both the USA (Salman *et al.*, 1990) and Australia (Corrigan and Seneviratna, 1990). Heptachlor was detected in 30 of 241 samples in American cattle, and violations of the MRL for heptachlor were detected in 0.02 % of Australian cattle. In both instances, heptachlor was among the most frequently detected organochlorine.

Contamination of a variety of foods in India (Kannan *et al.*, 1992b) and Vietnam (Kannan *et al.*, 1992a) by heptachlor was relatively low, when compared with other organochlorines, such as DDT and PCBs. In both countries, the estimated daily intake was below the ADI of 30 µg/person/day recommended by the Joint FAO/WHO Meeting on Pesticide Residues (JMPR). Kannan et al. (1990a) estimate a daily intake of 0.25 µg/person/day (for heptachlor and heptachlor epoxide combined, based on a 60 kg person) for Vietnam. The estimated daily intake for India is 0.07 µg/person/day (for heptachlor alone) (Kannan *et al.*, 1992b).

5.9 MlREX

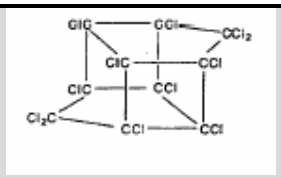
5.9.1 Introduction

Mirex was first synthesized in 1946 but was not introduced as a pesticide until 1959 (Smith, 1991). Technical grade mirex contains 95.12% mirex and 2.58% chlordecone. Mil-ex is synthesized by the dimerization of hexachlorcyclopentadiene in the presence of aluminium chloride (WHO, 1984c). Mirex is very resistant to breakdown, is very insoluble in water and has been shown to bioaccumulate and biomagnify. Due to its insolubility, Mirex binds strongly to aquatic sediments.

Mirex is a stomach insecticide with little contact activity. It's main use was against fire ants in the southeastern United States (WHO, 1984c), but it has also been used to combat leaf cutters in South America, harvester termites in South Africa, Western harvester ants in the USA, mealybug of pineapple in Hawaii and has been investigated for possible use against yellow jacket wasps in the USA (IARC, 1979b). It has also been used as a fire retardant in plastics, rubber, paint paper and electrical goods (Merck, 1991).

5.9.2 Chemical properties

CAS Chemical Name: 1,1a,2,2,3,3a,4,5,5a,5b,6- dodecachloroacta-hydro - 1,3,4 – metheno - 1H – cyclobuta[cd]pentalene



Synonyms and Trade Names (partial list): Dechlorane, Ferriamicide, GC 1283.

CAS No.: 2385-85-5; molecular formula: $C_{10}Cl_{12}$; formula weight: 545.5.

Appearance: White crystalline, odourless solid.

Properties: Melting point: 485°C; vapour pressure: 3 x 10^{-7} mm Hg at 25°C.

5.9.3 Toxicology

5.9.3.1 Studies in humans

There are no reports of injuries to humans resulting from exposure to Mirex (Hayes, 1 82). Mirex residues in human adipose have been reported. One study reported a range of 0.16 - 5.94 ppm in 6 of 1400 samples collected in 1971-1972 in the southern USA (Kutz *et al.*, 1974). Another study collected samples from 8 southeastern US states, and detected residues in 10.2 percent of those tested, with a geometric mean of 0.286 ppm in lipid (Kutz et at., 1985).

Organochlorines such as dioxins have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alterations in immune function December 1995 87(Holsapple *et al.*, 1991). Some organochlorines, such as DDE, may have weak estrogenic properties, and some authors have suggested a possible role in estrogen receptor positive breast cancer (Wolff *et al.*, 1993) while others have been unable to demonstrate such a role for DDT or its metabolites (Krieger *et al.*, 1994). Although Mirex itself has not been directly linked to these effects per se, some halogenated

aromatic hydrocarbons are known to affect endocrine function and reproductive systems (Peterson *et al.*, 1992; Gray, 1992; Thomas and Colborn, 1992). This requires further investigation of such possible outcomes.

5.9.3.2 Studies in laboratory animals

In acute studies, the oral LD_{50} of Mirex to rats ranges from 600 to > 3000 mg/kg, depending on sex of the test animal and nature of the formulation tested (Table 5.9-1). The dermal LD_{50} is > 2000 mg/kg (Gaines, 1969). Short term effects included decreased body weight, hepatomegaly, induction of mixed function oxidases, and morphological changes in liver cells (WHO, 1984c).

Rats which were fed 5 ppm Mirex in their diets for 30 days prior to mating and for 90 days after, showed reduced litter size and increased parental mortality (Ware and Good, 1967). Reduced litter sizes, and viability of neonates, along with formation of cataracts were observed in rats fed 25 ppm, mirex in the diet (Gaines and Kimbrough, 1970).

IARC (1979b) has concluded that while there is inadequate evidence for the carcinogenicity of Mirex in humans, there is sufficient evidence in experimental animals. IARC has classified Mirex as a possible human carcinogen (Group 2B).

Table 5.9-1 Acute toxicity for mirex in mammals.*

Species	Route	Sex	Vehicle	LD_{50} (mg/kg body weight)	Reference
Rat	oral	F	corn oil	600	Gaines (1969)
		M and F	-	2000	Gaines (1969)
Hamster	oral	F	-	125	Cabral *et al.* (1979)
Dog	oral	M	corn oil	1000	Larson *et al.* (1979) c)

* Taken from WHO (1984c).

5.9.3.3 Plants

A reduction in germination and emergence in several plant species was observed, which increased as the concentrations of Mirex increased (Rajanna and de la Cruz, 1975). Uptake, accumulation (de la Cruz and Rajanna, 1975) and translocation (Mehendale et *al.*, 1972) of Mirex by a variety of plant species has also been seen. These results are questionable, however, as lipophilic compounds such as mirex are generally not known to be taken up and translocated by plants. Contamination of plants is primarily a surface phenomenon resulting from aerial deposition of emissions or deposition of compound that has volatilized from the surface of the soil (Fries, 1995).

5.9.3.4 Wildlife

Crustaceans are the most sensitive aquatic organisms, with larval and juvenile stages being the most sensitive. Delayed mortality is typical of Mirex poisoning in crustaceans. Larval crabs exposed to 0.1 and 10 μg/L did not exhibit any adverse effects on survival for 5 days after hatching. Delayed mortality then occurred at the 1 and 10 μg/L exposure levels (Bookhout and Costlow, 1976). Mirex is also toxic to fish and can affect fish behaviour.

Mirex has a low short term toxicity to birds (Table 5.9-2).

Table 5.9-2 Acute oral toxicity of mirex to selected avian species.*

Species	LD_{50} (mg/kg body weight)	Reference
Mallard	2400	Waters (1976)
Japanese Quail	10 000	Waters (1976)
Pheasant	1400-1600	Waters (1976)

* Taken from WHO (1984c).

There is growing evidence linking persistent halogenated aromatic hydrocarbons (especially PCBs and dioxins) to reproductive and immunotoxic effects in wildlife (Fox, 1992; Reijinders and Brasseur, 1992). Although Mirex has not been directly linked to these effects in wildlife, residues of mirex have been detected in arctic organisms in conjunction with these compounds (refer to chapter 3 for levels detected).

5.9.4 Persistence/fate

Mirex is considered to be one of the most stable pesticides, with a half life of up to 10 years (WHO, 1984c). This persistence, combined with lipophilicity, provides the conditions necessary for mirex to bioconcentrate in organisms. Bioconcentration factors of 2,600 and 51,400 have been observed in pink shrimp and fathead minnows, respectively (Lowe *et al.*, 1971.; Huckins *et al.*, 1982). As with other chemicals, the amount taken up depends on the species tested, the concentration and duration of exposure.

The chemical properties of mirex (low water solubility, high stability, and semi-volatility) favour its long range transport, and mirex has been detected in arctic freshwater and terrestrial organisms (Lockhart *et al.*, 1992; Thomas *et al.*, 1992). See ch 3 for a more detailed explanation of this process and levels detected.

5.9.5 Exposure

WHO (1984c) concluded that the main route of exposure of mirex to the general population is through food, especially meat, fish and wild game, and that intake will be below established residues tolerances. Mirex residues were found in only one of 806 milk sample composites collected in a survey of US pasteurized milk (Trotter and Dickerson, 1993). No residues of mirex were detected in any samples of Egyptian fish (Abdallah *et al.*, 1990), nor in any samples from the fat of domestic farm animals in Ontario, Canada (Frank *et al.*, 1990).

5.10 POLYCHLORINATED BIPHENYLS

5.10.1 Introduction

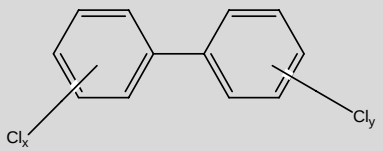
Polychlorinated biphenyls (PCBs) are mixtures of chlorinated hydrocarbons that have been used extensively since 1930 in a variety of industrial uses, including as dielectrics in transformers and large capacitors, as heat exchange fluids, as paint additives, in carbonless copy paper and in plastics. The value of PCBs for industrial applications is related to their chemical inertness, resistance to heat, non-flammability, low vapour pressure and high dielectric constant (WHO, 1993).

PCBs are produced by the chlorination of biphenyl by anhydrous chloride, under heated reaction conditions and in the presence of suitable catalysts. The degree of chlorination varies depending on the reaction conditions, and ranges from 21% to 68% (w/w). The result is a mixture of different congeners, and contains many impurities, including polychlorinated dibenzofurans (PCDFs) (WHO, 1993). Commercial PCB mixtures were sold based on the percentage of chlorine by weight, with each manufacturer utilizing their own system for identifying their products. In the Aroclor series, a 4-digit code is used; biphenyls are generally indicated by 12 in the first 2 positions, while the last 2 numbers indicate the percentage of chlorine in the mixture; i.e. Aroclor 1260 is a polychlorinated biphenyl mixture containing 60% chlorine (WHO, 1993).

There are 209 possible PCBs, from three monochlorinated isomers to the fully chlorinated decachlorobiphenyl isomer. Generally, the water solubility and vapour pressure decrease as the degree of substitution increases, and the lipid solubility increases with increasing chlorine substitution. PCBs in the environment may be expected to associate with the organic components of soils, sediments, and biological tissues, or with dissolved organic carbon in aquatic systems, rather than being in solution in water. PCBs volatilize from water surfaces in spite of their low vapour pressure, and partly as a result of their hydrophobicity; atmospheric transport may therefore be a significant pathway for the distribution of PCBs in the environment (Delzell *et al.*, 1994).

5.10.2 Chemical properties

Trade Names for different mixtures (partial list): Aroclor, Pyranol, Pyroclor, Phenochlor, Pyralene, Clophen, Elaol, Kanechlor, Santotherm, Fenchlor, Apirolio, Sovol.



X, Y = 1-5

CAS No.: 1336-36-3

Congener Group	Molecular Weight (g/mol)	Vapour Pressure (Pa)	Water Solubility (g/m^3)	log K_{ow}
Monochlorobiphenyl	188.7	0.9-2.5	1.21-5.5	4.3-4.6
Dichlorobiphenyl	223.1	0.008-0.60	0.06-2.0	4.9-5.3
Trichlorobiphenyl	257.5	0.003-0.22	0.015-0.4	5.5-5.9
Tetrachlorobiphenyl	292.0	0.002	0.0043-0.010	5.6-6.5
Pentachlorobiphenyl	326.4	0.0023-0.051	0.004-0.02	6.2-6.5
Hexachlorobiphenyl	360.9	0.0007-0.012	0.0004-0.0007	6.7-7.3
Heptachlorobiphenyl	395.3	0.00025	0.000045-0.0002	6.7-7
Octachlorobiphenyl	429.8	0.0006	0.0002-0.0003	7.1
Nonachorlobiphenyl	464.2	-	0.00018-0.0012	7.2-8.16
Decachlorobiphenyl	498.7	0.00003	0.000001-0.0000761	8.26

Mackay *et al* (1992).

5.10.3 Toxicology

There is a vast amount of information of the effects of PCB mixtures and congeners on humans, laboratory animals and wildlife. The evaluation the toxicity of PCBs is complicated because studies have been conducted using both mixtures and individual congener groups. Further, there is no consistent methodology used in analysis, data are not directly comparable, and contaminants such as PCDFs are toxic in their own right and invariably contribute to some of the effects observed (WHO, 1993).

The toxicology of PCBs is affected by the number and position of the chlorine atoms, as substitution in the *ortho* position hinders the rotation of the rings. PCBs without *ortho* substitution are generally referred to as coplanar and all others as noncoplanar (WHO, 1993). Coplanar PCBs, like dioxins and furans, bind to the AL-receptor and may exert, thus, dioxin-like effects in addition to AL-receptor independent effects which they share with non-coplanar PCBs (e.g. tumor promoters).

5.10.3.1 Studies in humans

The effect of acute exposure to high levels of PCBs in humans is well documented as a result of two incidents involving the consumption of PCB contaminated rice oil, although it is doubtful that all the effects observed are attributable to PCBs alone as PCDFs were detected in samples of rice oil tested. In 1968, rice oil in Japan was found to be contaminated with Kanechlor 400, a 48% chlorinated biphenyl, at 2000-3000 mg/kg. PCDFs at concentrations of 5 mg/kg were detected in 3 samples of rice oil containing PCB concentrations of approximately 1000 mg/kg (Nagayama *et al.*, 1976). The average estimated intake was 633 mg PCBs and 3.4 mg PCDFs, which is equivalent to approximately 157 μg PCBs/kg per day and 0.9 μg PCDFs/kg per day (Chen *et al.*, 1985; Masuda

et al., 1985). Signs and symptoms of exposure included enlargement and hypersecretion of the Meibomian glands of the eyes, swelling of the eyelids, and pigmentation of the nails and mucous membranes, occasionally associated with fatigue, nausea and vomiting. This was followed by hyperkeratosis and darkening of the skin with follicular enlargement and acneform eruptions, often with a secondary staphylococcal infection (Goto and Higuchi, 1969; Okumura and Katsuki, 1969). These symptoms are essentially the same as those observed in the second incident, referred to as the Yu-Cheng accident. In 1979, rice-bran oil in Taiwan was contaminated with PCBs. The estimated intake of PCBs and PCDFs was 0.7-1.84 g and 3.8 mg, respectively (Chen *et al.*, 1985).

Children born between 1978 and 1985 to mothers exposed in the Yucheng incident had hyperpigmentation, deformed nails and natal teeth, intrauterine growth delay, poorer cognitive development up to 7 years of age, behavioural problems and higher activity levels (Rogan *et al.*, 1988; Gladen *et al.*, 1990; Chen *et al.*, 1992; Hsu *et al.*, 1994). Yu *et al.*, (1994) evaluated the behaviour of children born between July 1978 and June 1985 (referred to as “early born Yucheng children”) annually between 1985 and 1991. Results indicate that the children scored 14 -38% worse than controls on the Rutter's Child Behaviour Scale A. Lai et at., (1994) found that these same children showed a mild but consistent cognitive deficit in comparison to the control children. The affected children scored consistently lower in all age groups, until 12 years of age, where they appeared to "catch up" to controls. Guo *et al.*, (1994) evaluated the development of children born seven to twelve years (born between July 1985 and December 1991) after maternal exposure. Results indicate that the children experienced mildly delayed development, but no differences in behaviour. Effects observed in the children born 7-12 years after maternal exposure is likely a result of the persistence of PCBs in the human body, resulting in prenatal exposure long after the exposure took place. These effects are consistent with the observations of poorer short term memory functioning in early childhood, observed by Jacobson *et al.*, (1990), in the children exposed prenatally by mothers who had high consumption of Lake Michigan sports fish.

Association between elevated exposure to PCB mixtures and alterations in liver enzymes, hepatomegaly, and dermatological effects such as rashes and acne (WHO, 1993) has been reported. Adverse effects are predominantly associated with higher blood concentrations. Lu and Wu (1985) found that people exposed in the Yucheng incident had low resistance, and suffered from a variety of infections. Examination during the first year revealed decreased concentrations of IgM and IgA, but not IgG; decreased percentages of total T-cells, active T-cells and helper T-cells, but normal percentages of B-cells and suppressor T-cells; suppression of delayed type response to recalling antigens; enhancement of lymphocyte spontaneous proliferation and an enhancement in lymphoproliferation to certain mitogens. After three years, some, although not all, of the effects had disappeared.

Bertazzi *et al.*, (1987) studied the mortality of 2100 workers employed in the manufacture of electrical capacitors between 1946-1982. Cancer deaths in both male and female workers were significantly increased. An increase in haematological neoplasms in workers was observed. The increase was significant in female but not male workers. A significant increase in gastrointestinal cancers was observed in male workers, and a higher than expected, though not statistically significant increase in lung cancer was observed. The study was, however, limited by the small numbers of deaths.

5.10.3.2 Studies in laboratory animals

PCBs have a low acute toxicity (Table 5.10-1). Their effects are manifested primarily through chronic exposure. Effects on the liver, skin, immune system, reproductive system, gastrointestinal tract and thyroid gland have been observed associated with exposure to PCB mixtures or individual congeners (WHO, 1992b).

Adverse reproductive effects observed in several studies in monkeys exposed to PCBs include low birth weights, skin hyperpigmentation, behavioural disturbances, atrophy of the thymus and lymph nodes, bone marrow hypoplasia and hyperplasia of the gastric mucosa (McNulty, 1985). Female rhesus monkeys (*Macaca mulatta*) fed diets containing 0, 0.25 or 1.0 mg Aroclor 1016/kg diet were bred after 7 months of dietary exposure (Barsotti and van Miller, 1984). There was no significant difference in the number of breedings between experimental and control groups, however neonatal weights in the 1.0 ppm group were significantly lower. PCBs have not been observed to be teratogenic in studies involving rats and non-human primates when tested orally, during critical periods of organogenesis (WHO, 1993).

PCBs were orally administered to Rhesus monkeys at levels of 0, 5, 20, 40 or 80 μg Aroclor 1254/kg body weight/day, and tests for immunomodulation began after 55 months of exposure (Tryphonas *et al.*, 1991). Statistically significant changes included a dose related decrease in IgM and IgG response to sheep blood cells and a dose related decrease in lymphoproliferation in response to certain mitogens. The authors concluded that moderate but statistically significant inhibitory effect on the immune system of rhesus monkeys results from chronic, low level exposure to Aroclor 1254 and that these effects may be due to altered T-cell and / or macrophage function.

IARC (1987c) has concluded that there is limited evidence for the carcinogenicity of PCBs in humans, and there is sufficient evidence in experimental animals. PCBs are therefore classified as probable human carcinogens (Group 2A).

Table 5.10-1 Acute oral toxicity of PCB mixtures in mammals.*				
Aroclor	Species/strain	Sex/age	LD_{50} (mg/kg body weight)	Reference
1254	rat/Wistar	male/120 d	2.0	Grant and Phillips (1974)
1221	rat/Sherman	female/-	2.0	Nelson *et al.* (1974)
1260	rat/Sherman	-/adult	4-10	Linder *et al.* (1974)
1242	rat/Sprague-Dawley	male/adult	4.25	Bruckner *et al.* (1973)

* Compiled by WHO (1993).

5.10.3.3 Plants

PCBs are not generally phytotoxic, with effects observed at approximately 1000 mg/kg (WHO, 1993).

5.10.3.4 Wildlife

The acute toxicity of selected PCB mixtures to some aquatic organisms is summarised in Table 5.10-2. Fathead minnows (*Pimephales promelas*) were exposed to Aroclor 1242, 1248 or 1254 in a continuous flow bioassay for 9 months (Nebeker *et al.*, 1974). Reproduction occurred at and below 5.4 µg Aroclor 1242/L, however, results were highly variable. Eggs exposed at concentrations of 15 and 51 µg/L were more resistant than fry. A significant reduction in spawning was observed in fish exposed to 1.8 µg Aroclor 1254/L.

Early life stages of fish are more sensitive to the effects of dioxins, furans, and PCBs (Walker and Peterson, 1992). Parts per trillion concentrations of these structurally related chemicals in lake trout and rainbow trout eggs produce toxicity through sac fry mortality associated with yolk sac edema and haemorrhages.

Table 5.10-2 Toxicity of PCB mixtures to select aquatic organisms.*					
Organism	Size/ Age	Temp (°C)	PCB Type	96-h LC_{50} (mg/L)	Reference
Gammarus fasciatus (scud)	mature	21	Aroclor 1248	0.052	Mayer and Ellersieck (1986)
Ischnura verticalis (damselfly)	late instar	15	Aroclor 1242	0.4	Mayer and Ellersieck (1986)
Salmo gairdneri (rainbow trout)	1.8 g	17	Aroclor 1260	>0.23	Mayer and Ellersieck (1986)
Pimephales promelas (fathead minnow)	fry	24	Aroclor 1254	0.008	Nebeker *et al.* (1974)
	fry	24	Aroclor 1242	0.015	Nebeker *et al.* (1974)
Lepomis macrochirus (bluegill)	0.8 g	18	Aroclor 1248	0.69	Mayer and Ellersieck (1986)
	0.8 g	18	Aroclor 1254	2.74	Mayer and Ellersieck (1986)
	2.0 g	22	Aroclor 1260	0.4	Mayer and Ellersieck (1986)

* Taken from WHO (1993).

PCBs have a low acute toxicity to birds (Table 5.10-3). Reproductive effects such as reduced hatchability and embryotoxicity were observed, even after dosing had ended. Broiler breeder and leghorn hens who were fed diets containing 0, 20 and 50 ppm Aroclor 1242 for one week experienced reduced hatchability (67.3 and 27.8 % of controls, respectively) (Briggs and Harris, 1973). The reduced hatchability of eggs continued into the fourth week, although administration of PCBs had ceased after the first week.

Table 5.10-3 Toxicity of dietary PCBs to birds.*

Species	Age	PCB type (Aroclor)	5 day LC_{50} (mg/kg diet)	Reference
Colinus virginianus (bobwhite quail)	10 days	1221	> 5000	Hill *et al.* (1975)
	10 days	1248	1175	Hill *et al.* (1975)
	10 days	1260	747	Hill *et al.* (1975)
Coturnix coturnix japonica (Japanese quail)	14 days	1221	> 5000	Hill and Camardese (1986)
	14 days	1248	4819	Hill and Camardese (1986)
	14 days	1260	2195	Hill and Camardese (1986)
Anas platyrhynchos (mallard)	10 days	1221	> 5000	Hill *et al.* (1975)
	10 days	1248	2798	Hill *et al.* (1975)
	10 days	1260	1975	Hill *et al.* (1975)
Phasianus colchicus (ring-necked pheasant)	10 days	1221	> 5000	Hill *et al.* (1975)
	10 days	1248	1312	Hill *et al.* (1975)
	10 days	1260	1260	Hill *et al.* (1975)

* Adapted from WHO (1993).

Aulerich and Ringer (1977) undertook a series of studies to investigate reproduction in ranch mink fed Great Lakes Coho salmon. Mink-fed Lake Michigan Coho salmon containing between 10 and 15 ppm PCBs as 30% of their diet for five months failed to whelp as did those fed a diet containing 5 ppm Aroclor 1254. The clinical signs and lesions observed in December 1995 95mink fed a diet containing Lake Michigan coho salmon, including anorexia, bloody stools, fatty liver, kidney degeneration and gastric ulcers, were similar to those fed a diet supplemented with PCBs. Mink removed from the contaminated diets were able to reproduce the following year, indicating that the reproductive effects observed may not have been permanent.

There is growing evidence linking persistent halogenated aromatic hydrocarbons such as PCBs to reproductive and immunotoxic effects in wildlife. Reijnders (1986) has studied the effects of fish contaminated with PCBs on reproduction of common seals (*Phoca vitulina*). Two groups of 12 female seals were fed diets of fish from the western part of the Wadden Sea, or from the north-east Atlantic. Residue analysis showed statistically significant differences between the two diets for PCBs and DDE. The average daily intake for group 1 was 1.5 mg PCBs and 0.4 mg DDE, and 0.22 mg and 0.13 mg for group 2. Females were mated with undosed males. There were no differences in circulatory hormone levels between the groups, but reproductive success was significantly lower in group 1.

5.10.4 Persistence/fate

The degradation of PCBs in the environment depends largely on the degree of chlorination of the biphenyl, with persistence increasing as the degree of chlorination increases. Half-lives for PCBs undergoing photodegradation range from approximately 10 days for a monochlorobiphenyl to 1.5 years for a heptachlorobiphenyl (WHO, 1993). The persistence of PCBs, combined with the high partition coefficients of various isomers (log K_{ow} ranging from 4.3 to 8.26) provide the necessary conditions for PCBs to bioaccumulate in organisms. Fathead minnows exposed to 3 µg Aroclor 1260/L for 250 days had a bioconcentration factor of 120,000, and those exposed to 2.1 µg Aroclor 1260/L of for 250 days had a bioconcentration factor of 270,000 (DeFoe *et al.*, 1978).

Concentration factors in fish exposed to PCBs in their diet were lower than those for fish exposed to PCBs in water. Channel catfish (*Ictalurus punctatus*) exposed to 1 mg Aroclor 1254/kg diet for 193 days had a bioconcentration factor of 2, compared to 61,190 for catfish exposed to 2.4 µg/L of Aroclor1254 for 77 days (Mayer *et al.*, 1977). This suggests that PCBs are bioconcentrated (taken up directly from the water) as opposed to being bioaccumulated (taken up by water and in food).

The chemical properties of PCBs (low water solubility, high stability, and semi-volatility) favour their long range transport, and PCBs have been detected in arctic air, water and organisms (Barrie *et al.*, 1992; Lockhart *et al.*, 1992; Thomas *et al.*, 1992; Muir *et al.*, 1992). See ch 3 for a more detailed explanation of this process and levels detected.

5.10.5 Exposure

The main source of PCB exposure to the general population is through food, especially fish (WHO, 1993). PCB residues (as Aroclor 1254 and 1260) were detected in 8.5 % of san1ples taken during a survey of the fat of domestic farm animals in Ontario, Canada between 1986 and 1988 (Frank *et al.*, 1990). The highest level detected was 0.30 mg/kg fat, in both sheep and pork fat. Residues of PCBs have declined in all species since the surveys inception in 1967. In a survey of foods in Vietnam, the highest levels of PCBs were detected in fish and shellfish, with levels of 760 and 1400 ng/g fat. The main sources of PCBs in the Vietnamese diets is cereals (including rice) and vegetables, and the daily intake of 3.7 µg/person/day is comparable to those of some industrialized countries (Kannan *et al.*, 1992a). A survey of foods in India also found that the highest levels of PCBs were in fish, with an average of 330 ng/g fat (Kannan *et al.*, 1992b). Again, the main source of PCB dietary intake (0.86 µg/person/day) was cereal and vegetable oil.

5.11 TOXAPHENE

5.11.1 Introduction

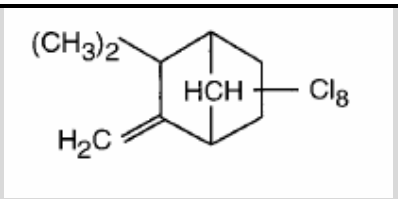
Toxaphene has been in use since 1949 and was the most widely used insecticide in the USA in 1975. It is a complex mixture of chlorinated camphenes containing 67-69%chlorine by weight produced by the chlorination of pine resins. Toxaphene is highly insoluble in water, and has a half life in soil of up to 12 years. It has been shown to bioconcentrate in aquatic organisms and is known to undergo atmospheric transport (WHO, 1984d).

Toxaphene is a nonsystemic and contact insecticide and was used primarily on cotton, cereal grains fruits, nuts and vegetables. It has also been used to control ticks and mites in livestock (WHO, 1984d).

Toxaphene has been banned in 37 countries, including Austria, Belize, Brazil, Costa Rica, Dominican Republic, Egypt, the EU, India, Ireland, Kenya, Korea, Mexico, Panama, Singapore, Thailand and Tonga. Its use has been severely restricted in 11 other countries, including Argentina, Columbia, Dominica, Honduras, Nicaragua, Pakistan, South Africa, Turkey and Venezuela (Environment Canada, 1995).

5.11.2 Chemical properties

CAS Chemical Name: Toxaphene



Synonyms and Trade Names (partial list): Alltex, Alltox, Attac 4-2, Attac 4-4, Attac 6, Attac 6-3, Attac 8, Camphechlor, Camphochlor, Camphoclor, Chemphene M5055, chlorinated camphene, Chloro-camphene, Clor chem T-590, Compound 3956, Huilex, Kamfochlor, Melipax, Motox, Octachlorocamphene, Strobane-T, Strobane T-90, Texadust, Toxakil, Toxon 63, Toxyphen, Vertac 90%.

CAS No.: 8001-35-2; molecular formula: $C_{10}H_{10}Cl_8$; formula weight: 413.82.

Appearance: Yellow, waxy solid with a chlorine/terpene-like odour.

Properties: Melting point: 65-90°C; boiling point: >120°C (decomposes); K_H: 6.3 x 10^{-2} atm m^3/mol at 20°C; log K_{OC}: 3.18 (calculated); log K_{ow}: 3.23-5.50; solubility in water: 550 μg/L at 20°C; vapour pressure: 0.2-0.4 mm Hg at 25°C.
(source: Montgomery, 1993).

5.11.3 Toxicology

5.11.3.1 Studies in humans

Symptoms of acute toxaphene intoxication include nausea, mental confusion, jerking of arms and legs and convulsions (Smith, 1991). In a human volunteer study, twenty-five subjects were exposed in a closed chamber to an aerosol of toxaphene for 30 min/day for 10 consecutive days at a maximal nominal concentration of 500 mg/m3. After 3 weeks, the same exposure was repeated for three days. Assuming a retention of 50%, the dosage was approximately 1 mg/kg/day. Physical examination, blood and urine tests did not reveal any toxic effects (Keplinger, 1963).

Eight women working in an area that had been sprayed with toxaphene at a rate of 2 kg/ha had a higher incidence of chromosome aberrations (acentric fragments and chromatid exchanges) than in control individuals (Samosh, 1974).

A survey of 137 workers involved in the manufacture of toxaphene (average exposure of 3.7 years) was reported. Annual physical examination of these workers did not reveal adverse effects associated with the exposure (Deichmann, 1973). Similarly, a survey of 199 employees who had worked with toxaphene (mean exposure of 5.2 years) found that none of the deaths appeared to be directly related to the exposure (US EPA, 1976).

Organochlorines such as dioxins have been linked to immunotoxic effects including suppression of antibody and humoral immune responses in laboratory animals and studies in exposed populations and non-human primates have shown that halogenated aromatic hydrocarbons have been associated with measurable alterations in immune function (Holsapple *et al.*, 1991). Some organochlorines, such as DDE, may have weak estrogenic properties, and some authors have suggested a possible role in estrogen receptor positive breast cancer (Wolff *et al.*, 1993), while others have been unable to demonstrate such a role for DDT or its metabolites (Krieger *et al.*, 1994) Halogenated aromatic hydrocarbons are also known to affect endocrine function and reproductive systems (Peterson *et al.*, 1992; Gray, 1992; Thomas and Colborn, 1992). Although toxaphene itself has not been directly linked to these effects *per se*, the similarity of structure and chemical properties shared by halogenated aromatic hydrocarbons suggests a possible adverse role for this chemical.

5.11.3.2 Studies in laboratory animals

Acute toxicity of toxaphene in mammals is shown in Table 5.11-1.

In a 13 week study, rats were fed diets containing 0, 4, 20, 100, or 500 ppm toxaphene. No clinical signs of toxicity were observed, and weight gain and food consumption were unaffected. Liver/body weight ratio and hepatic microsomal enzyme activities were increased in rats fed 500 ppm. Dose dependent histological changes were observed in the kidney, thyroid and liver. The NOAEL was determined to be 4.0 ppm (0.35 mg/kg) (Chu *et al.*, 1986). In another study, beagle dogs were fed toxaphene at 0, 0.2, 2.0 and 5.0 mg/kg body weight/day for 13 weeks. Food consumption and growth rate were unaffected. The liver/body weight ratio and serum alkaline phosphatase were increased in dogs fed 5.0 mg/kg. Mild to moderate dose dependent histological changes were observed in the liver and thyroid. The NOAEL for dogs was determined to be 0.2 mg/kg (Chu *et al.*, 1986).

Male and female rats were fed toxaphene in their diets at 0, 4.0, 20, 100 or 500 ppm in a one genre, two litter reproduction study. The rats received this diet for a total of 13 weeks (prior to, during, and after the reproduction period). The treatment levels studied had no effect on litter size, pup weight, fertility or gestation and survival indices. Effects in both the Po and Pi adults at levels from 20 to 500 ppm included increased liver and kidney weight, and histological changes in the thyroid, liver and kidney (Chu et al., 1988).

IARC (1979a) has concluded that while there is no adequate evidence for the carcinogenicity of toxaphene in humans, there is sufficient evidence in experimental animals. IARC has classified toxaphene as a possible human carcinogen (Group 2B).

Table 5.11-1 Acute toxicity of toxaphene to mammals.*

Species	Route	Vehicle	LD_{50} (mg/kg)	Reference
Rat	oral	corn oil	60	US EPA (1976a)
	dermal	xylene	780-1075	Gaines (1969)
Mouse	oral	corn oil	112	US EPA (1976a)
Dog	oral	corn oil	49	US EPA (1976a)
Guinea pig	oral	kerosene	365	US EPA (1976a)
Rabbit	oral	peanut oil	75-100	US EPA (1976a)
	dermal	peanut oil	>250	US EPA (1976a)

* Taken from WHO (1984d).

5.11.3.3 Plants

Toxaphene is essentially nontoxic to plants. In general, toxic effects have been observed only at levels much higher than the recommended usage level. Toxaphene applied at 44.8 kg/ha did not affect emergence, growth, yield and chemical composition of soybeans (Probst and Everly, 1957). Toxaphene applied at level of 72.3 kg/ha produced mild effects on cotton in a greenhouse study (Franco *et al.*, 1960). Toxaphene applied to tomato seedlings at a level of 15.7 kg/ha was phytotoxic two weeks after treatment (Hagley, 1965).

5.11.3.4 Wildlife

The acute toxicity of toxaphene to selected aquatic organisms is given in table 4.10-2. Brook trout exposed to toxaphene for 90 days experienced a 46 % reduction in weight at 0.039 μg/L, which was the lowest concentration tested (Mehrle and Mayer, 1975b). Egg viability in female trout was significantly reduced upon exposure to a concentration of 0.075 μg/L or more (Mayer *et al.*, 1975). Long term exposure to 0.5 μg/L reduced egg viability to zero. No reduction in hatchability was observed in eggs from unexposed females incubated in water with toxaphene concentrations between 0.0309 to 0.502 μg/L for 22 days prior to hatching.

Acute toxicity of toxaphene to selected aquatic organisms is given in Table 5.11-2

Table 5.11-2 Toxicity of toxaphene to selected aquatic organisms.*				
Organism	Size/Age	Temp (°C)	96-h LC_{50} (μg/L)	Reference
Penaeus duorarom (pink shrimp)	nauplii	-	2.2	Schimmel *et al.* (1977)
Lepomis macrochirus (bluegill)	0.6-1.7 g	18	21 (14-30)	Macek and McAllister (1970)
Pimephales promelas (fathead minnow)	0.6-1.5 g	12.7	3.2 (2.8-3.7)	Macek *et al.* (1969)
Salmo gairdneri (rainbow trout)		18.3	1.8	Cope (1965)

* Taken from WHO (1984d).

Bush *et al.*, (1977) exposed chickens from 1 day of age to maturity with toxaphene ranging from 0.5 to 100 mg/kg diet. No significant effects on egg production, fertility or hatchability were observed. Female ring-necked pheasants exposed to 100 and 300 mg toxaphene/kg diet resulted in reductions in egg laying and hatchability at the high dose level (Genelly and Rudd, 1956b).

Acute toxicity of toxaphene to selected bird species is given in Table 5.11-3.

Table 5.11-3 Toxicity of toxaphene to selected avian species.*			
Species	Age	LD_{50} (mg/kg body weight)	Reference
Mallard	3-5 mo	70.7	Trucker and Crabtree (1970)
Bobwhite quail	3 mo	85.4	Trucker and Crabtree (1970)
Mourning dove		200-250	Dahlen and Haugen (1954)
Fulvous tree duck	3-6 mo	99.0	Trucker and Crabtree (1970)

* Taken from WHO (1984d).

There is growing evidence linking persistent halogenated hydrocarbons (especially PCBs and dioxins) to reproductive and immunotoxic effects in wildlife (Fox, 1992; Reijinders and Brasseur, 1992). Although toxaphene has not been directly linked to these effects, residues have been detected in arctic air (Barrie *et al.*, 1992), and the similarity of structure and chemical properties shared by these compounds suggests a possible adverse role for this chemical.

5.11.4 Persistence/fate

The half-life of toxaphene in soil ranges from 100 days up to 12 years, depending on the soil type and climate (WHO, 1984d). This persistence, combined with a high partition coefficient (log K_{ow} = 3.23-5.50) suggests that toxaphene is likely to bioconcentrate. Mosquito fish exposed to toxaphene at a concentration of 44.4 μg/L concentrated toxaphene by a factor of 4247 (Sanborn *et al.*, 1976). Brook trout fry exposed to concentrations ranging from 0.041 to 0.5 μg/L concentrated toxaphene between 4,900 and 76,000 times (Mayer *et al.*, 1975).

The chemical properties of toxaphene (low water solubility, high stability, and semivolatility) favour its long range transport, and toxaphene has been detected in arctic air (Barrie *et al.*, 1992). See Chapter 3 for a more detailed explanation of this process and levels detected.

5.11.5 Exposure

Exposure of the general population is most likely through food however levels detected are generally below maximum residue limits (WHO, 1984d). Due to its ban in many countries, recent food surveys have generally not included toxaphene and hence recent monitoring data are not available.

6. USES, SOURCES, ALTERNATIVES AND BARRIERS TO ADOPTION OF ALTERNA TIVES

6.1 INTRODUCTION

Due to the nature of the selected persistent organic pollutants listed below most of this report deals with pesticides used on agricultural crops, since 9 of the 12 compounds are primarily used for this purpose (International Experts Meeting on Persistent Organic Pollutants, 1995). The following compounds are known internationally as the *"Dirty Dozen"* and are the focus of this report:

DDT, Aldrin, Dieldrin, Endrin, Chlordane
Heptachlor, Hexachlorobenzene, Mirex, Toxaphene
Polychlorinated biphenyls
Dioxins and Furans

Since the late 1970s all of above compounds have been either banned or subjected to severe use restrictions in most countries. Information is limited as to what countries are using these compounds, for what specific uses or purposes they are being used, and how they are applied. Although there appears to be considerable (albeit disjointed and sometimes contradictory) information and data that describes the aggregate volume of persistent organic pollutants produced and used throughout the world, there is very little data about specific uses in each country or the possible alternatives to persistent organic pollutants in each situation.

6.2 USES AND SOURCES OF PERSISTENT ORGANIC POLLUTANTS

Two important conclusions were reached regarding the use of persistent organic pollutants:

- Most, if not all, of the persistent pesticides and industrial chemicals are still in use in many countries, and
- It is not possible to accurately measure or qualitatively ascertain:
  - how much of these persistent organic pollutants are being used in specific countries,
  - specific uses or crops they are being applied to, or
  - the direction that is being taken regarding the complete elimination of these products.

Gathering of use data for these 12 compounds is very difficult. There are no central registers for the production or use of these or other hazardous compounds (Voldner and Li, 1995). Where data exists, they are plagued with a variety of limitations making it difficult to develop comprehensive and accurate use profiles. Some of these limitations that affect the quantity, accuracy and reliability of data where encountered in a recent FAD survey (UNEP, 1995) which are highlighted below:[2]

[2] These points are largely derived from UNEP, 1995.

- In some countries, customs data suffers from a lack of precision. The chemical product being reported on may not be 100% technical product but many include various additives and ingredients to constitute a formulation containing small percentages of the active ingredient. For example, 1 ton of a product containing 20% hexachlorocyclohexane may be recorded as 1 ton hexachlorocyclohexane.

- In the case of dicofol for example, the FAO survey specified "dicofol containing <78% p,p' - dicofol or > 1 g/kg of DDT and DDT related compounds". Statistics data may not be as precise when indicating such details about content.

- Some sources of information provide use data in terms of value (in US dollars) instead of weight, which makes it difficult to understand and compare use data.

- Chemicals having frequently used synonyms may be recorded in different countries under different names and may not be readily recognized within these countries as substances of concern.

- Confidentiality and the competition policy of individual companies may also inhibit data provision so long as there is no legislation requiring trade data to be reported. In some countries, legislation protects data confidentiality and prevents data access.

- In many countries, particularly in developing countries, such use and trade data are simply not available because they lack the infrastructure to measure and record it.

According to Han (1994) there is growing activity among nations to develop emissions inventories for persistent organic pollutants, including work of the United Nations Economic Commission for Europe (UN ECE) under the European Monitoring and Evaluation Program (EMEP). The completeness and reliability of the emissions inventories at that time appeared to be best for P AH, somewhat less for PCBs, dioxins and furans, and brominated flame retardants, and worst for pesticides. Voldner and Li (1995) reported on the use status of four hazardous pesticides (toxaphene, DDT, technical HCH, and lindane) as part of an ongoing investigation into 10 pesticide compounds.

Summarized below are data from a variety of published sources that indicates the volume of production/use of certain persistent organic pollutants, the countries that still permit their importation and the reasons for their continued use. (Note: the reader is cautioned to bear in the mind the limitations discussed above regarding data collected for this and other reports.)

6.2.1 Aldrin/dieldrin/endrin

These three cyclodiene chemicals were used extensively as insecticides in the USA until the early 1970s. Use of these three cyclodiene compounds continues in Central and South America, and Asia (Voldner and Ellenton 1987; Barrie *et al.*, 1992).

Aldrin is used as primarily as a termiticide. It is oxidized to dieldrin by soil bacteria, insects and mammals. Aldrin is banned in the USA and Russia but its use is permitted in Canada for below-ground termite control. Aldrin is permitted for import to Congo, Ethiopia, Malaysia, Nepal, Sri Lanka, Sudan, Tanzania, Thailand, Trinidad and Tobago, and Venezuela (Murray, 1995). Aldrin is permitted in certain countries for agricultural or public health purposes, such as:

- Kenya, tsetse fly control;
- USA, dipping of non-food roots and tops and moth-proofing by manufacturing processes in closed systems.

Dieldrin is permitted for import to: Congo, Ethiopia, Malaysia, Nepal, Sri Lanka, Sudan, Tanzania, Trinidad and Tobago, Uganda and Venezuela, primarily for termite control, but other minor uses are reported such as:

- Kenya, banding of coffee trees;
- USA, dipping of non-food roots and tops and moth-proofing by manufacturing processes in closed systems;
- Venezuela, vector control, ant control (granular formulation) in soils, and emergency agricultural uses.

The latest available data (Voldner and Ellenton, 1987) estimates cumulative production of Aldrin and Dieldrin in the USA at 99,788 tonnes, and 29,937 tonnes in South and Central America. No data are available for global production.

Endrin is used as an insecticide and has been reported for agricultural use in the Dominican Republic. It is manufactured or imported in the USA, Philippines and Japan (FAO/UNEP, 1995).

6.2.2 Chlordane

Chlordane was used as a general insecticide from 1945-1988. The estimated cumulative production of chlordane is approximately 274,650 tonnes in the USA. No data were available for either global or production in other countries (Voldner and Ellenton, 1987). Chlordane has been reported as being produced by one industrial country, being exported by two industrialized countries and being imported (693 tonnes imported over the period 1990-94) by four countries (83 % imported by developing countries and 17 % imported by industrial countries (Murray 1995).

Chlordane is used in:

- Mexico, for restricted use on only two crops, maize and sorghum;
- Canada, China, and the United Kingdom have limited its use to the control of soil pests only;
- Belgium has restricted its use solely for ant control and China reportedly permits use as a seed dressing, and;
- Belize, Canada, and Cyprus have retained use for termite and structural pest control.

Chlordane is permitted for import to Australia, Cuba, Ethiopia, Malaysia, Mexico, Oman, Philippines, Sri Lanka, Sudan, Tanzania, Thailand, Trinidad and Tobago (FAO/UNEP, 1995). Chlordane has been banned in Austria, Belgium, Bolivia, Brazil, Chile, Columbia, Costa Rica, Denmark, Dominican Republic, EU, Ecuador, EI Salvador, Fiji, Germany, Guatemala, Hong Kong, Ireland, Italy, Kenya, Korea, Lebanon, Lichtenstein, Mozambique, Netherlands, Norway, Panama, Paraguay, Philippines, Poland, Portugal, Santa Lucia, Singapore, Spain, Sweden, Switzerland, Tonga, Turkey, United Kingdom, Yemen and Yugoslavia.

6.2.3 DDT

DDT is a wide-spectrum organochlorine pesticide which was first registered in 1946 and has been used extensively worldwide (Han, 1994). DDT is used today principally for public health use for vector control for malaria and bubonic plague (FAO/UNEP, 1995).

Although DDT has been banned or restricted for nearly two decades in Canada, the United States and Europe, it does continue to be manufactured and used in Southern Asia, Africa, Central America and South America (Barrie et at., 1992). DDT has been reported as being produced between 1990-94 by 3 developing and one industrial country in the amount of 29,709 tonnes. It is reported as exported by 3 developing and 2 developed countries in the amount of 11,900 tonnes. DDT is imported by 8 developing countries on each continent in the quantity of 4,705 tonnes (FAO/UNEP, 1995).

Voldner and Ellenton (1987) have estimated cumulative production of DDT at 1.36 million tonnes and is distributed as follows:

USA = approximately 996,000 tonnes
Other = approximately 181,000 tonnes
Global = approximately 1,360,000 tonnes

DDT is permitted for import to Bhutan, Bolivia, Ethiopia, Guinea, India, Kenya, Malaysia, Mauritania, Mexico, Nepal, Philippines, Sri Lanka, Sudan, Switzerland, Tanzania, Thailand, Venezuela and Vietnam (FAO/UNEP, 1995). DDT has been banned in Argentina, Australia, Austria, Bulgaria, Burkina Faso, Colombia, Costa, Rica, Cuba, Cyprus, Denmark, Dominican Republic, Egypt, EI Salvador, Ethiopia[3], Finland, Fiji, Hong Kong, Indonesia, Ivory Coast, Japan, Korea, Lebanon, Liechtenstein, Mozambique, New Zealand, Nicaragua, Paraguay, Poland, Santa Lucia, Singapore, Switzerland, USA, Yemen, and Zimbabwe (International Experts Meeting on Persistent Organic Pollutants, 1995).

[3] The discrepancy that allows the same country to import the chemical as well as report it as a banned substance is due to the fact that the information is from two different sources indicating the sometimes contradictory nature of the information obtained.

6.2.4 Dioxins and furans

Dioxins and furans are not only formed as undesirable by-products of waste combustion, but may also be formed in any process where chlorine and carbon are present at high temperatures (Government of Canada, 1991). As such, dioxins and furans are contaminants and often enter the environment along the pathways of their host compounds such as PCBs, pentachlorophenol and chlorinated pesticides (International Joint Commission, 1994).

While there are numerous industry and government programs to investigate the sources of dioxins and furans and to develop control technologies there are no known use or emission data available at this time specific to dioxins and furans (International Joint Commission, 1994).

6.2.5 Hexachlorobenzene

Hexachlorobenzene is an organochlorine product which has been manufactured as an industrial chemical, and is currently produced in combustion processes as a by-product or impurity in the production of certain chlorinated pesticides and industrial chemicals (Voldner and Smith, 1989; Axenfel et at., 1992). It has been reported in agricultural use primarily as a fungicide for seed protection in wheat (against common bunt and wheat smut) and other cereals (FAO/UNEP, 1995).

Hexachlorobenzene was reported as being exported by both the OECD countries and non-OECD countries (FAO/UNEP, 1995). The latest global use and production data dates back to the mid 1970's. Cumulative production in the USA is approximately 100,000 tonnes (Courtney, 1979). Estimates of global annual production in the mid 1970's range from 1 to 2 tonnes (Courtney, 1979).

Hexachlorobenzene had been banned in Austria, Belgium, Czechoslovakia, Denmark, the EU (as a pesticide), Germany, Hungary, Liechtenstein, Netherlands (as a pesticide), Panama, Switzerland, Turkey, Untied Kingdom (as a pesticide), USSR (for use as a pesticide), and Yugoslavia (International Experts Meeting on Persistent Organic Pollutants, 1995).

6.2.6 Heptachlor

Heptachlor is a chlorinated-hydrocarbon insecticide with contact and stomach poison action, particularly effective on soil insects (Barrie et al., 1991). It was first produced by Velsicol Chemical Company in 1948 and is still produced by some OECD countries (F AO/UNEP , 1995).

Heptachlor is permitted for import to: Burkina Faso, Costa Rica, Ethiopia, Pakistan, Sudan, Tanzania, Thailand, Togo, Trinidad and Tobago (FAO/UNEP, 1995).

Heptachlor is used primarily for agriculture purposes in:

- Mexico, which has restricted the use to only two crops, maize and sorghum;
- Bulgaria which has limited its use to seed treatment only, and
- The USA reports termite control and dipping of roots or tops of non-food plants.

6.2.7 Mirex

Mirex is a chlorinated hydrocarbon insecticide, first developed by Allied Chemical Corporation.

Mirex is believed to be used today as an insecticide, for the control of various ant species, but no survey data h.1S identified specific uses. It is thought that this compound may also be used as flame or fire retardant (FAO/UNEP, 1995). There are currently no known manufacturers of mirex, and no use or production data were available.

6.2.8 Polychlorinated Biphenyls

PCBs have been manufactured since 1929, first by Monsanto Chemical Corporation in the USA, and were used widely in transformer and capacitor oils, hydraulic and heat exchange fluids and lubricating and cutting oils (Barrie et at., 1992). From the early 1970s to the mid-1980s, the use of PCBs was progressively restricted to closed electrical systems, where they remain in use throughout most of the world.

The latest available data (Voldner and Ellenton, 1987) estimates cumulative production of PCBs at 1.17 million tonnes, broken down as follows:

USA = 543,000 tonnes
Other = 362,000 tonnes
Global = 1,170,000 tonnes

No data are available that indicate the volume of PCBs in use in each country or how much is being held in waste storage facilities. PCBs have been banned in Austria, Czechoslovakia, Finland, Germany, Liechtenstein, Netherlands, Norway, Switzerland, and USA (International Experts Meeting on Persistent Organic Pollutants, 1995).

6.2.9 Toxaphene

Toxaphene is a chlorinated-hydrocarbon insecticide first produced by BFC Inc. in 1946. Toxaphene became commercially available in 1948, and was the most heavily used insecticide in the USA during the 1960's and 1970's. It was mainly used on cotton (75% of the use was in the southern and south central states) with smaller amounts used for weed control in soybeans. Its use in the USA was banned in 1983 (Han, 1994). There are still agricultural uses reported in Nicaragua and Zambia.

Toxaphene is manufactured in China, Pakistan, and Nicaragua at this time (FAO/UNEP, 1995).

Toxaphene has a cumulative production from 1947 to the present of 454,000 tonnes in the USA and approximately 181,000 tonnes in other countries (Voldner and Ellenton, 1987). Global cumulative production of toxaphene equals approximately 450,000 tonnes according to Voldner and Li (1995).

Toxaphene has been banned in Austria, Belgium, Belize, Bolivia, Brazil, Bulgaria, Burkina Faso, Costa, Rica, Cuba, Denmark, Dominican Republic, Ecuador, Egypt, EI Salvador, the EU, Finland, Germany, Guatemala, India, Ireland, Kenya, Korea, Liechtenstein, Mexico, Mozambique, Panama, Paraguay, Peru, Philippines, Portugal, Santa Lucia, Singapore, Switzerland, Thailand, Tonga, and United Kingdom (International Experts Meeting on Persistent Organic Pollutants, 1995).

6.3 RELIABILITY OF USE AND PRODUCTION DATA

Although the above discussion reported global production figures (where available) the most complete data was specific to the USA. If one hopes to develop meaningful recommendations regarding policies and programs to reduce or eliminate the use of persistent organic pollutants, precise and accurate data regarding uses by country must be compiled. Some specific data, relating to uses on certain crops, such as the use of pesticides on rice in the Philippines (Table 6-1) does exist. This leads one to believe that there may be crop and country specific data for a variety of pesticide compounds in other regions. It must be recognize that no central use database exists in most countries in the developing world, let alone on a global or regional basis.

It was possible to glean some information from personal interviews regarding the general usage of some of these persistent organic pollutants. For example, a warehouse full of DDT was observed in Madagascar, despite the "official" record that no such products have been imported into the country. The owner had no intentions of using the chemical but has no avenue to dispose of it either (Brown, 1995).

It is known that chlordane and toxaphene are still being used in Honduras for seed grain treatment (Grenier, 1995). Discussions with international development experts confirm that the general lack of knowledge of the alternatives to persistent organic pollutants. For example, Grenier (1995) states that there is general lack of knowledge regarding alternatives to chlordane and toxaphene at the farmer and vendor level in Honduras in addition to a lack of records regarding how much of these persistent organic pollutants are being used.

Table 6-1 Types of insecticide applied by season, in percent of fanners reporting, Nueva Ecija, Phillippines 1979-1991.

	Farmers reporting (%)					
Insecticide	Wet season			Dry Season		
	1979	1985	1991	1979	1985	1991
Organochlorines						
Endrin	11	1	0	10	0	0
Endosulfan	6	4	31	4	9	27
Organophosphates						
Methyl parathion	12	8	5	8	9	5
Monocrotophos	36	59	33	33	55	36
Azinphos ethyl	7	4	2	12	4	1
Diazinon	9	0	0	15	3	0
Carbamates						
Isoprocarb	26	10	20	36	10	33
Isoprocarb + lindane	4	29	0	3	33	0
BPMC + chlorpyrifos	54	40	7	54	26	14
Carbofuran	9	3	3	9	1	0
Methomyl	9	3	0	12	3	1
Pyrethroids						
Cypermethrin	0	24	24	0	20	16

Source: Rota, Agnes, C and Prabhu L. Pingali. Pesticides, rice productivity, and farmers' health and economic assessment.. World Resources Institute. Washington D.C. 1993. pg.35.

Due to the lack of information regarding usage of these compounds in the developing countries, it was not possible to find the data necessary to answer the questions of how much of these chemicals are still being used specifically in each country, where are they being used and what they are being used for. Without such in-depth quantitative information we are constrained in or ability to address the remaining research questions with any degree of detail relating to each of the 12 persistent organic pollutants, or to discuss alternatives to these uses or barriers to specific alternatives.

However, through the limited information that was obtained as well as the insights provided through personal interviews it was possible to develop reasons for the use of hazardous chemicals in general as well as some alternatives to the persistent organic pollutants, and some of the barriers to adoption of these. The balance of this report is therefore intended to be generic in nature and not limited to the persistent organic pollutants listed above, but instead refers to hazardous chemicals in general, again mostly pesticides. Based discussions with selected international development experts, the term "hazardous chemicals" as used here refers to those chemicals identified by the World

Health Organization and which generally include a mix of organochlorine and organophosphate pesticides.

6.4 ALTERNATIVES TO PERSISTENT ORGANIC POLLUTANTS

A variety of chemical alternatives that are available for the persistent organic pollutants are discussed in this section. One list compiled from a recent international forum is reproduced in Table 6-2. Table 6-3 was supplied by FAO/UNEP (1995) and includes chemical alternatives for specific uses. None of the alternatives are perfect substitutes for the use of the persistent organic pollutants, since they do not have the same persistence as the persistent organic pollutants. Table 6-2 and Table 6-3 are not exhaustive listings of either the uses of the persistent organic pollutants or of the alternatives to the these substances. However, they do show that possible alternatives do exist.

Table 6-2 Persistent organic pollutants, their uses and their possible alternatives.

Persistent organic pollutant	Uses of persistent organic pollutant	Alternatives to persistent organic pollutant
PCBs	dielectric fluids in transformers and capacitors	Silicone oils
Dioxins and Furans	By-products of manufacturing/combustion	None
Aldrin	Insecticide on cotton Seed treatments	Other pesticides that are less persistent[4] Biological controls Integrated pest management
Dieldrin	Insecticide on cotton Insecticide on agricultural crops	Other pesticides that are less persistent Biological controls Integrated pest management
DDT	Primarily for malaria control	Biological controls Integrated pest management
Endrin	Insecticide on agricultural crops Rodenticide against meadow mice and voles	Other pesticides that are less persistent Biological controls Integrated pest management
Chlordane	Earthworm and insect control on lawns, gardens and agricultural crops Insecticide for termite control	Other pesticides that are less persistent Biological controls Integrated pest management
Hexachlorobenzene	Pesticide By-product during manufacture of chlorinated solvents	Other pesticides that are less persistent

[4] See Table 6-3 for a brief discussion of some chemical substitutes.

Table 6-2 Persistent organic pollutants, their uses and their possible alternatives.		
Persistent organic pollutant	Uses of persistent organic pollutant	Alternatives to persistent organic pollutant
Mirex	Insecticide against fire ants Fire retardant	Other pesticides that are less persistent Biological controls Integrated pest management
Toxaphene	Pesticide on crops Control of scabies on sheep and cattle	Other pesticides that are less persistent Biological controls
Heptachlor	Earthworm and insect control on lawns, gardens and agricultural crops Insecticide for termite control	Other pesticides that are less persistent Biological controls
Source: Integrated Pest Management, Gips, T. 1987. International Experts Meeting on Persistent Organic Pollutants: Towards Global Action. June 1995. Breaking the Pesticide Habit: Alternatives to 12 Hazardous Pesticides.		

Table 6-3 Specific chemical alternatives for persistent organic pollutants identified in the FAO survey. 1995.		
Chemical	**Country**	**Alternatives**
Heptachlor	Australia	Uses not specified: chlorpyrifos, azinphos-ethyl, terbufos, diazinon, carbaryl, cyfluthrin, bendiocarb, fenamiphos, ethoprophos, pirimiphos-ethyl, prothiofos
	Sri Lanka	Termite Control: chlorpyrifos Rhizome borers: carbofuran granules
	United States	Agricultural crops: carbaryl, diazinon, bendiocarb, chlorpyrifos, acephate, isazophos, fonofos, synthetic pyrethroids Other sites: propoxur, diazinon, malathion, bendiocarb, chlorpyrifos, dichlorvos, acephate, propetamphos, synthetic pyrethroids
Aldrin	Australia	Uses not specified: chlorpyrifos, methomyl, diazinon, phorate, permethrin
	Colombia	Uses not specified: chlorpyrifos, pyrethroids
	United States	Seed treatment: chlorpyrifos, methoxychlor, acephate Preplant soil treatment: diazinon, chlorpyrifos Quarantine use: chlorpyrifos Termite use: chlorpyrifos, isofenphos, synthetic, pyrethroids
	Cuba	Uses not specified: diazinon (up to 0.9 kg/ha) Thiodan (endosulfan)

Table 6-3 Specific chemical alternatives for persistent organic pollutants identified in the FAO survey. 1995.		
Chemical	**Country**	**Alternatives**
Chlordane	Australia	Uses not specified: bendiocarb, carbaryl, chlorpyrifos, cyfluthrin, cyromazine, deltamethrin, diazinon, dichlorvos, fenthion, malathion, : -- permethrin, propoxur, pyrethrin
	Indonesia	Uses not specified: chlorpyrifos, permethrin, alphametrin, cyfluthrin, creosote, phoxim, fenitrothion
	Sri Lanka	Termites in agricultural land: chlorpyrifos
	United States	Agricultural crops: carbaryl, diazinon, bendiocarb, chlorpyrifos, trichlorfon,, acephate, isazofos, fonofos, synthetic pyrethroids Other sites: propoxur, diazinon, malathion, bendiocarb, chlorpyrifos, dichlorvos, acephate, propetamphos, synthetic pyrethroids
Dieldrin	Australia	Uses not specified: diazinon, pirimiphos-ethyl, prothiofos, acephate, methomyl, permethrin, endosulfan, azinphos-ethyl, deltamethrin, monocrotophos, sulprofos, carbaryl, bendiocarb, cyfluthrin, dichlorvos, fenthion, propoxur, pyrethrins and chlorpyrifos
	Niger	Uses not specified: fenitrothion 1000 ULV
	Sri Lanka	Termites in agricultural land: chlorpyrifos
	United States	Uses not specified: diazinon, malathion, chlorpyrifos, endosulfan, carbaryl, bendiocarb, cypermethrin, propoxur, isazofos, pyrethrins
DDT	Australia	Uses not specified: azinphos-ethyl, diazinon, carbaryl, fenthion, malathion, methidathion, methomyl, monocrotophos, phosmet, chlorpyrifos, dimethoate, trichlorfon, rotenone, endosulfan, dichlorvos, methamidophos, permethrin, deltamethrin, fluvalinate
	Cuba	Uses not specified: pyrethroids
	Thailand	Uses not specified: thiodicarb, synthetic pyrethroids
	United States	Uses not specified: cblorpyrifos, malathion, carbaryl, acephate, diazinon, esfenvalerate, methomyl, dicofol, phorate, permethrin, sulfur

There are many possible reasons that developing countries, still using some persistent organic pollutants have not eliminated or reduced their use. Not all developing countries, however, use persistent organic pollutants and not all developing countries use persistent organic pollutants to the exclusion of the alternatives. In Honduras, for example, there are many situations where advanced pest control technologies are used within an Integrated Pest Management (IPM) system and an environmentally sustainable framework (Grenier, 1995). A well developed distribution network for newer and safer products, as well as a good knowledge base of information on the hazards of the older products exists in these areas. However, some areas in Honduras do not have a high

concentration of agricultural producers and those that are located there may be more subsistence based. These areas tend not to have a "critical mass" of agricultural production and thus do not have the same distribution networks for the newer products and may not have access to the same amount of information about hazards or risks. In fact, the alternatives may not even be marketed or available in these areas so there are no substitutes for the older compounds (Grenier, 1995).

For the most part, farmers in remote areas or those producing subsistence-based food and crops may harbour a social attitude that fosters the continued use of older and sometimes hazardous products. For example, Grenier (1995) observed that many small-scale producers are illiterate, adverse to change, and generally poorly informed about safe pesticide use, storage or handling procedures. Many of these farmers firmly believe that "since older pesticides have been used effectively in the past, they should stick with what they know works". Grenier (1995) is quick to point out that in Honduras, like many other Latin American Countries, one can find examples of both highly progressive and "traditional" pest control practices. Gulliver (1995), Brown (1995), and Anderson (1995) all agree with this view for other developing regions in the world.

Not all alternatives are transferable to a developing country. Each crop and location has a unique pest-crop relationship which must be accommodated. When alternatives are not given the cultural and social sensitivity that they deserve in the developing countries results regarding adoption are not promising. All barriers to alternatives must be examined for each individual situation before recommendations can be made.

6.5 CONSTRAINTS TO ADOPTION OF ALTERNATIVE TECHNOLOGIES

In the spirit of this report, a generic list of the constraints to the adoption of alternative technologies was formed through the literature and the interviews that were conducted. These constraints include:

Costs of alternatives
Availability of alternatives
Compatibility of alternative technologies to existing technologies
Education of end users
Knowledge of alternatives and uses
Regulatory constraints

Each of these individually can constitute a significant barrier to the adoption of new technologies. Often there are a number of these constraints that are in place at one time and must be resolved. As indicated above, the general lack of use data on the persistent organic pollutants makes any discussion of the alternatives difficult since it is not known what these older compounds are used for and how much is being used. Nonetheless, one can illustrate the barriers to alternative technologies in a generic context by focusing again on crop protection in reference to all hazardous chemicals. The rest of this section focuses on the generic barriers to adoption and describes some specific observations by the experts interviewed for this study.

6.5.1 Costs of alternatives

When one first investigates the rationale for why farmers use a specific crop protection technology, the immediate consideration is cost. Given the choice between a " $2 solution and a $5 solution" for pest control, one can always expect a rational farmer to gravitate towards the lower cost solution, whether in a developed or developing world context. Since the persistent organic pollutants and most other hazardous compounds identified by the WHO tend to be older, inexpensive, and relatively easy to apply, they continue to be used if they are effective relative to newer alternatives.

This is readily seen from a comparison of pesticide use on rice in the Philippines. Endrin, which was registered for use as recently as 1991, had a retail price 50.88 P/L in the wet season and 54.75 P/L in the dry season. The price of less persistent organophosphorous pesticides was 52.51 P/L or kg in the wet season and 60.86 P/L or kg in the dry season. The retail prices of newer pyrethroid compounds were much higher, ranging from 84.47 P IL or kg in the dry season to 92.81 P/L or kg in the wet season. Similar data can probably be found for other persistent organic pollutants in other regions, which demonstrate a significant cost difference between older and newer pesticide compounds.

The cost is less for the organochlorine pesticides, such as endrin, due in part to the fact that the development costs and capital costs related to these "older" chemicals have already fully depreciated. Therefore, the marginal costs of using older products such as endrin or DDT are negligible. On the other hand, the available alternatives, like the pyrethroids are newer and, as such, contain higher development and capital costs.

The price of the compounds versus the alternatives is not, however, the only cost barrier to the alternative technologies. Wood-Thomas (1995) noted that the very nature of persistence that makes these compounds environmentally hazardous often makes them economically efficient. The fact remains that these compounds persist for long time periods, such that they don't need as many applications to achieve the desired result. However, in the same cases, the alternatives are twice as expensive and need twice the number of applications to do the same job, in some instances. Consequently, there is a recognition that pest control costs can be significantly affected with the use of newer pesticide products, at least in some cases. It is important to note that specific evidence of this was not uncovered in this study, despite the identification of this cost impact in two of our expert interviews.

6.5.2 Compatibility of alternative technologies to existing technologies

If new pest control technologies can be rapidly integrated within current farming practices, then the new technologies tend to be more attractive to farmers and, as such, are accepted at a quicker pace. If, however, these new technologies require new farming practices or application technologies that are both knowledge and time intensive then they tend not to be adopted very quickly, for obvious reasons.

The gains from switching to an alternative are generally not immediately visible. Thus, not only may the alternatives be more expensive, but the benefits of switching are not accruing to the immediate user of the compounds. This, in combination with the fact that people may have to learn new methods of farming to use the alternatives and completely switch away from the methods that they have used for generations, suggests that the incentives to switching to the new technologies are not great nor are the new technologies easily implemented. For example, the use of biological controls within an IPM framework provides an effective and cost-efficient alternative to some hazardous chemicals. However, this new set of technologies requires new pest monitoring techniques, new application technologies and possibly a new crop production mix. Such necessary components of IPM are costly to implement and require a relatively high degree of knowledge. This does not necessarily mean that IPM and other new pest control technologies are not used in the developing world. In fact, there are very common practices, however, they tend to be used almost exclusively by larger and more "advanced" farmers or producer organizations. The problem remains primarily with smaller and often non-mainstream producers who tend to be less advanced in production technologies and usually rely on "traditional" technologies and approaches to crop production.

The users of the older compounds trust the products to do what is needed and they know the products well enough to know how to use them in a general sense. It may also be the fact that the pesticides that are currently being used are seen as the only means or input to control pests and thus they are readily accepted (Kebede *et al.*, 1990). The users of these products have a certain affinity towards the products in the sense that their fathers and grandfathers used the products and so they will continue to use what is proven to work. A common characteristic with this group of producers is they tend to be less educated and more comfortable with known systems. Many producers are illiterate or simply do not have the same degree of information available to them about alternatives as other producers. This reflects a common problem of poor information dissemination throughout the developing world. In an effort to improve this deficiency it is also necessary that accurate data be available on where and how hazardous products continue to be used. More important, it is necessary that one understands the reasons and rationale for their continued use.

6.5.3 Knowledge of alternatives and education of pesticide users

As noted previously, the continued use of some persistent organic pollutants or other hazardous compounds is a cultural issue as much as it is an educational issue. The users of the older products may not have all of the information necessary to make the decision between all of the possible alternatives, assuming that they are offered all the alternatives. To the farmers and labourers in the Philippines, there is a common belief that insecticides are always a solution to insect pest problems and never the cause of them. Even without a threat from serious pest attacks farmers increasingly applied pesticides, particularly insecticides (Pingali and Roger, 1995).

In the case of rice production in the Philippines, farmers obtain their knowledge about pesticide dosage from government technicians, pesticide sales people, pesticide labels and from other farmers. Proper training about the correct usage of the pesticides is vital at all of these levels. Training on assessing the presence of pests, the perceived intensity of infestation, the transplanting date, and other factors such as fertilizer application dates should all be included in training on the proper use of pesticides (Pingali, 1995). It is when the end user does not know the hazardous nature of the chemicals that improper and dangerous uses can occur.

Likewise, in Honduras, vendors are the primary source of information regarding pesticide use. They are also the suppliers of the pesticides. In this context, there is very little information regarding risks to farmers and alternatives products. The exception to this appears to be the larger corporate farms

that employ educated crop protection specialists. The problem remains that smaller and unorganized producers do not get the same information. Local vendors to these smaller producers are often the only point of contact regarding the use of pesticides.

The inefficient use of the persistent organic pollutants could be combatted through increased training at the vendor level as to how to use these chemicals, when to use them, and how much to use (Grenier, 1995). One of the recommendations that has been suggested to encourage the use of the alternatives is to ensure that the exporters of the pesticides from developed countries are training the farmers and the pesticide vendors. A simple manual could be prepared for the vendors to ensure that they have the information that they need. The manual should include or training should be done on what the individual products are used for, as well as how much to use and when (Grenier, 1995).

One such initiative to improve communication about pesticide uses, risks and alternatives has been undertaken by the Ministry of Agriculture in Honduras with the help of the German Government. This program resulted in the development of a manual/guide on pesticide use, handling, risks, and alternatives, that is easy to read and understand. More important, this manual was sent to rural primary school teachers for use in the classes. The objective is to educate the younger generation in the hopes that they will make more informed, environmentally sound decisions regarding pesticide use, and that they can influence common practices being conducted by their parents. The impact of the guide is expected to be seen in 5 to 6 years once the children go home and use their knowledge as well as teach their parents and other workers what they have learned from the manual. This project has the potential to achieve these objectives, however, the manuals are not being used in the schools. Although is not entirely understood why they are not being used, it could be that the teachers simply do not have the resources necessary to use the guide, or that they do not have the skills to teach the guide. This suggests that, if the policies brought forth to help the end users switch from the persistent organic pollutants are not implemented at the lower levels of the distribution chain, then the resources used to initiate the policy are wasted. Policies must be as simple and as cheap as possible in order to ensure implementation down the line (Grenier, 1995).

It is evident that a large concern for the people of developing countries is the misuse of the hazardous chemical compounds. Some of the pesticides are being used to such an extent that they are sprayed up to 10 times more frequently then necessary (Grenier, 1995). "Both anecdotal evidence and available data on Philippine farmers' use of pesticides indicate that they do not typically utilize recommended doses nor do they utilize the chemical industry's recommended practices for safe storage, handling, and application" (Pingali). Such misuse is not only economically inefficient but also hazardous to the people in the area being sprayed. This type of misuse also results in more rapid selection of resistance so that the compound becomes ineffective against its target sooner.

Most experts in developing countries fully recognize that, when spraying a broad-spectrum pesticide such as DDT, many other organisms are affected, not just the targeted pest. All natural predators to the pests as well as all other organisms being sprayed are likely to be affected. When a crop is over-treated to the degree described above, the possibility of secondary pest outbreaks becomes a concern. For example, currently, in Honduras, whiteflies have become a problem, especially on tomatoes. The whiteflies have been selected for resistance to the pesticide and now applications ten times the necessary levels have to be used routinely to control the pests because their natural enemies have been eliminated because they are susceptible. When the ecological balance is disrupted, the effects are sometimes surprising as well as damaging. This is a real concern for new products, particularly in the early adoption period as farmers learn to use them

appropriately. It is very important that the end users of the chemicals are fully trained in their use and understand the consequences of misuse (Grenier, 1995).

Not only is over-application a concern but so is under-application. When not enough of the chemical is used, the application may only serve to increase the selection pressure for resistance to be selected to the chemical.

Less persistent pesticides are often more toxic to humans. The application use of these without proper equipment presents a safety issue. Specialized protective clothing is not generally available (Pingali, 1995) nor is it entirely clear that the protective clothing used in the developed countries is adaptable to the climates in the developing countries. All respondents to the Philippine study use a metal knapsack sprayer, both for rice and vegetables. Sprayers may leak, so that the chemical drips on to the applicators' back, or from the hose onto his hands. Even though the sprayers are used a great deal, their maintenance appears to be a very low priority. Also, most farmers in the study spray ahead of themselves and therefore walk through the freshly sprayed vegetation where dislodgeability is highest. Without the proper protective clothing they are thus exposed to the chemical through the skin (Schillhorne, 1995). Precautions must be taken to ensure that the applicator as well as the surrounding area is fully protected from such hazards. It is not, however, evident what these precautions should be and how they should be implemented.

One way to encourage efficient usage of the compounds as well as the alternatives, is to ensure that the labelling of the chemicals is appropriate. Sometimes these labels are not in the native language or the user is illiterate and can't read the instructions. Pictograms have been used but are sometimes vague and not informative enough. If the users cannot understand the instructions, then misuse of the product is very probable and may be hazardous. This may contribute to the long history of excessive pesticide use in some of the developing countries.

There is a perception that the older persistent organic pollutants are less hazardous to human health compared to other products. This is particularly important to farmers when they observe the efforts being made to implement safer pesticide use and handling practices with hazardous compounds like organophosphorous compounds in mind. The point remains that such compounds are believed to be and are, in fact, more acutely toxic to humans than some persistent organic pollutants.

6.5.4 Regulatory constraints

From a policy standpoint, neither the government infrastructure nor the human resources necessary to make such a system work are sufficiently developed in many countries. Schillhorne (1995) commented that most of the developing countries simply do not have the regulatory framework, such as an environmental protection agency, in place and, if they do, they do not have the resources or manpower to implement and/or enforce the regulations. In Honduras, for example, there is an official list of products that can be imported into the country. Theoretically if an item is not on the list, then it is not allowed into the country. However, due to these regulatory constraints there exists an "informal trade network" that may allow the products into the country. While it is good policy to have a restricted list such as this, as well as regulations regarding hazardous chemicals, there is the need to enforce them as well. Currently, many developing countries simply can not afford the necessary regulatory infrastructure. Some countries, like Honduras, are in the process of re-organizing, after which they should be in a better position to enforce the regulations (Grenier, 1995).

There is also export policy issues in some pesticide producing countries where the policies in place have often induced higher pesticide use in developing countries through the provision of direct or

indirect subsidies on chemical pesticides. This created an environment where large quantities of pesticides could be exported to developing countries at low prices. Therefore regulatory problems are not simply those of the developing countries but are issues in developed countries as well, (Furan, 1994).

It has been noted by some international development experts that one of the greatest mistakes that the developed world made was to try to implement the regulatory and institutional structures in the developing countries that are in place in the developed countries (Brown, 1995; Gulliver, 1995). Some of the developing countries that have adopted these structures have only one person to run the whole department for the entire country while in the developed country the same department would have 100 employees (Taylor, 1995). Most developing countries, such as Honduras, simply do not have the resources to follow an EPA or Canadian model (Grenier, 1995).

The regulations are also a problem onto themselves. They must be as uncomplicated as possible. The structures and regulations used in developed countries are in many cases too complex to be adaptable to developing countries. An infrastructure model that is uncomplicated, inexpensive and more in line with the real issues in developing countries should be developed to solve this problem (Grenier, 1995; Brown, 1995).

Schillhorne (1995) also points out that the older chemicals are the only ones manufactured in the developing countries. This is because they are relatively easy to make compared to the newer alternatives. As a result, it is the developing countries, not the developed countries, that are producing these compounds now (Harris, 1995; Taylor, 1995). Another trend in the manufacturing of these compounds seems to be that it is not so much the multinational corporations that are producing the persistent organic pollutants but instead it is the small, independent manufacturers in the developing countries (Taylor, 1995). This makes the monitoring and enforcement of regulations in these small, independent manufacturers much more difficult. These manufacturers are also being very aggressive in their advertising campaigns in the developing countries.

There are also significant problems in the export market for produce treated with persistent organic pollutants. This is due to the lack of international cooperation in setting maximum residue limits (MRLs) that are globally accepted. For example, Guatemala's major export is snowpeas. In fact, they are the largest snowpea exporters in the world. This crop demands high labour and low capital costs and is thus dominated by small farms. These small farms probably do not have the same degree of knowledge about pesticide uses and their possible alternatives. They, therefore, have little choice but to use the older compounds. This, however, becomes a problem in the export market. The smaller farms have an increased risk of contamination on their crops since the product must travel through 2 or 3 different vendors before reaching the export packager. This makes it very difficult to trace back to its source a residue of a product that has exceeded a guideline or that may not be registered for the crop. Regulation then becomes difficult in the best of situations. Guatemala does not have the influence or resources to register the alternative for use on the minor crop in the United States. To illustrate the point Guatemala's other major export is broccoli. Due to the fact that broccoli is a major crop in the USA, this crop is registered to use the same pesticide that is being used on snowpeas. Thus, when exported into the USA the residues on the broccoli are acceptable since pesticide is registered for use on the vegetable but the same residues on snowpeas are not acceptable. There is no real reason why the same pesticide can not be used on both vegetables. Therefore, the only factor is the regulations in the USA. Snowpeas are not a major crop and are therefore not registered through the USA's pesticide regulations. The fall back position for snowpea producers is therefore to use the older chemicals that are allowed to some degree on the produce as opposed to the alternatives that are disallowed. This issue has the potential to destroy the snowpea

industry which in 1994 was worth $6,000,000 (US) in Guatemala. One solution may be to initiate something akin to the minor crops registration program that Canada or the USA presently uses (Gulliver, 1995).

Guatemala also exports approximately $400,000/year in fresh fruits and vegetables to the USA. The strict quarantine regulations in the USA make it commercially risky to use products that may leave residues of products that are not registered for these products. If such residues are found, the product can be turned away from the US border and therefore its market. As a crop, fresh fruits and vegetables demand low labour but high capital costs. Production in this type of crop is dominated by corporate farms. The corporate farms are more knowledgeable and more educated due to the degree of capital and expertise needed to run the farms. This would include knowledge and information on alternatives and their uses of the persistent organic pollutants. These corporate farms also have the advantage of having their crop go from the field directly to the export packager. It is very easy then to trace back to the corporate farm any pesticide residues that may not be approved for the product (TerKuile, 1995). This type of structure lends itself very well to the use of alternatives, and they are used on these larger corporate farms. In Guatemala, therefore, both the alternatives and the older compounds are being used in the country for different crops, different reasons, as well as within different agricultural structures.

Often developing countries do not have the infrastructure in place for the safe disposal of these containers for water storage. There are many examples of this type of misuse and lack of understanding about the potential hazardous of the chemicals being used. One solution to this problem would be to make the containers returnable and have the developed countries help finance this initiative (Brown, 1995). In this way, the containers could be properly cleaned and either reused, recycled, or disposed of without the large potential for accidental poisonings. Another example is the fact that the majority of farmers in the Philippines have extremely unsafe pesticide storage and disposal practices, indicating a high probability of accidental exposure to the chemicals (Pingali, 1995). Again, this is of particular concern when farmers switch to seemingly more hazardous organophosphorous compounds.

These initiatives need to be tailored for use in developing countries. It must be understood that the market signals that influence those users in the developed world may not work in the same manner in the developing world. The use of incentives such as refunds on pesticide containers may ensure that all containers are brought back to the vendor in the developed world but this may not be possible in the developing world due to a lack of transportation infrastructure (including roads) or a host of other issues that are taken for granted in most developed countries.

7. RISK REDUCTION

7.1 RISK MANAGEMENT

The management of risks builds on the results of risk assessment using a broader set of tools that include technical and engineering techniques, economic assessments, societal approaches, and policy instruments to mitigate of health and environmental effects. The outcome of all risk management actions should be **the reduction of risk**. Prior to undertaking risk management, it is necessary to decide the priority in which risks must be managed and to maximize the reduction of risk for the available resources. This reduction in risks includes the consideration of the risk of alternative activities.

7.2 PRIORITIZING THE RISKS AND BENEFITS

Significant saving in resources can be achieved if risks are managed in order of priority. Harwell *et al.*, (1992) have proposed a method for evaluating and prioritizing risk to human welfare and the environment. The risks to be prioritized are disaggregated into a series of issues that can be ranked as follows and can also be applied to benefits (as modified from Harwell *et al.*, 1992):

The potential intensity of the risk or benefit. Intensity is ranked on an ordinal scale of 6 ranging from low to high as follows: "In some cases, high" < Low < Medium < High < Very High < Extremely High.

The extent of the risk or benefit. Extent is ranked on an ordinal scale of 4 ranging from low to high as follows: Community (very local) < Ecosystem (local) < Regional < Biosphere.

The recovery time or period of benefit. Recovery or benefit time is ranked on an ordinal scale of 4 ranging from low to high as follows: Very short (months) < Short (years) < Medium (decades) < Long (centuries).

On the basis of this approach, the risks from the persistent organic pollutants rank relatively highly as their intensity is medium, they range over the biosphere and the recovery time from these effects is of the order of decades. This rank applies to persistent organic pollutants that have no specific uses, such as the dioxins and PCBs. However, for some other persistent organic pollutants, this risk ranking must be interpreted using the same criteria to rank benefits. When this is done for those persistent organic pollutants that are used as pesticides to control vectors of human and animal diseases, the benefit (in the use area) is very high, the extent of the benefit is regional but the period of benefit is short because of reinvasion of the disease and its vectors. The critical point is the fact that there is a net benefit associated with the use of these substances in some areas whereas there is a net risk associated with their movement to and effects in other areas.

7.3 RISK MITIGATION AND TOOLS FOR MITIGATION

There is one objective in risk mitigation -to reduce exposure. Although the tools for use in mitigation are as varied as the number of substances, there are, in general, two major types of tools - technical and regulatory.

7.3.1 Technical tools

Technologies utilized to reduce production and releases of persistent organic pollutants are continuously changing and evolving. As a result, the legal framework for most regulations dealing with releases of persistent organic pollutants to the environment requires periodic review of these releases and the technology available to reduce them. In theory, this will ensure a continuous decline in releases to the point of *de minimus*. However, this process has a number of limitations. Even in developed countries, many regulatory agencies find themselves understaffed and are unable to undertake these regular reviews. As a result, public pressure is often necessary to initiate reviews within regulatory agencies (SETAC, 1996). This may hinder the development of control policies based on both credible science and economic risk/benefit analyses.

Technical tools for mitigation include a wide range of procedures, many of which are specific to the situation. Further treatment of effluents is commonly applied to industrial settings and, in the use of pesticides, many options from alternative chemicals to cultural practices are available. These options are the province of the process engineer and pest manager and will not be discussed further here.

7.3.2 Regulatory tools

The basic options for the regulation of risk mitigation are the following in increasing order of severity (SETAC, 1996):

(1) to provide better information and communication to prevent uninformed misuse (e.g., safety data sheets on use and disposal and hazard or transportation labels). This option has little relevance to the persistent organic pollutants because their chemical and physical properties are the major determinants of the problem, not their use pattern.

(2) to control discharges and releases of chemicals to particular levels that are judged to be safe at various points in the life-cycle of the substance, such as the establishment of air or water criteria or voluntary emission reduction programs. Given the lack of technical resources in areas where many of the persistent organic pollutants are used, this option is not viable.

(3) to restrict chemical use and release, such as restrictions on the use of lindane and pentachlorophenol in some countries.

(4) to impose a total manufacturing ban, such as the ban on the use of DDT and PCBs in some countries.

The implementation of restrictions on use and release of substances such as the persistent organic pollutants is a common regulatory practice. A significant number of chemicals have targeted environmental concentration limits such as air and water criteria established by regulatory agencies such as those in the US EP A environmental criteria for water (US EPA, 1992).

An alternative strategy is to review all phases of production, use, and disposal of substances that are to lead to the formation of persistent organic pollutants. These life-cycle assessments allow identification of substances released and their sources and assists in the choice of appropriate risk mitigation strategies.

Bans and restrictions have most often been used to control the use and release of specific substances (DDT) rather than process contaminants (dioxins and furans). While these restrictions have been effective in halting production within certain jurisdictional boundaries, production and use has often continued in other jurisdictions (DDT, hexachlorocyclohexane). In addition, these restrictions are obviously not retroactively effective in decontaminating manufacturing sites or other contaminated sites nor geographic regions such as the poles.

Implementation of bans may not be immediate, but may take place over a given time period. This option is referred to as a phase out. An example, where significant risks were identified as well as the need for time to convert manufacturing process, is the Montreal protocol and the London amendments for CFCs. In this case, replacement chemicals were identified (such as HFCs) that were acceptable from environmental, human health, and performance perspectives. The original protocol provided for a decade to convert manufacturing processes in industrialized countries and did not set specific deadlines for less developed countries.

7.3.3 Economic incentives/disincentives

There are several potential economic methods that can be used as strategies to reduce exposure from organochlorine chemicals. Basically, government actions impose or raise charges for activities that incur greater risks or attempt to reduce charges for those activities with lower risks. However, experience with these models is limited. These methods would be in addition to the growing environmental consciousness of both businesses and consumers in the purchase of materials and products.

Taxes. A "use tax" can be levied on the producer or user of a chemical where that chemical is deemed by policy makers to be harmful to the environment. Obviously, the effective tax must be equal to or exceed the costs of implementing normal technological emission controls. However, whether the economic disincentive provides better overall reductions in emissions than regulatory strategies is untested. This also results in a hidden tax to the consumer since these costs must be recovered by the producer.

Disposal fees. When a chemical of concern is a waste that cannot be recovered and where emission reductions are desired, disposal fees could be enacted to encourage various waste reduction/prevention processes. However, large manufacturing sites are increasingly utilising on-site disposal or destruction. For smaller manufacturing sites and small users, a disposal tax could become an incentive for improper disposal and dumping of wastes. This unfortunate situation is already recognized as an issue for hazardous wastes.

Trade sanctions. It has been proposed that trade sanctions could be applied to countries that continue to produce clearly harmful chemicals. However, given the increasing complexity of international trade agreements such as the General Agreement on Tariffs and Trade (GATT), it is unlikely that such sanctions could be implemented without extensive negotiations. National laws preventing use and importation are already in place for a number of persistent and bioaccumulative compounds like PCBs. Environmental treaties and protocols such as those for CFCs and hazardous wastes would be a more limited or more likely avenue to approach.

The following presents some possible targets for the wider application of economic instruments under the Canadian Environmental Protection Act (CEPA). The sections of CEPA cited are those that provide for regulation in the existing CEPA (Table 7-1, from Environment Canada, 1994).

Table 7-1 Potential application points for the use of economic instruments to control persistent organic pollutants under the Canadian Environmental Protection Act (CEPA).

Action	Target	Economic Instruments
Part II (Toxic Substances)		
34(1)(a)	the quantity or concentration of substance released alone or in combination with other substances	tradeable permit; emission/effluent charge
34(l)(e)	the manner in which and conditions under which there is release	tradeable permit ;
34(1)(e)	the quantity of the substance that may be manufactured, processed, used, offered for sale or sold	input/product charge; tradeable permit/quota
34(1)(f)	the purposes for which substance may be imported, manufactured, processed, used, offered for sale or sold (e.g.) • import-export • import/manufacture-recycle • import for specific purpose	deposit-refund; financial incentive
34(1)(g)	the manner in which and conditions under which the substance or a product containing the substance may be imported, manufactured, processed or used (e.g.) • import-export • packaging	deposit-refund; financial incentive
34(1)(h)	the quantities or concentrations in which the substance may be used	input/product charge
34(1)(k)	the manner, conditions and purposes of import or export	deposit-refund
34(1)(m)	the quantity or concentration of substance contained in any product manufactured, imported, exported or offered for sale (e.g.) • import-export • manufactured	deposit-refund; input/product charge
34(1)(p)	the packaging and labelling of a substance or a product containing a substance	deposit-refund; product(waste) charge-refund
35	authority to create interim orders	same as above ss.34(1)(a), (d), (e), (t), (g), (k), (m), (p)
36(1)	release in excess or regulated limits or in excess of permit	tradeable permit; emission/effluent charge
47(a)	specified concentration or quantity	input/product charge of any element, component or additive in fuel
Part III (Federal Departments, Agencies, Crown Co orations -Works Undertakings and Lands)		
54(1)	the protection: of environment with respect to federal works, federal undertakings or federal lands where no other Act of Parliament makes explicit provision for such regulations.	emission/effluent charge; product(waste) charge; tradeable permit
54(2)(a)	limits for emissions and effluents by federal departments, boards, agencies, and federal Crown corporations	emission/effluent charge; tradeable permit

Table 7-1 Potential application points for the use of economic instruments to control persistent organic pollutants under the Canadian Environmental Protection Act (CEPA).		
Action	**Target**	**Economic Instruments**
54(2)(b)	stipulation of permissible waste handling and disposal practices	product (waste) charge; deposit-refund; financial incentives
57	releases of a substance in contravention of a regulation under s.54(1) or s.54(2)	emission/effluent charge; tradeable permit
Part V (International Air Pollution)		
63(1)(a)	control of air contaminant alone or in combination with other air contaminants from a single source or more than one source	emission charge; tradeable permit; input/product charge

7.4 ASSESSMENT OF MITIGATION

Mitigation strategies to reduce the risks from persistent organic pollutants must be followed by assessments of the effectiveness of the decisions. There are three principle environments to consider:

(1) environments where the production of the persistent organic pollutant takes place,
(2) environments where the persistent organic pollutants are used, and
(3) environments where the persistent organic pollutants may appear as a result of their transportation.

Monitoring activities are needed to support the accuracy of assessments in receiving media such as water and air, in the vicinity of industrial sites, and in specific sinks such as sediments and biota. It is important to identify the specific purpose of such monitoring; whether to study long range transport, environmental persistence, bioaccumulation, or to assess cause and effect linkages. There are few arrangements in place to ensure that sampling and analytical procedures are comparable or that there is sufficient spatial and temporal co-ordination of sampling either among parties or among sites. This is a significant area for data improvement, particularly for long term trends (SETAC, 1996).

8. CONCLUSIONS

- The persistent organic pollutants described in this Review are characterized by their lipophilicity, persistence and semi-volatility. These characteristics pre-dispose these substances to long environmental persistence and to long-range transport. These substances are also known for their ability to bio-magnify and bio-concentrate under typical environmental conditions, thereby potentially achieving toxicological relevant concentrations. The semi-volatility of these substances facilitates their long-range transport to and accumulation in the cooler and polar regions of the world, far removed from any source of use.

- A number of the substances described in this Review have been implicated in a broad range of adverse human health and environmental effects including impaired reproduction and endocrine dysfunction, immunosuppression and cancer. In many cases, the substances are considered as possible human carcinogens by the International Agency for Research on Cancer.

- Many of the substances described in this Review are still in use in at least some countries. The paucity of reliable data regarding use and disposal has meant that it has not been possible to accurately determine the quantities still in use, where they are used, the specific crops to which the pesticidal substances are being applied, and the direction and initiatives underway to eliminate these substances throughout the world. Where data does exist, it is plagued with a variety of limitations making it difficult to develop comprehensive and accurate use profiles.

- While convincing substantive evidence exists for the actual and potential toxic impact of these substances to both human health and the environment, a comprehensive, accurate and reliable inventory of global manufacture, use and disposition must be developed to allow the effective and efficient elimination of these substances throughout the world.

- Several risk reduction strategies are available for the persistent organic pollutants. They involve greater use of alternatives to substances still in use and proper disposal of persistent organic pollutants in storage or in closed systems (e.g., PCBs). As this is a global problem, these strategies need to be coordinated on a global level and must be tailored to the available resources and socio-economic constraints of user nations.

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10. GLOSSARY

bioconcentration: the uptake of a contaminant by aquatic organisms where water is the sole contaminant source (Macek *et al.,* 1979).

bioaccumulation: the uptake of a contaminant from both water and dietary sources (Macek *et al.,* 1979).

biomagnification: the processes of both bioconcentration and bioaccumulation that result in increased tissue concentrations of a contaminant as it passes through two or more trophic levels in the food chain. (Macek *et al.,* 1979).

CAS: Chemical Abstract Services registry number congener: refers to a group of dioxins, furans or PCBs having the same number of chlorine atoms. e.g. tetrachlorodibenzo-p-dioxins are a congener group

IARC Classes (IARC 1991):

Group 1-The agent (mixture) is carcinogenic to humans.
This category is used only when there is sufficient evidence of carcinogenicity in humans.

Group 2A-The agent (mixture) is probably carcinogenic to humans.
This category is used when there is limited evidence of carcinogenicity to humans and *sufficient evidence* of carcinogenicity in experimental animals.

Group 2B-The agent (mixture) is possibly carcinogenic to humans.
This category is generally used for agents (mixtures) for which there is *limited evidence* of carcinogenicity in humans in the absence of *sufficient evidence* of carcinogenicity in experimental animals. It may also be used when there is *inadequate evidence* of carcinogenicity in humans or human data are nonexistent but there is *sufficient evidence* of carcinogenicity in experimental animals.

Group 3-the agent (mixture) is not classifiable as to its carcinogenicity to humans.
Agents (mixtures) are placed in this category when they do not fall into any other group.

Group 4-The agent (mixture) is probably not carcinogenic to humans.
This category is used for agents (mixtures) for which there is *evidence suggesting lack of carcinogenicity* in humans together with *evidence suggesting lack of carcinogenicity* in experimental animals.

isomer: refers to a specific chemical. e.g. 2,3,7 ,8,-tetrachlorodibenzo-*p*-dioxin.

K_H: Henry's Law Constant is defined as the ratio of the partial pressure of a compound in air to the concentration of the compound in water at a given temperature under equilibrium conditions. It provides an indication of the relative volatility of a substance (Montgomery, 1993). If $K_H < 10^{-7}$ atm m^3/mol, the substance has a low volatility. If $K_H > 10^{-7}$ but $< 10^{-5}$ atm m^3/mol, the substance will volatilize slowly. Volatilization becomes an important transfer mechanism in the range of $10^{-5} < K_H < 10^{-3}$ atm m^3/mol. Values of $KH > 10^{-3}$ atm m^3/mol indicate volatilization will proceed rapidly (Lyman et at., 1982: cited in Montgomery, 1993).

LOEL: lowest observed effect level.

LOAEL: lowest observed adverse effect level.

LC_{50}: concentration required to kill 50 % of the test organisms.

LC_{50}: dose required to kill 50 % of the test organisms.

log K_{oc}: The soil/sediment partition or sorption coefficient is defined as the ratio of adsorbed chemical per unit weight of organic carbon to the aqueous solute concentration. It provides an indication of the tendency of a chemical to partition between particles containing organic carbon and water (Montgomery, 1993).

log K_{ow}: The K_{ow} of a substance is the *n*-octanol/water partition coefficient and is defined as the ratio of the solute concentration in the water-saturated *n*-octanol phase to the solute concentration in the *n*-octanol-saturated water phase. It is an important parameter in predicting the environmental fate of organic compounds, and has been shown to be linearly correlated with log bioconcentration factors in aquatic organisms (Montgomery, 1993).

NOEL: no observed effect level.

NOAEL: no observed adverse effect level.