

MONS 223164



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Subject: THE TOXICITY OF PCBs, THEIR CONTAMINANTS AND COMBUSTION PRODUCTS IN HUMANS, AND THEIR PERSISTENCE IN THE ENVIRONMENT

Gentlemen,

I am pleased to send you the English version of the above mentioned report of which I am the author. It has now been published.

If you need any further information, do not hesitate to contact me at the following telephone number (514) 289-2970.

Good reading!

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THE TOXICITY OF PCBs, THEIR CONTAMINANTS AND COMBUSTION PRODUCTS IN HUMANS, AND THEIR PERSISTENCE IN THE ENVIRONMENT

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June 1986

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INTRODUCTION

Polychlorinated biphenyls (PCBs) pollute land and water ecosystems and are found in almost every part of the world. They have been involved in two large-scale poisonings caused by food contamination, the first in Japan in 1968 and the second in Taiwan in 1979. In the early 1970s, at a time when polychlorinated biphenyls were quite common, a number of studies carried out in the United States showed that they appreciably contaminated the food chain, both directly and indirectly. Several health problems among exposed populations in industrial environments have been attributed to PCBs or their contaminants.

More recently, a new problem has been brought to light, i.e. the combustion of polychlorinated biphenyls. Under the effect of heat and given certain conditions, a portion of PCBs in combustion are converted into polychlorodibenzodioxins (PCDDs) and polychlorodibenzofurans (PCDFs), of which a number of congeners are deemed to be extremely toxic.

The principal source of the polychlorinated biphenyls still in use is electrical equipment insulated with these products, commonly called askarels. The generic term askarels designates fire-proof dielectric liquids, composed of polychlorinated biphenyls, to which polychlorobenzenes (tri- and tetrachlorobenzenes) have been added.

Moreover, we have been aware for several years that mineral oils used to insulate electrical equipment have often been contaminated by polychlorinated biphenyls; between 10 per cent and 15 per cent of such oils appear to be contaminated with PCBs in concentrations above 50 ppm (parts per million). These oils are covered by regulations governing hazardous wastes, designed to prevent discharging of PCBs in the environment and to eliminate these substances in Canada.

For the general public, the expression polychlorinated biphenyls is synonymous with danger. Given their persistence in the environment and in living organisms, people fear their effects on health and, more specifically, their consequences for reproduction and breast-fed babies, and their carcinogenic effects.

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The considerable number of quality publications devoted to PCBs bears witness to the extensive broadening of knowledge of the potential effects of PCBs on humans and the environment. For more than 10 years, scientists have closely followed poisoning victims in Japan and Taiwan and individuals subjected to long-term occupational exposure. Thanks to such follow-up and experiments on animals, it is possible to derive a clearer idea of the possible pernicious effects of these products on humans.

In light of documentation available on polychlorinated biphenyls, their contaminants and combustion products, we will attempt in this study to:

- assess the overall behavior of PCBs in the environment;
- assess the potential effects of these products on the health of humans and animals;
- assess, as far as possible, the relationship between exposure dose and effects;
- estimate, bearing in mind legislation currently in force, current and future risks of the appearance of these effects in the population and among the most heavily exposed workers;
- ascertain future prospects concerning these products.

We hope to answer a number of questions which many people are asking themselves with regard to the persistence of these products in the environment and their toxicity for humans.

1. HISTORICAL OVERVIEW

1881 (Germany)	PCBs were synthesized for the first time.
1929 (United States)	PCBs were marketed in the United States by Universal Manufacturing Equipment Co., under a licence from F.M. Clark for the use of PCBs as a dielectric, under the brand name Askarel.
1932 (United States)	Monsanto manufactured PCBs under the brand names Aroclor and Pyroclor.
1941 to 1947	Use of PCBs became more widespread in Europe and Japan.
1966 (Sweden)	Dr. Jensen discovered the presence of PCBs in the tissues of fish and piscivorous birds. To study residues of pesticides such as DDT and p,p'-DDE in these animals, he analysed samples by chromatography and discovered new contaminants which did not correspond to any known chlorinated pesticide (see Figure 1). This discovery showed that PCBs accumulate in organic tissues.
1967 (Sweden)	Through mass spectrometry, Wilmark identified the chemical nature of these contaminants, which proved to be PCBs. Subsequently, it was discovered that, because of their solubility in lipids and their resistance to degradation, PCBs accumulate in the food chain and are dispersed in the environment in a manner similar to polychlorinated pesticides such as DDT.

Since 1966, researchers around the world have focused their attention on the possible toxic effects of PCBs. Numerous publications have appeared concerning acute and chronic toxicity in several animal species.

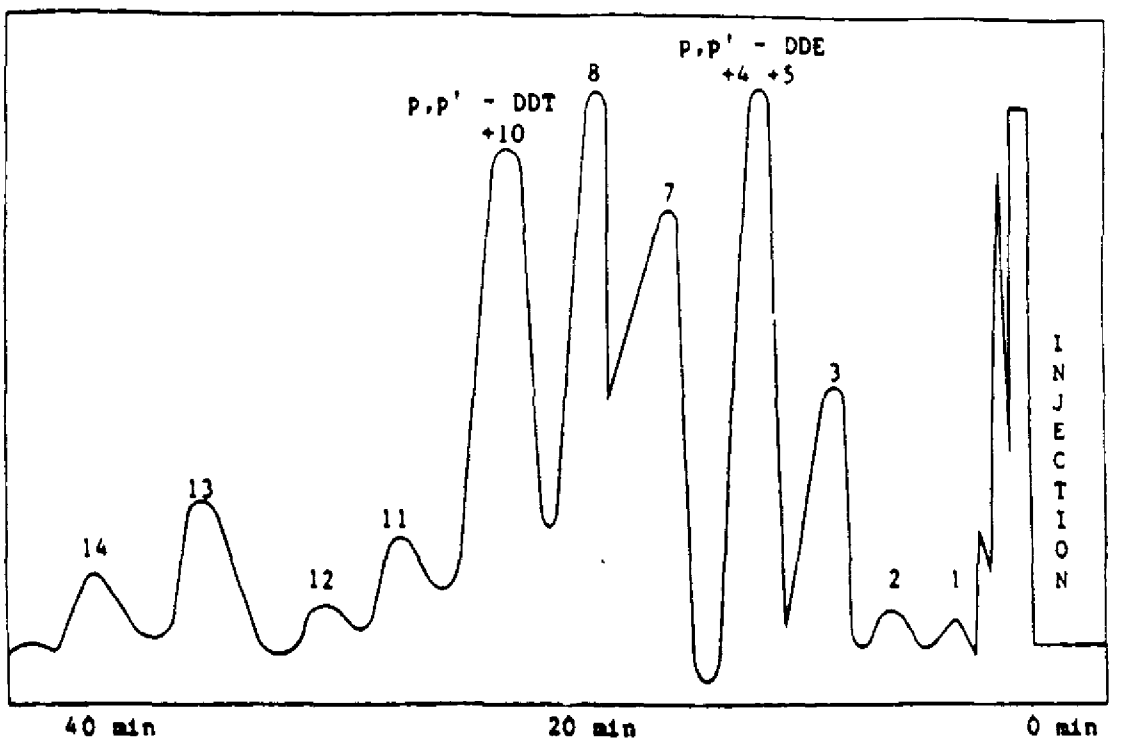


FIGURE 1: Chromatogram of e Tissue Sample from a Dead Eagle Found in the Stockholm Archipelago. Except for DDT and DDE Peaks, All Peaks Appearing on the Graph Were Unknown (courtesy of Dr. Jensen)

1968 (Japan)

The Yusho incident, a mass PCB poisoning, occurred. For more than eight months, Japanese consumed rice oil contaminated with PCBs and furans from a heat exchanger used on the production line. More than 1800 people displayed clinical symptoms (chloracne, edema of the eyelids, meibomian gland disorders, hepatic lesions, lethargy and so on).

Today, it is estimated that victims consumed between 0.5 g and 2 g of PCBs and between 2.5 mg and 7 mg of furans.

The Yusho incident led to measures around the world to regulate the use and production of PCBs.

1969 (United States)	Contaminated milk was found in West Virginia, attributable to pastureland contaminated by herbicides containing PCBs.
1970 (United States)	The Environmental Protection Agency (EPA) established a group responsible for evaluating the extent of PCB pollution and coordinating the efforts of U.S. agencies working on the problem.
1970 to 1971 (United States)	A series of incidents occurred involving the contamination of food products by PCBs.
	Contaminated milk was found in Ohio and other states.
	At the Campbell Soup Co. in New York, chicken was contimated by wrapping peper containing PCBs.
	In Illinois, flour made from meat by-products was contaminated because of a leak in heat transfer equipment.
	In North Carolina, fish flour was contaminated by a heat exchanger during pasteurization. Prior to discovery of the problem, the flour had contaminated many chickens and eggs.
1971 (United States)	Monsanto, manufacturer of Aroclor, limited its products to applications in closed systems such as transformers and capacitors and introduced a new product, AR 1016, that is more biodegradable than previous Aroclors.
1972 (United States)	The Council of Environmental Quality (CEQ) published a report which concluded that PCBs undeniably caused pollution. It recommended that PCBs be used only in applications where they were truly essential and could not be replaced by other products. It also recommended limiting contact with PCBs.
1972 (Japan)	Manufacturing and use of PCBs were prohibited.

1973 (OECD)	The council of the Organization for Economic Cooperation and Development (OECD) decided to regulate the use of PCBs in member countries. The notion of open systems (in which PCBs were henceforth banned) and closed systems was introduced.
1973 (United States)	The Food and Drug Administration (FDA) introduced a new regulation limiting the use of PCBs, in any form, in food production chains. The FDA also established the maximum concentrations of PCBs tolerated in food and materials coming into contact with food.
1975 (Canada)	Environmental Contaminants Act, Ch. 72.
1976 (United States)	The Toxic Substances Control Act (TSCA) stated that PCBs are extremely toxic and carcinogenic and requested that Congress regulate their use.
1977 (United States)	The EPA prohibited all discharges of PCBs in waterways. The TSCA asked the EPA: (1) to prohibit the manufacture of PCBs; and (2) to regulate the identification and elimination of material containing. or having contained, PCBs.
1977 (United States)	Monsanto ceased manufacturing Aroclors in the United States and Pyroclors in England.

1977 (United States)	It was noted that mineral oil in many transformers was contaminated by PCBs. A campaign to systematically determine the PCB content of such oil was launched.
1977 (Canada) August	Chlorobiphenyls Regulation No. 1 respecting the use of PCBs was adopted pursuant to the Environmental Contaminants Act (ECA).
1977 (Canada) December, Adelaide	A fire occurred in a transformer containing 380 L of askarel, part of which evaporated. Soot samples collected after the fire contained approximately 5 ppm of polychlorodibenzofurans (PCDFs). Following a study of reports of analyses effected during and after the fire, the Department of the Environment concluded that there was no longer a public health risk.
1978 (Sweden) September, Stockholm	Fire broke out in a substation, destroying 18 capacitors containing askarel; no poisonings were reported.
1971 (United States)	The EPA established a classification of material according to its PCB content. Category 1: 0-50 ppm - Fluids and apparatus with this content are deemed to be free of PCBs. Category 2: 51-500 ppm - Fluids and apparatus with this content are deemed to be contaminated by PCBs. Category 3: ≥ 500 ppm - Fluids are considered to be ''pure'' PCBs and apparatus to contain ''pure'' PCBs.
1979 (Taiwan)	Eleven years after Japan, Taiwan experienced a poisoning through PCBs and furans, also from contaminated rice oil. More than 2000 people displayed symptoms similar to those observed among victims of Yusho disease.

1980 (Canada)	Amendment of Regulation No. 1 on the use of PCBs.
1980 to 1982 (United States)	The Electric Power Research Institute (EPRI) and producers and distributors of electricity in the United States launched extensive studies to ascertain the toxicity of PCBs and develop accurate methods for identifying them in insulating oils.
1981 (United States) February, Binghamton, NY	A fire occurred in a transformer containing 680 L of insulating fluid, comprising 60 per cent PCBs and 40 percent tetrachlorobenzene. Soot containing PCBs and products such as TCDDs and TCDFs contaminated the entire 18-storey building.
1981 (United States)	A new EPA regulation was introduced respecting the use of PCBs and material containing them. However, the 0-50 ppm, 51-500 ppm and ≥ 500 ppm categories were maintained.
1983 (United States) San Francisco	A fire occurred in a transformer, contaminating the building with PCBs and breakdown products.
1984 (Canada)	Proposed regulations respecting environmental contaminants: Regulation No. 2: standards setting the maximum concentration of PCBs in certain products. Regulation No. 3: standards setting the maximum concentration and quantity of PCBs which can be discharged in the environment.
1984 (Canada) November, Québec	A fire occurred in the high-voltage laboratory of the Institut de recherche d'Hydro-Québec (IREQ); 670 L of PCBs and 2560 L of mineral oil burned. The entire building was contaminated by smoke and soot containing PCBs and its breakdown products, especially PCDFs.
1985 (Canada) January, Québec	A fire inside Hydro-Québec's Wellington substation destroyed 6 L of PCBs in a capacitor.

1985 (Canada) April, Kenora, Ontario	PCBs were spilled from a leaking transformer being shipped from Québec to Alberta.
1985 (Canada)	Several small spills of oil contaminated with PCBs occured in various locations in Canada during the shipment of transformers.
1985 (Canada) July	Chlorobiphenyls Regulations No. 2 (Product) and No. 3 (Release) were adopted with respect to PCBs.
1985 (Québec)	The Hazardous Waste Regulation was adopted, which is much more stringent with respect to PCBs than regulations elsewhere in Canada. Maximum concentrations of PCBs: . 0.15 mg/L or 0.15 ppm in liquid residues; . 0.01 mg/L or 0.01 ppm in solid residues of lixiviation.

This brief overview of the history of PCBs indicates the concerns of the general public, workers and governments with regard to these products.

2. THE CHEMISTRY OF POLYCHLORINATED BIPHENYLS (PCBs)*

2.1 Definition of PCBs

Polychorinated biphenyls (PCBs) do not exist in a pure state in nature, but are chemicals which have been synthesized from benzene and sodium chloride. The basic unit of BPCs is the biphenyl, which is obtained by vapor treatment of benzene at high temperatures in the presence of metal catalyzers. By replacing hydrogen molecules with chlorine (Cl), molecules of chlorinated biphenyls or chlorobiphenyls are created.

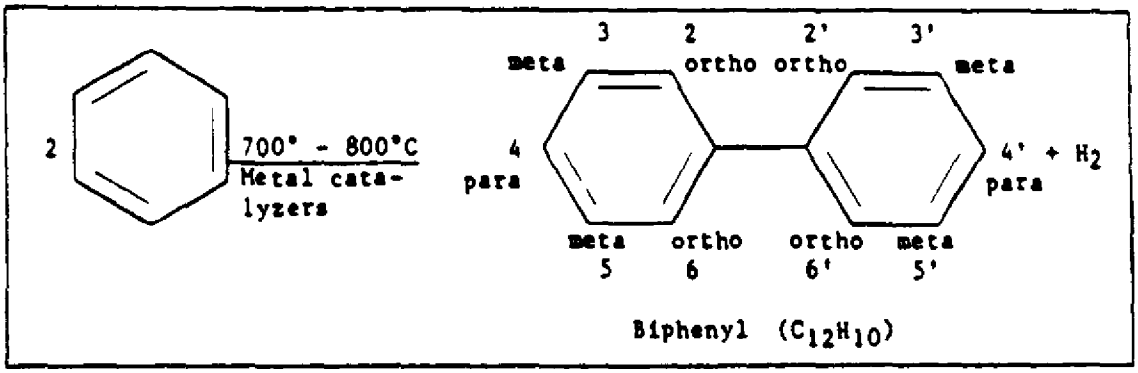


FIGURE 2: The Biphenyl Nucleus Containe Hydrogen at Positions: 2, 3, 4, 5, 6, 2', 3', 4', 5', 6'

There are 209 possible congeners corresponding to the different positions which Cl atoms can occupy on the biphenyl nucleus during their synthesis. The congenera can contain from 1 to 10 chlorine atoms, which represents 10 series of homologous compounds comprising congeners or isomers containing a given number of chlorine atoms.**

---

* See The Chemistry of PCBs, O. Hutzinger et al., 1974.

** Congener is a general term designating the isomers of all series of homologous compounds. There are 209 congeners, encompassing all possible molecules of polychlorinated biphenyls.

A homologous compound is defined as a group made up of isomers containing a given number of chlorine atoms. All congeners containing four chlorine atoms are isomers in the tetrachlorinated homologous compound series.

In a homologous compound series, isomers are distinguished by the position of chlorine atoms.

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2.2 Nomenclature of PCBs

The different molecules of polychlorinated biphenyls (congeners or isomers) are named according to the homologous compound series to which they belong and the positions of chlorine atoms on the biphenyl molecule (see Table 1).

TABLE 1: Nomenclature of 10 Groups of Isomers in the PCB Family

Number of chlorine atoms	Names of the isomers of the 10 series of homologous compounds
1	Monochlorineted biphenyl (monochlorobiphenyls)
2	Dichlorinated biphenyl (dichlorobiphenyls)
3	Trichlorinated biphenyl (trichlorobiphenyls)
4	Tetrachlorobiphenyls
5	Pentachlorobiphenyls
6	Hexachlorobiphenyls
7	Heptachlorobiphenyls
8	Octachlorobiphenyls
9	Nonachlorobiphenyls
10	Decachlorobiphenyls

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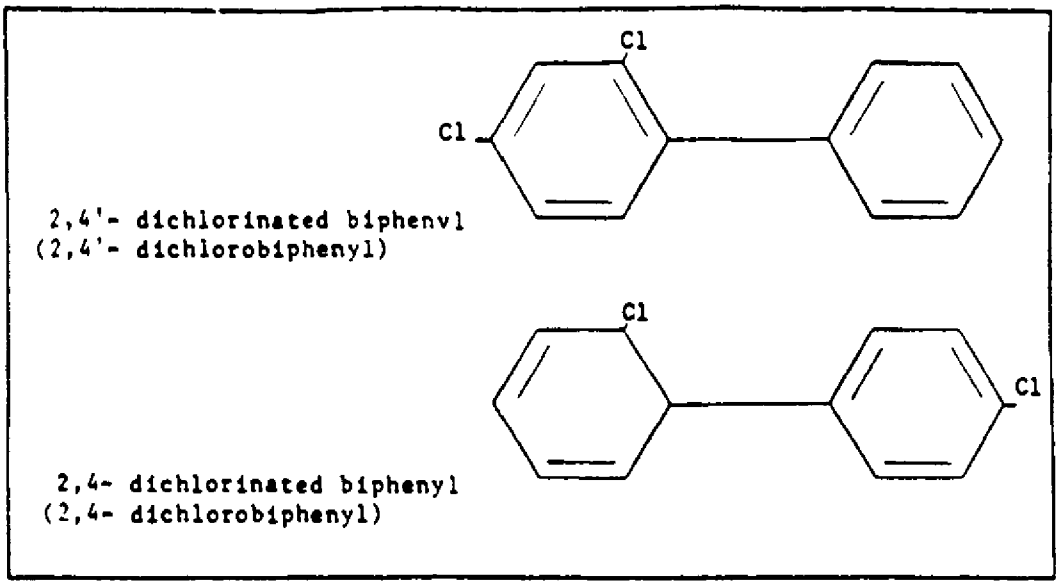


FIGURE 2.1: Two Examples of Dichlorinated Biphenyls or Dichlorobiphenyls

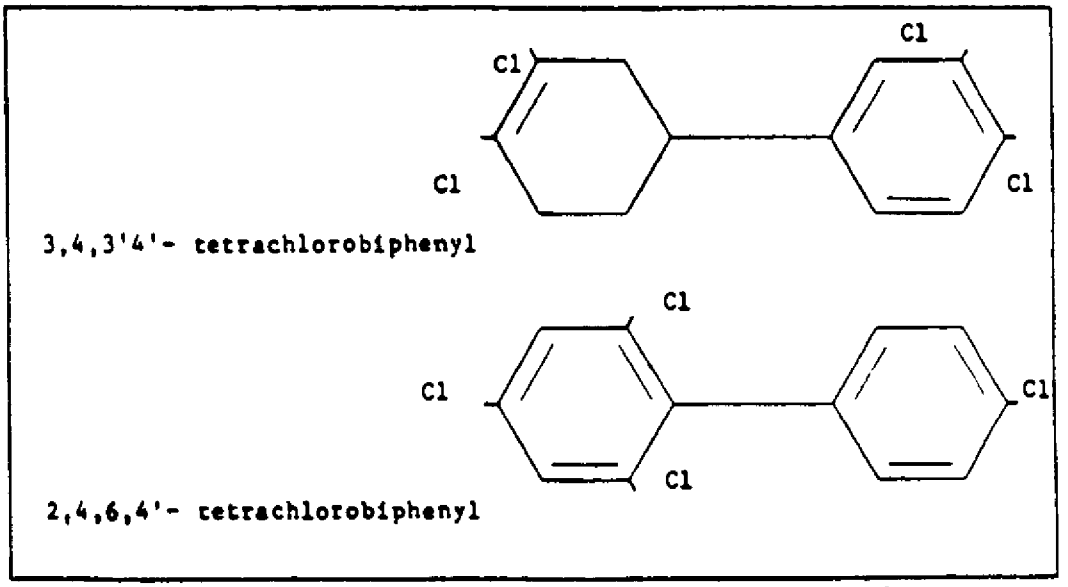


FIGURE 2.2: Two Examples of Tetrachlorobiphenyls

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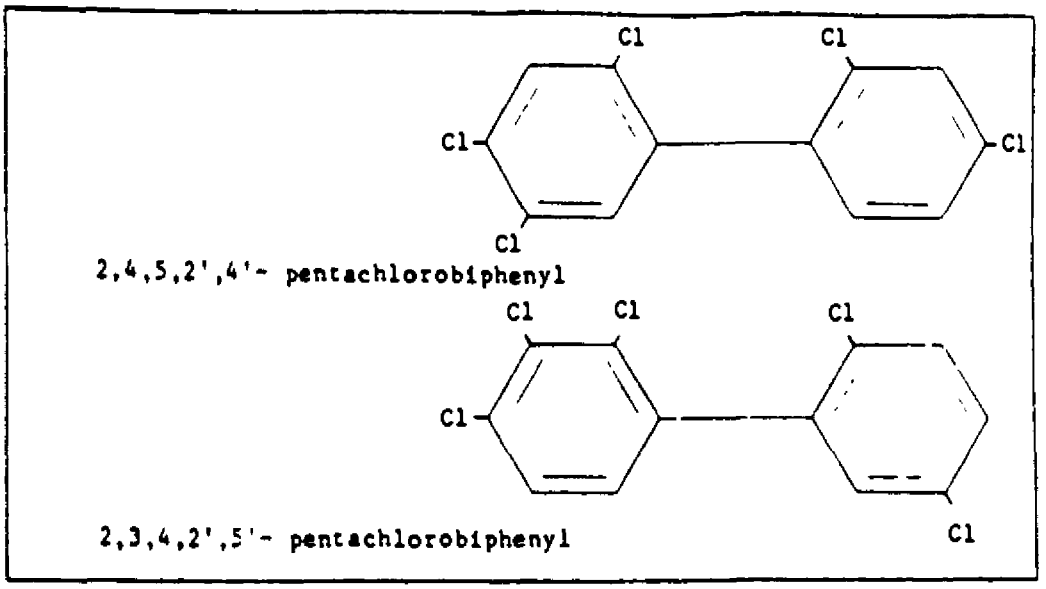


FIGURE 2.3: Two Examples of Pentachlorobiphenyls

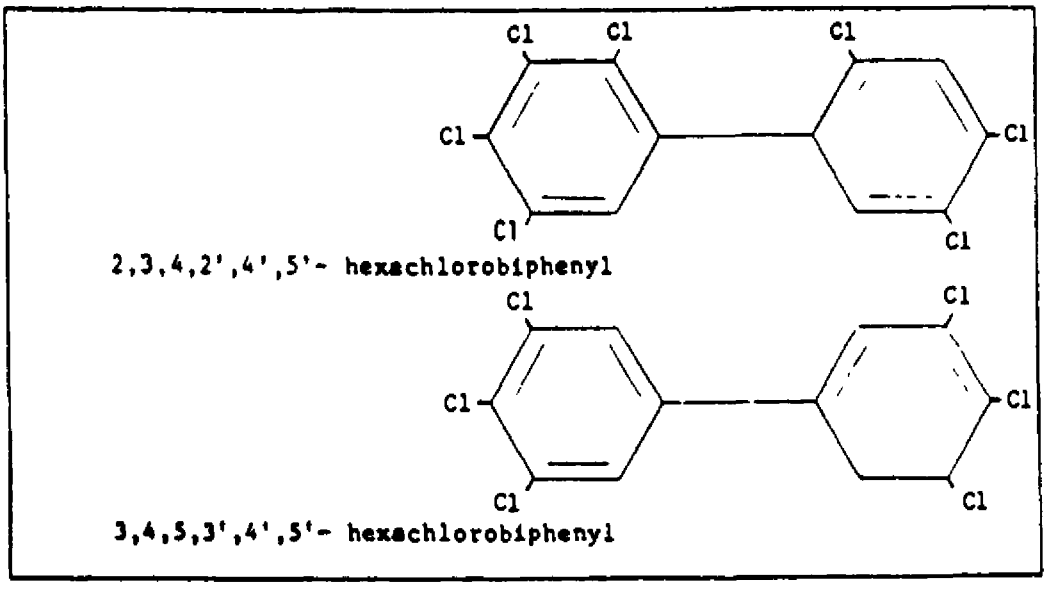


FIGURE 2.4: Two Examples of Hexachlorobiphenyls

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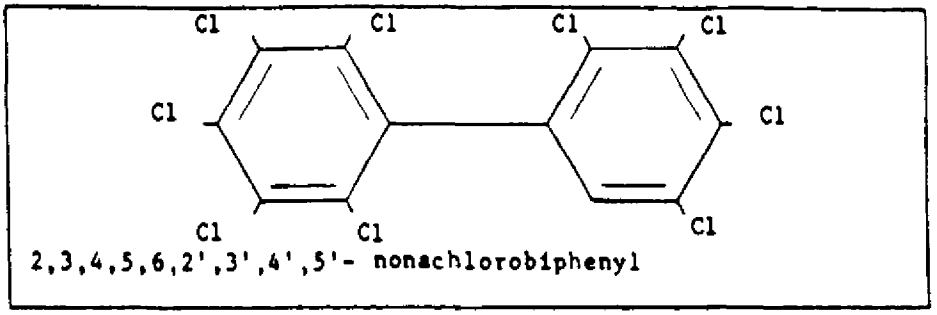


FIGURE 2.5: One Example of Nonachlorobiphenyls

2.3 Principal Properties of PCBs

PCBs have the following properties, some of which (those marked with an asterisk) are extremely important from a biological standpoint.

- Very heat stable, fire-resistant
- Very stable chemically*
- Very resistant to oxidation, acids, bases and other chemicals*
- Very high boiling points
- Very good dielectric properties (insulators)
- Only slightly soluble in water*
- Highly lipophilic*
- Very low volatility in air*
- Recognized toxicity*

2.3.1 Remarks on properties having a biological effect

2.3.1.1 Very stable chemically, very resistant to oxidation, acids, bases and other chemicals

These characteristics make PCBs in general very resistant to biological and chemical degradation. However, it is recognized that the lower the chlorine content of PCBs, the more readily biodegradable they are, and vice versa.

It must be pointed out that the position of chlorine atoms affects persist-.nce, i.e. the various isomers with the same chlorine content do not have the same effect.

2.3.1.2 Only slightly soluble in water

PCBs are not generally soluble in water and tend to settle in organic and inorganic sediments at the bottom of effluents. For this reason, concentrations of PCBs in water are always very low, usually below 1 ppb (part per billion). However, the higher the number of chlorine atoms, the less the molecules are soluble, and vice versa. As data in Table 2 indicate, the position of chlorine atoms affects the solubility of PCBs.

2.3.1.3 Highly lipophilic

PCBs are highly soluble in fats, oils and nonpolar liquids. The rate of lipidic solubility usually increases with their chlorine content. This characteristic, combined with their low solubility in water, fosters bioaccumulation of PCBs in the tissues of living organisms. Obviously, an organism's ability to metabolize a given congener also affects the bioaccumulation of PCBs. Metabolism will be examined in greater detail in Chapter 10, devoted to the pharmokinetics of PCBs.

2.3.1.4 Very low volatility in air

At room temperature, volatility is low and is affected by the number of chlorine atoms attached to the molecule (see Table 2). As the number of chlorine atoms increases, the rate of evaporation decreases. It is estimated that ambient air contains between 0.0001 and 0.01 $\mu g/m^3$ of PCBs. Of course, this concentration may be higher inside buildings polluted with PCBs, all the more so where the temperature is high, as volatility increases with temperature. The rate of air replacement also affects concentrations in buildings.

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TABLE 2: Water Solubility and Vapor Pressure of PCBs

PCB molecule	Water solubility µ mole/L, 25°C	Vapor pressure mm Hg, 25°C
2-PCB	26	$8,4 \times 10^{-3}$
4-PCB	12	$4,6 \times 10^{-3}$
2,2'-PCB	4,9	$2,1 \times 10^{-4}$
4,4'-PCB	0,3	$1,9 \times 10^{-5}$
2,5,2'-PCB	3,9	9×10^{-5}
2,3,4'-PCB	0,3	
2,5,2',5'-PCB	0,09	$3,7 \times 10^{-5}$
3,4,3',4'-PCB	0,3	
2,4,2',4'-PCB	0,23	$8,6 \times 10^{-5}$
2,3,4,5,2'-PCB	0,028	

According to the National Academy of Sciences (NAS) 1979, Mackay et al. 1980, Banerjee et al. 1980, Westcott et al. 1981, Neely 1983, McDonald (unpublished data), Smith et al. 1964 (table taken from an EPA report produced in 1983).

2.3.1.5 Recognized toxicity

Various congeners present in commercial products do not have the same toxicity. We will examine this question in greater detail in Chapter 8, which focuses on the toxicity and mechanism of action of PCBs.

However, the congeners usually recognized for their higher potential toxicity are several specific congeners belonging to groups of isomers containing between four and six chlorine atoms. The congeners of groups of isomers with three or fewer chlorine atoms, or those with seven chlorine atoms or more, are deemed to be scarcely toxic, or not toxic at all, compared with the first group.

Of all of these properties, it appears that it is the chlorine content of congeners present in various commercial products which affects the latters' persistence and toxicity. It should also be noted that considerable discrepancies are found in the persistence and toxicity of these molecules, depending on the animal species.

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3. COMMERCIAL PCB-BASED PRODUCTS

3.1 Principal Producers of PCBs

Between 1929 and 1977, PCBs were manufactured and sold by various companies throughout the world under different trade names. Table 3 lists the main producers.

TABLE 3: Principal Producers of PCBs

TRADE NAME	MANUFACTURER	COUNTRY
Apirolio	Caffaro	Italy
Aroclor[1]	Monsanto	United States
Clophen	Bayer	West Germany
Kaneclor[2]	Mitsubishi	Japan
Pyralene	Prodelec	France
Pyranol	G.E.	United States
Pyroclor	Monsanto (U.K.)	United Kingdom
Aceclor	ACEC	Belgium

[1] The most widely used product in Canada and the United States.

[2] This is the product implicated in the Yusho and Yu-Cheng poisonings.

3.2 Various Industrial Uses

3.2.1 Long-term uses (closed circuit)

- dielectric fluids called askarels in power transformers and capacitors;
- hydraulic machinery, vacuum pumps, gas turbines, heat-exchanger fluids.

3.2.2 Short-term uses (open circuit)

- lubricants
- power regulators (fluoreacent light ballasts)
- rubbers and synthetic resins
- various plastics
- caulking
- paints
- glues
- waxes
- textiles
- carbonless copy paper
- inks
- dust-control agents for dirt rosds
- solvents for spreading insecticides
- cutting oils (metal work)
- immersion oils (microscopy)
- military uses (electrical or electronic equipment).

3.3 Various Commercial Compounds

Commercial compounds consist of mixtures of several congeners of polychlorinated biphenyls selected for their particular properties and occasionally combined with mixtures of chlorobenzene isomers (tri- and tetrachlorobenzenes).

Each of these congeners affects the product's properties according to its chlorine content and the position of its chlorine atoms. For example, isomers with less chlorine, e.g., dichlorophenyls, are not very fire-resistant but have good dielectric (insulating) properties. On the other hand, isomers with more chlorine, e.g., pentachlorobiphenyls, are highly fire-resistant but are not as effective insulators.

Tri- and tetrachlorobenzenes added to PCBs make them less viscous.

To illustrate this diversity, let us take as an example Aroclor, manufactured by Monsanto (U.S.) for various applications (see Table 4).

TABLE 4: Use of Various Aroclor Compounds

APPLICATION	TYPES OF AROCLOR USED
Capacitors	1016, 1221, 1242, 1254
Transformers	1242, 1254, 1260
Vacuum pumps	1248, 1254
Hydraulic fluids	1232, 1242, 1248, 1254, 1260
Plastifiers (synthetic resins)	1248, 1254, 1260, 1262, 1268
Heat exchangers	1242
Waxes	1242, 1254, 1268
Dust-control agents	1254, 1260
Pesticide diluants, inks, lubricants, cutting oils	1254
Carbonless carbon paper	1242

For Aroclors, the first two digits (12 in this instance) indicate that it is a PCB-based compound. The last two digits give the percentage of chlorine in the product, e.g., AR 1242 contains 42 percent and AR 1260 contains 60 percent.

AR 1016 is an exception: it was manufactured in 1971 by distilling AR 1242. In this product, the percentage of chlorine has fallen to 41 percent and the percentage of low-chlorine isomers has increased. The resulting product is more biodegradable and less toxic than AR 1242.

Table 5 shows that the various compounds do not all contain the same isomers or the same proportions of isomers. It should be remembered that the higher the chlorine content is, the more persistent the product is in living organisms. The congeners of AR 1260 belong to groups of isomers with high chlorine content. For this reason, AR 1260 is much more persistent than AR 1242, which contains fewer high-chlorine isomers. Moreover, when a milieu has been contaminated with several Aroclor mixtures (1242, 1254, 1260), the distribution of the congeners present tends to evolve toward a chromatographic configura-

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tion which increasingly resembles AR 1254 or AR 1260. This can be explained by the fact that low-chlorine isomers in AR 1242 biodegrade more quickly.

TABLE 5: Chemical Formulas of Some Aroclors

GROUPS OF ISOMERS (series of homologous compounds)	% OF ISOMERS IN THE AROCLORS			
	1242	1248	1254	1260
$C_{12}H_9Cl$	3			
$C_{12}H_8Cl_2$	13	2		
$C_{12}H_7Cl_3$	18	28		
$C_{12}H_6Cl_4$	30	40	11	
$C_{12}H_5Cl_5$	22	36	49	12
$C_{12}H_4Cl_6$	4	4	34	38
$C_{12}H_3Cl_7$			6	41
$C_{12}H_2Cl_8$				8
$C_{12}HCl_9$				1

3.4 Identification of the Chemical Structure of PCB Mixtures

It is thanks to chromatography in the gaseous stage and mass spectrometry that we are currently able to identify the chemical structures of mixtures we wish to analyse. In this way, we can determine the content of various PCB congeners in mixtures of commercial products or in contaminated milieux such as the ground, water, sediments, animal and human tissues, blood and so forth. Moreover, it is possible, using high-resolution apparatus, to measure the concentrations of contamination or combustion products of PCBs, such as polychlorodibenzofurans (PCDFs), polychlorodibenzodioxins (PCDDs), quaterphenyls and so on (cf. Hutzinger et al. 1974, Albro et al. 1977, Bowes 1975, Curley et al. 1975, EPA 1983).

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The high-resolution technique most frequently used to identify congeners of PCBs is the capillary column chromatography in the gaseous stage (see Figure 3, dealing with the chromatograms of Aroclors 1221, 1232, 1242, 1248 and 1254). Generally, the relative concentrations of various products found in the sample are measured, e.g., 1242, 1254 and 1260, by comparing the relative quantities of five peaks of these various products with previously known standard products. Because of overlapping of certain isomers between similar products (1242 and 1248 or 1242 and 1254), this method has its limitations. However, as little overlapping exists between 1242 and 1260, a method which makes it possible to establish this distinction is certainly valid. It should be noted that the chromatographic characteristics of PCBs are similar to those of several organochlorinated pesticides, resulting in occasional interference which may complicate identification.

Let us stress that commercial products do not contain all possible configurations of PCBs; certain congeners are absent or cannot be detected. This is important as those congeners deemed to be the most toxic (3,4,3',4' tetra- and 3,4,5,3',4',5' hexa-) are either absent or found only in trace amounts. As an example, Table 6 indicates the composition of AR 1242 and AR 1016 and reveals that they have similar compositions up to the tetra- isomers. The most polar tetra- isomers, most of the penta- and essentially all of the hexachloro- isomers are absent from AR 1016.

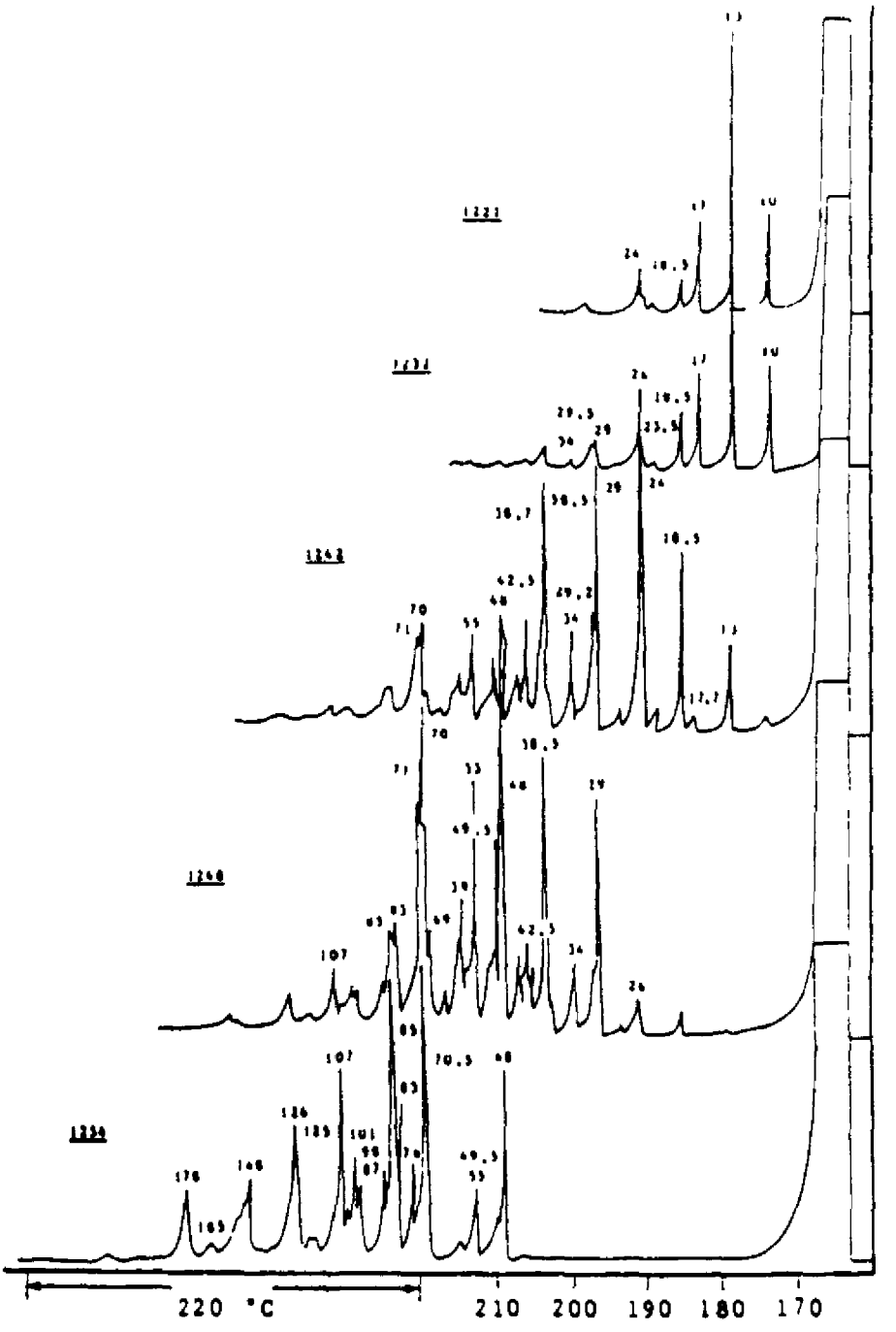


FIGURE 3: Capillary Column Chromatograms of Several Aroclors. The Number on Each Peak Corresponds to the Retention Time of Peaks in Relation to p,p' - DDT: Both Substances Were Studied Using Chromatography at 190°C (taken from J. Assoc. Offic. Anal. Chem. 55, 746, 1972, used with permission)

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TABLE 6: Chemical Composition of Aroclors 1242 and 1016
(taken from Albro and Parker, 1979)

PCB	Percentage		PCB	Percentage	
	1242	1016		1242	1016
2	0,68	0,80	2,4,2',4'	1,65	1,81
3	0,04	0,10	2,4,5,4'	2,02	1,35
4	0,22	1,00	2,5,3',4'	1,11	-
2,6	0,13	0,20	3,4,5,2'	trace	-
2,2'	3,99	4,36	3,4,5,3'	0,52	-
2,5	0,31	0,34	3,4,5,4'	0,28	-
2,4	1,04	1,16	3,4,3',4'	0,34	-
2,3'	1,24	1,37	2,5,3',5'	0,33	-
2,4'	8,97	10,30	2,4,3',4'	0,81	0,14
3,5	0,35	0,37	3,4,3',5'	0,24	-
3,4	0,09	0,11	2,3,4,4'	0,21	-
3,4'	0,12	0,12	2,4,6,3',5'	0,92	-
4,4'	0,99	1,07	2,4,6,2',3'	0,13	0,04
2,6,2'	0,97	1,08	2,3,6,2',5'	0,53	0,18
2,5,2'	9,36	10,87	2,3,6,3',5'	0,39	0,01
2,4,2'	2,92	3,14	2,3,6,2',4'	trace	-
2,3,2'	3,25	3,50	2,3,6,2',3'	0,38	0,01
2,6,3'	0,54	0,58	2,3,4,3',5'	0,46	0,16
2,6,4'	2,15	2,31	2,3,4,2',4'	0,40	-
2,5,3'	0,55	0,62	2,3,4,3',4'	0,09	-
2,5,4'	4,53	4,72	2,3,5,2',5'	0,25	-
2,4,3'	1,68	1,79	2,4,5,2',5'	0,12	-
2,4,4'	13,30	14,48	2,4,5,2',4'	0,55	-
2,3,3'	3,64	3,99	2,4,5,2',5'	0,27	-
2,3,4'	2,64	2,80	2,4,5,3',5'	0,31	-
3,4,2'	2,83	3,08	3,4,5,2',4'	0,36	-
3,4,3'	0,66	0,38	3,4,5,3',5'	0,05	-
3,4,4'	1,62	1,89	3,4,5,3',4'	0,03	-
3,5,4'	1,03	1,08	2,3,4,5,2',6'	0,07	-
2,6,2',6'	0,17	0,19	2,3,4,2',4',5'	0,08	-
2,3,6,2'	0,90	1,00	2,4,5,2',4',5'	0,02	-
2,5,2',6'	0,97	1,07			
2,5,2',5'	4,08	4,35	Monochloro-(%)	0,94	2,40
2,4,6,4'	2,18	2,40	Dichloro-(%)	17,23	19,40
2,3,5,2'	0,44	0,47	Trichloro-(%)	51,67	56,31
2,3,2',6'	0,31	0,33	Tetrachloro-(%)	24,65	21,32
2,4,5,2'	1,33	1,41	Pentachloro-(%)	5,24	0,40
2,4,2',5'	3,28	3,48	Hexachloro-(%)	0,17	-
2,3,2',5'	1,06	1,14			
2,3,4,2'	1,67	2,00			
2,3,2',3'	0,15	0,18			
2,3,3',4'	0,60	-			

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4. POLYCHLORODIBENZODIOXINS (PCDDs) AND POLYCHLORODIBENZOFURANS (PCDFs)

Like polychlorinsted biphenyls, both families belong to the broad family of halogenated aromatic hydrocarbons. Their members (congeners) have a structure quite similar to those of PCBs.

PCDDs and PCDFs formed during combustion of polychlorinated biphenyls result from the incomplete combustion of the latter. However, the quantity of PCDF formed is considerably more substantial than that of PCDD. PCDFs are also contaminants found in PCB-based commercial products, e.g., askarels.

A number of congeners belonging to these two families are deemed to be extremely toxic, much more so than the congeners of PCBs.

As these compounds can appear with the introduction of PCBs in the environment, it seems necessary to us to study the question of PCDFs and PCDDs to interpret the effects of PCBs on the environment and humans.

Without examining in detail their physiochemical properties, dispersal, source and levels in the environment, which would demand more extensive work than for PCBs, we will analyse certain aspects of their toxicity in Chapter 8. In this and subsequent chapters, we will make a general presentation devoted to contaminants and fires.

4.1 Polychlorodibenzodioxins (PCDDs) or Dioxins

The number of chlorine atoms can vary from 1 to 8. There are 75 congeners, including 22 tetrachlorodibenzodioxins, of which 2,3,7,8-TCDD is the best known and most toxic. This molecule is one of the best known pollutants; it is the topic of considerable research and many published articles.

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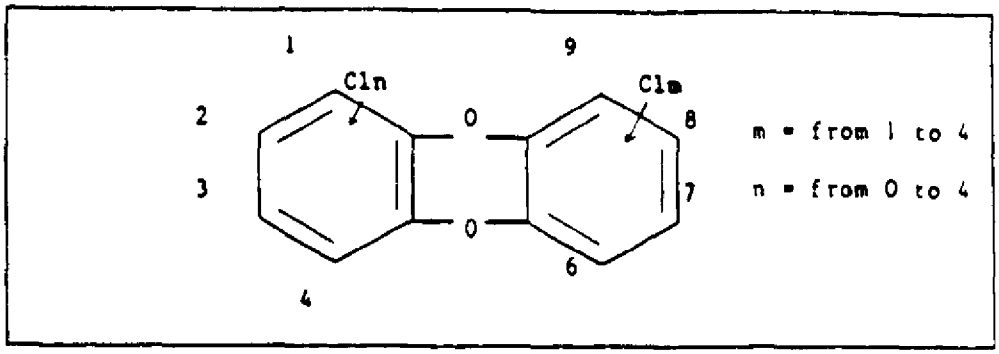
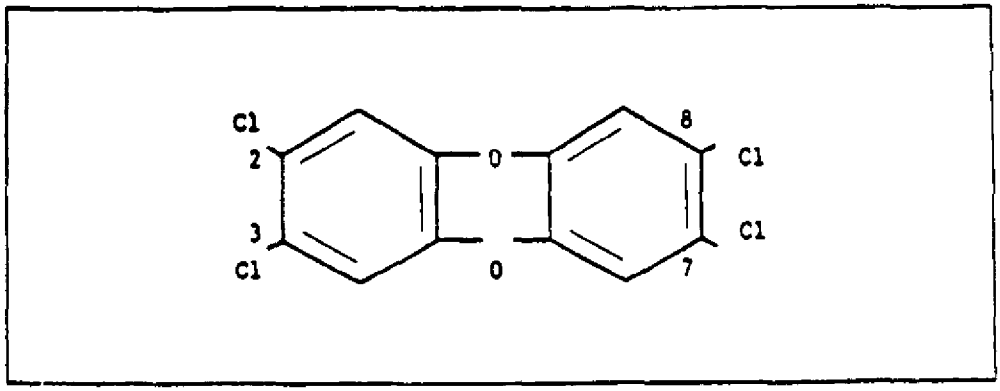


FIGURE 4: General Formula of PCDDs

The structure of PCDDs is quite similar to that of PCBs. PCDD molecules can be formed through the combustion of tetrachlorobenzene and, in limited amounts, through the pyrolysis of PCBs. They are also a by-product of the manufacture of trichlorophenol, a basic component in insecticides such as DDT.

FIGURE 5: Structure of 2,3,7,8-TCDD, the Most Toxic Molecule



4.2 Polychlorinated Dibenzofurans (PCDFs) or Furans

Furans have 135 congeners, including 38 tetrachlorodibenzofurans (TCDFs). The number of chlorine atoms can vary from 1 to 8. Their structure is also similar to that of PCBs and they can be formed by the hydrolysis or combustion of PCBs. Moreover, they are contaminants of commercial PCB-based products.

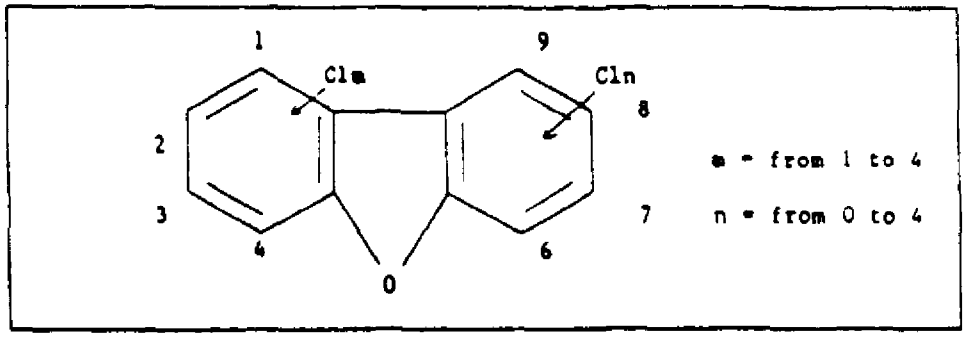


FIGURE 6: General Formula of PCDFs

The most toxic molecule is 2,3,7,8-TCDF; it has the same structure as 2,3,7,8-TCDD, except that it has one less oxygen molecule.

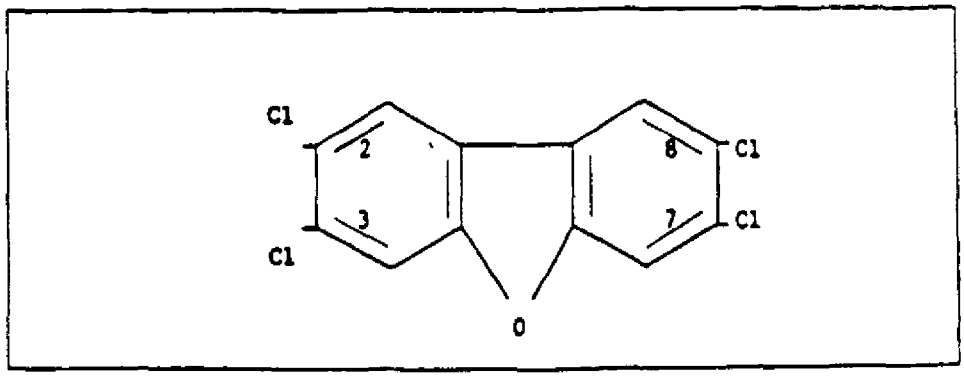


FIGURE 7: Structure of 2,3,7,8-TCDF, the Most Toxic Molecule

5. PCB CONTAMINANTS

Commercial PCB-based products are usually contaminated with small amounts of chlorinated naphthalene and traces of polychlorodibenzofurans (PCDFs).

Furans are generally considered to be highly toxic. Today, 17 years after the poisoning in Japan and six years after the incident in Taiwan, experts have concluded that symptoms displayed by the victims of both massive poisonings are caused primarily by polychlorodibenzofurans in the rice oil (Kashimoto et al., 1981 and 1985).

5.1 Presence of Polychlorodibenzofurans in Commercial Products Prior to Use

Table 7 indicates the PCDF content detected in various commercial products manufactured in the United States, Japan, Germany and France. American products contain less PCDFs, overall average concentration of which in Aroclors is 2 ppm (1242, 1248 and 1254). In products from Japan, Germany and France, this concentration varies between 8 ppm and 18 ppm .

It should be noted that Bowes (1975) and Miyata (1976, 1985) have identified 2,3,7,8-TCDF and 2,3,4,7,8-PCDF compounds in AR 1242, 1248 and 1260 and in Kaneclors 300, 400, 500 and 600. These two congeners are the most toxic in the PCDF family.

5.2 Concentration of PCDF in PCBs According to Length of Use

It has been shown (Chittim et al., 1979; Randoux, 1983) that the concentration of PCDFs in askarels increases with the length of use of transformers. Table 8 describes this phenomenon in relation to the level of T_4CDF (isomers in the tetrachlorinated homologous compound series). All measurements of TCDF were effected after 1975.

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TABLE 7: Concentration of Total PCDFs in PCB-Based Commercial Products

COUNTRY	PRODUCT AND YEAR OF PRODUCTION	TOTAL PCDF CONCENTRATION ppm	REFERENCE
United States	(Aroclors)		
	AR 1242	0,2	Albro (1977)
	(1969)	3	Miyata et Kashimoto (1978)
	AR 1248 (1969)	2	Bowes et al. (1975)
		2	Miyata et Kashimoto (1976)
	AR 1254 (1969)	1,2	Bowes et al. (1975)
	(1970)	1,9	Miyata et Kashimoto (1978)
		undetectable	
	AR 1016	< 0,005	Albro (1977)
	(1972)	< 0,001	Bowes et al. (1975)
Japan	(Kaneclors)	9	Miyata et al. (1975)
	KC 300 (before 72)	18	Miyata et al. (1975)
	KC 400 (before 68)	14	Miyata et al. (1975)
	KC 500 (before 72)	16	Miyata et al. (1975)
	KC 600 (before 72)		
Germany	Clophen A60	8,6	Bowes et al. (1975)
	Clophen A50	17	Miyata et Kashimoto (1976)
France	Phenoclor DP-4	8,0	Miyata et Kashimoto (1976)
	Phenoclor DP-5	11	Miyata et Kashimoto (1976)
	Phenoclor DP-6	11,6	Bowes et al. (1975)

TABLE B: Variation According to the Length of Use and the Concentration of PCDFs in Transformers* Insulated with Askarel (cf. Chittim et al., 1979)

YEAR OF INSTALLATION	AROCLOR	Chlorobenzenes/PCBs (%)	T_4CDF (mg/kg of askarel)
1957	1260	51/49	0,780
1961	1260	53,6/46,4	1,110
1963	1260	50,6/49,4	1,020
1963	1260	52,4/47,6	0,790
1963	1260	38,6/61,4	0,350
1965	1260	37,2/62,8	0,290
1970	1254	29,6/70,4	0,550
1970	1254	28,0/72,0	0,470
1971	1254	29,0/71,0	0,290
1974	1254	28,4/71,6	0,051
not uaed	1254	30,6/69,4	0,041

*1500-kW transformers.

5.3 Concentration of PCDFs in PCBs and Temperature (Below or Equal to Ignition Point)

Another factor which markedly affects the production of PCDFs is the increase in temperatura when oxygen is present. The presence of catalyzers such as steel may accelerata the formation process. Miyata and Kashimoto demonstrated this in 1978 by heating KC 400 to 360°C for 20 daya. Table 9 indicates that the original PCDFs increased by a factor of between 2.3 and 6.4, depending on the temperature and catalyzers present.

TABLE 9: Effect of Heat on the Formation of PCDFs in KC 400 (Miyata and Kashimoto, 1978)

TEMPERATURE	DURATION IN DAYS	CATALYZER	TOTAL PCDFs mg/kg of PCBs	TOTAL TCDFs mg/kg of PCBs
Ambient	0	none	33	26.2
360°C	20	none	65	52.1
360°C	20	stainless steel	77	32.7
360°C	20	water	212.1	152.4
360°C	20	steel + water	72.1	46.6

As Table 10 indicates, Miyata (1985) noted a marked increase of PCDFs when comparing unused KC 400 with KC 400 used as a conductor in heat exchangers. One sample came from an exchanger used for 14 years at 180°C-270°C; the second came from an exchanger used for three years at 200°C-220°C. He also demonstrated the presence of polychlorinated quaterphenyls (PCQs).

TABLE 10: Concentration of PCBs, PCQs and PCDFs in Two Samples of KC 400 Used in Heat Exchangers (cf. Miyata, 1985)

KC 400 Sample	Temperature	PCBs (ppm)	PCQs (ppm)	PCDFs (ppm)
S.1 (14 years)	180 to 270°C	968 400	31 000	510
S.2 (3 years)	200 to 220°C	999 000	690	277
S.3 (not used)		999 800	209	33

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Contamination of the rice oil which caused Yusho disease in Japan and Yu-Cheng disease in Taiwan was caused by a heat exchanger. While the ratio of PCDFs to PCBs in the unused KC 400 was about 1/50 000 on the average, it was only about 1/200 and 1/400 in the contaminated oils in question. The PCQ/PCB ratio was 1/1 in Japan and 1/3 in Taiwan. The high PCDF and PCQ content can be explained by the high temperature prevailing in the heat exchanger. In Chapter II, we will discuss the role PCDFs and PCQs played among victims of these diseases.

6. PCBs AND FIRES

At present, the main problem with PCBs is that of fires in electrical equipment insulated with pure PCBs or with PCBs combined with other solvents such as tri- and tetrachlorobenzenes, which are often present in askarels. A related, but less serious problem is arousing more and more concern among the general public and workers, especially firefighters, i.e. fires in electrical equipment insulated with mineral oil contaminated by PCBs in concentrations of between several ppm and more than 500 ppm. It is estimated that about 10 percent of oil-insulated transformers currently operating are contaminated with more than 50 ppm of PCBs.

The seriousness of fires where PCBs are present is due to the formation, under intense heat, of other extremely toxic organochlorinated products, i.e. PCDFs and PCDDs. Polychlorinated quaterphenyls and methylsulfones, deemed to be less toxic than PCBs, and polychlrinated biphenyls are also formed; documentation on the latter family is limited.

When fire breaks out in equipment containing PCBs, such as a transformer or a capacitor, attention focuses on safety measures to be taken during the fire to protect firefighters combating the flames and, after the fire, to protect workers responsible for decontaminating and rebuilding the site.

To estimate risk to such workers and residents living near the site, it is important to know under what conditions and in what proportions these toxic products are formed, and the quantities of contaminants found in soot.

6.1 Formation of PCDFs, PCDDs and Hydrochloric Acid

Several factors determine the types of PCDF and PCDD congeners formed: the products used (chlorine content, presence or absence of tetrachlorobenzenes), temperature, the presence of oxygen, the presence of catalyzers in other materials burned, and so on.

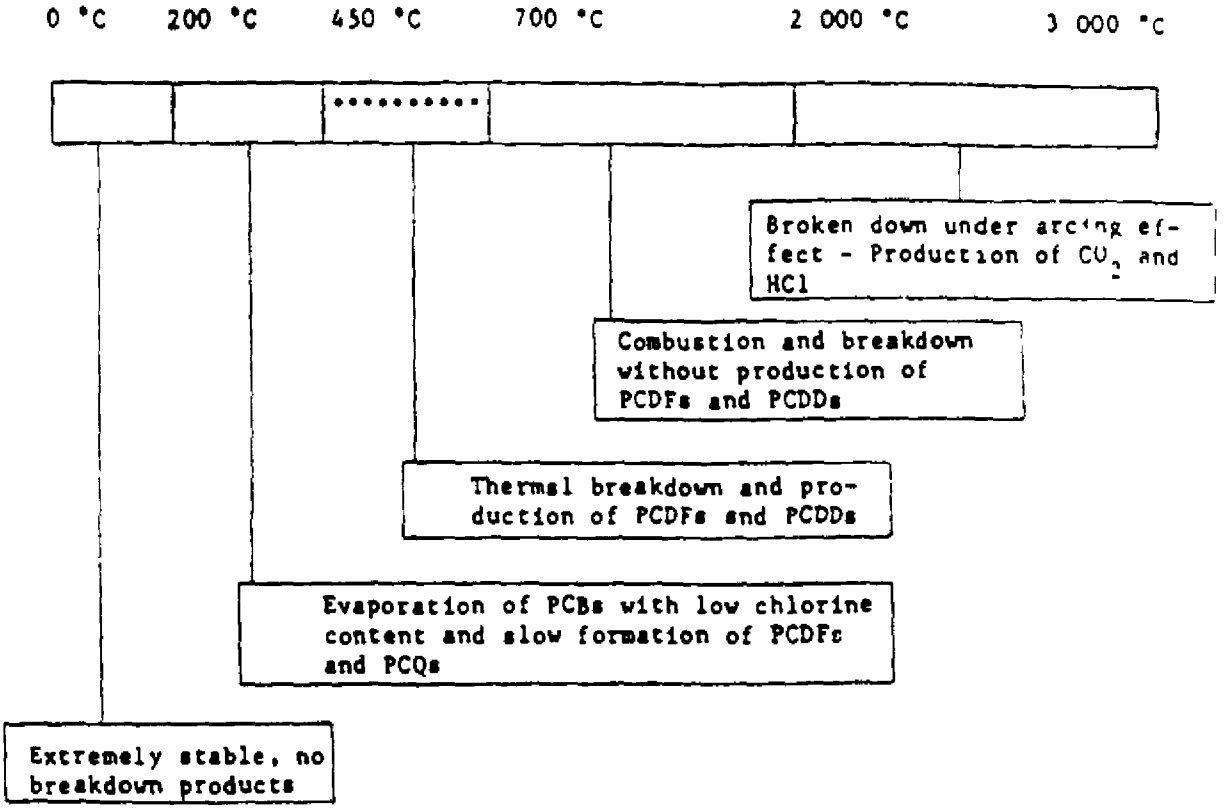


FIGURE 8: Schematic Representation of the Thermal Breakdown of PCBs and the Production of PCDFs, PCDDs and HCl

Let us analyse in greater detail the thermal breakdown of PCBs (Figure 8).

0°C to 200°C PCBs are extremely stable; no breakdown occurs.

200°C to 450°C These temperatures are below the ignition point of PCBs, although evaporation does occur and, as we noted in section 5.3, PCDFs and PCQs form slowly.

450°C to 700°C Several authors have conducted laboratory studies of incomplete combustion at these temperatures (Buser et al., Marita et al., Chittim et al.). They showed that fairly substantial quantities of PCDFs formed in a few seconds, i.e. between 10 mg/kg and 20 g/kg of PCBs depending on the temperature and the factors present. For example, Buser et al. (1978) found that by pyrolyzing hexachlorobiphenyls (H_6CB) at 550°C, 20 g of PCDFs/kg of PCBs formed, while at 700°C, only 16 mg formed. The PCDFs obtained were primarily the congeners 2,3,7,8-T_4CDF, 1,3,4,7,8-P_5CDF and 1,2,3,7,8-P_5 CDF.

When AR 1254 and AR 1260 were heated in the laboratory to about 550°C (the temperature at which optimum formation of PCDFs occurs) in a glass bubble in the presence of oxygen, about 20 g of PCDFs/kg of PCBs formed, belonging to series of homologous compounds of mono- to penta-CDF for AR 1254 and of tri- to octa-CDF for AR 1260.

700°C to 2000°C PCDFs were produced in negligible quantities, which explains why combustion ovens employed in the United States and Europe to destroy PCBs usually operate between 1000°C and 1400°C. Rappe et al. (1979) showed that no discernable PCDFs and PCDDs were formed by heating PCBs in a rotary cement oven to temperatures between 1400°C and 2000°C, which seems to indicate that this method of destroying the substances is fairly safe.

2000°C or more Under the effect of electric arcs, CO_2 and HCl, with its characteristic odor of bleach, are produced. We noted this phenomenon at the Hydro-Québec Wellington substation fire in the Maisonneuve region. Negligible quantities of PCDFs were measured in soot near the seat of combustion; the concentration was below the standard in force in Québec, i.e. below 1/500 of the Non Observed Effect Level (NOEL) in 2,3,7,8-TCDD equivalent.

6.2 Estimate of the Quantity of PCDFs Formed During a Fire

To estimate health risks in the case of fires involving PCBs, it is very important to ascertain the quantity of PCDFs formed. In this section, by extrapolating data at our disposal, we will attempt to estimate the maximum quantities of PCDFs formed.

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We know that the quantity of PCDFs formed during the combustion of PCBs depends upon temperature and the presence of oxygen. However, during a fire, all of the conditions for the optimum formation of PCBs simulated in the laboratory are not necessarily present. Temperature varies in time and space, with discrepancies of several hundred degrees Celsius between the periphery and the seat of the fire. All PCB molecules are not, therefore, subjected to the same combustion temperature. Moreover, it appears that certain PCB molecules do not obtain the oxygen needed to burn and form a PCDF molecule. These molecules spread across the ground without being pyrolyzed, as the concentrations of PCBs noted in soot testify.

Thus, it may be inferred, with little risk of error, that during a fire, the quantities of PCDFs formed may be below 20 g of PCDF/kg of PCB, but probably higher than 16 mg, i.e. at a level somewhere between the minimum and maximum values found in the laboratory.

In the following pages, we will endeavor, using data drawn from IREQ's experience, to establish in what proportion PCDFs were formed on the basis of the combustion of a known quantity of PCBs.

6.2.1 Initial hypothesis

Let us verify the following hypothesis: that under normal conditions prevailing during a fire, the quantity of PCDFs formed per kilo of PCBs does not exceed the proportional value at mid-point between 16 mg and 20 g, i.e. 565 mg of PCDFs/kg of PCBs.

$$\frac{20\,000\ \text{mg}}{565\ \text{mg}} = \frac{565\ \text{mg}}{16\ \text{mg}} = 35.35$$

To simplify calculations, let us take the value 500 mg of PCDFs/kg of PCBs.

6.2.2 Brief description of a fire at IREQ

On November 20, 1984, an explosion occurred in a capacitor bank during tests on a power transformer in the annex of the high-voltage laboratory (see Figure 9). The explosion started a fire which led to a series of explosions in other capacitors, some of which were insulated with oil, and others with PCBs. All told, 2560 L of mineral oil and 670 L of PCBs (AR 1242) were destroyed. For two hours, during which it is estimated that virtually all of the oil and PCBs were destroyed, thick, black smoke lingered inside the building. It spread to the annex, the main hall and spaces between walls and ceilings. A hole then opened up in the ceiling above the seat of the fire and the smoke escaped. However, samples taken after the fire revealed negligible contamination of the ground surrounding the building. It should be mentioned that almost all of the materials were incombustible, except, of course, the mineral oil and PCBs.

6.2.3 Verification of the initial hypothesis: that the maximum formation of PCDFs is below 500 mg/kg of PCBs

Overall, 3230 L of combustible material was destroyed, i.e. 670 L of PCBs and 2560 L of mineral oil. Taking into account densities (0.9 for oil and 1.5 for AR 1242), this meant a total of 3309 kg of fluids.

Analysis of a typical sample of soot collected near the seat of the fire indicates that it contained 39 ppm of PCDFs (mg of PCDF/kg of soot) and 2200 ppm of PCBs (mg of PCB/kg of soot). The walls of the annex were covered with a layer of oily soot between several micrometres and several millimetres thick; the walls of the main hall were covered in places with a thin film of soot at least several micrometres thick.

According to our hypothesis, the overall quantity of polychlorodibenzofurans (PCDFs) formed should be below 502 g.

(670 L of PCBs) x (1.5 kg of PCBs/L) x (500 mg PCDFs/kg of PCBs) = 502 g

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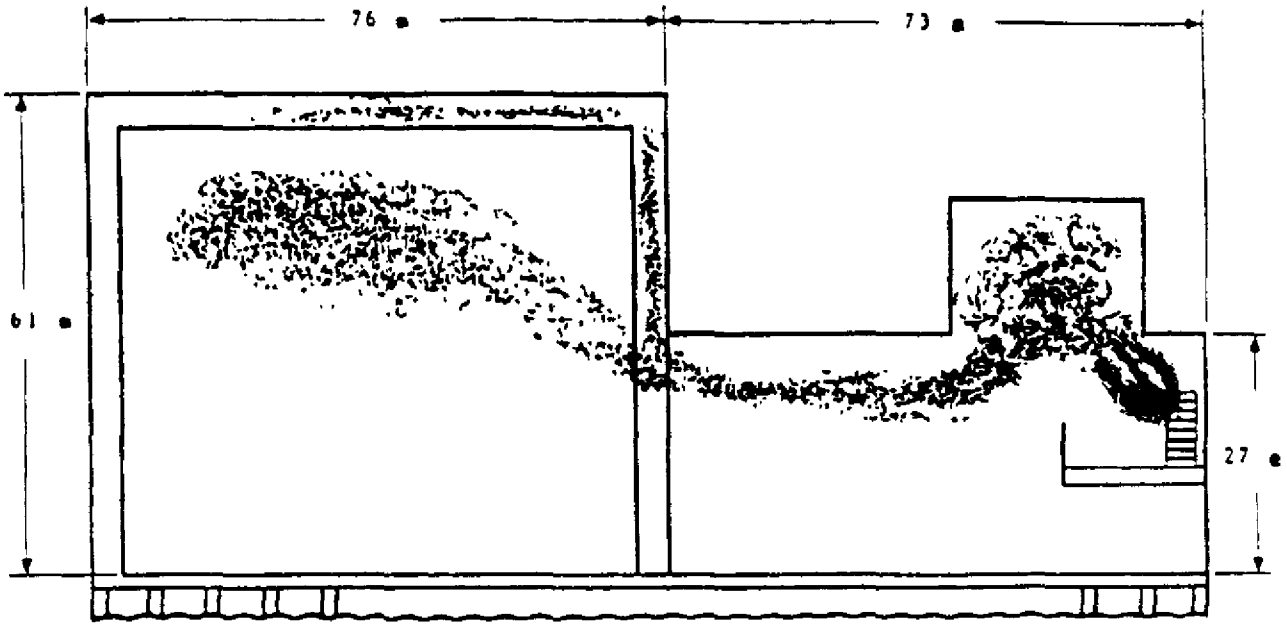


FIGURE 9: Location of the Fire in the IREQ High-voltage Laboratory (Figure Taken from a Speech by David Train and Danielle Dupont Given in September 1985 in Seattle, Washington)

6.2.4 Is this hypothesis realistic?

Where the average concentration of PCDFs in soot is equivalent to our typical sample, i.e. 39 ppm, it would take 12 871 kg of soot to obtain 502 g of PCDFs. In this case, there were only 3309 kg of combustible materials (oils and PCBs); as there could never be four times more soot than combustible materials, it may be inferred that the formation of a maximum of about 125 mg/kg of PCDFs would be more realistic (500 ÷ 4 = 125 mg/kg).

Let us verify the hypothesis by another method. The walls, ceilings and floors of the annex and the hall are considered to be smooth and to have a total surface area of about 45 000 m^2, i.e. 30 000 m^2 in the main hall and 15 000 m^2 in the annex. According to IREQ chemist Jacques Castonguay, who supervised decontamination procedures, it would be realistic to estimate that soot in the annex was an average of 1 millimetre thick on surfaces. Taking into account the low density of soot (0.01 to 0.015), he estimates that it weighs approximately 10 g per m^2 of surface. In the worst case, where the content of soot per surface unit of the main hall was equal to that of the annex, we would arrive at a total of 450 kg of soot (45 000 m^2 x 10 g/m^2). This corresponds to a total of 17.55 g of PCDFs (450 kg of soot x 39 mg of PCDFs/kg of soot), a quantity 28.6 times lower than that in our initial hypothesis. We must, therefore, infer that the quantity of PCDFs per kilogram of PCBs is 500 mg/kg (initial hypothesis) ÷ 28.6, i.e. 17.5 mg of PCDFs/kg of PCBs.

On this basis, it can readily be inferred that the quantities of PCDFs formed at IREQ were below 500 mg/kg of PCBs. According to our reasoning, they appear to be between 17.5 mg/kg and 125 mg/kg of PCBs.

6.3 Principal Fires in Electrical Equipment Insulated with Askarels

In North America, the most serious fires involving PCBs occurred in February, 1981, in an 18-storey government building in Binghamton; in May 1983, in a commercial building in San Francisco; and in November 1984, at IREQ.

In all three instances, soot covering the surface of interior walls contained PCBs, PCDFs, PCDDs and polychlorobiphenylenes.

Table 11 indicates the typical quantities of PCBs, total PCDFs, total TCDFs and 2,3,7,8-TCDD from soot samples collected near the seat of fires, close to burned electrical equipment, i.e. transformers in Binghamton and San Francisco and capacitors at IREQ.

TABLE 11: Typical Concentration of Total PCBs, PCDFs and TCDFs, and TCDD in Soot Samples Collected Near the Seat of Fires

FIRE	PCBs	Concentration (ppm) in soot [mg/kg] Total PCDFs	Total TCDFs	2,3,7,8-TCDF	2,3,7,8-TCDD
San Francisco, CA	86 000**	286	16.29	6.7	undetected
Binghamton, NY	7 200*	438	145	72	3
IREQ	2 500**	39	4.89	0.61	undetected***

* AR 1254 and 35% tri- and tetrachlorobenzenes
** 100% AR 1242
*** Detection limit between 10 u g/kg and 50 u g/kg

In all three cases, the quantity of PCDFs is relatively high; on the other hand, in California and at IREQ, total quantities of PCDDs are negligible and the concentration of 2,3,7,8-TCDD undetectable. The greater quantities of PCDDs and 2,3,7,8-TCDD in Binghamton can be explained by the presence of tetrachlorbenzene (35 percent) in the askarel. It has been shown that tetrachlorobenzene breaks down into dioxine through incomplete combustion; PCBs tend to produce furans. The mechanism by which the combustion of PCBs produces small quantities of dioxins is not known.

It should be noted that at IREQ and in San Francisco, 2,3,7,8-TCDFs accounted for 1.5 percent and 2.3 percent, respectively, of total PCDFs, while in Binghamton they represented approximately 16 percent. This can probably be explained by the fact that AR 1254 produces more tetra-CDF isomers than AR 1242. At IREQ, more than 62 percent of PCDFs were tri-CDF isomers. From

this it must be concluded that health risks are higher with AR 1254 than with AR 1242, and that the presence of tetrachlorobenzene in askarel increases this risk. Obviously, this depends on the amounts burned.

6.4 Health Risks During an Askarel Fire and Decontamination

Without claiming to accurately estimate the risks of acute and chronic toxicity among people exposed, it appears possible to obtain approximate data by effecting comperisons with incidents where PCDFs were involved in human poisonings. As there are two thoroughly documented cases of PCDF-related poisonings (in Japan in 1968 and in Taiwan in 1979), it appears eminently worthwhile to ascertain whether risk is as considerable in the case of fires with PCBs.

As explained in section 11.2, it is estimated that victims of Yusho and Yu-Cheng diseases ingested an average of between 0.5 g and 2 g of PCBs (Kaneclor 400) and from 2.4 mg to 7 mg of PCDFs.

Moreover, we now know that PCDFs were the principal cause of these poisonings, which provides us with a useful reference point. In the following section, we will endeavor to estimate whether it is probable that a fire in electrical equipment insulated with PCBs engenders as great a risk as in the Japanese and Taiwanese incidents.

6.4.1 Risk during the fire

To estimate this risk, we will refer to the IREQ fire as we are thoroughly familiar with the circumstances surrounding it. It took firefighters approximately six hours to control the flames, which reached their peak intensity during the first two hours. It is thought that almost all of the PCBs were burned during this time. Immediately after the fire, a sample taken in the burned building revealed that the air contained less than 10 u g/m^3 of PCBs (the standard in force in Québec for AR 1242 is 1 mg/m^3 for daily exposure of eight hours). However, we do not have data on the PCDF content of the air at that time.

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6.4.1.1 Exposure to PCBs

A human being inhales about 2 m^3 of air every hour. For a firefighter to inhale between 0.5 g and 2 g of PCBs, the air would have to contain between 125 mg/m^3 and 500 mg/m^3 of the substances. Given the low vapor pressure of PCBs, even at temperatures between 35°C and 45°C, it is virtually impossible to attain such concentrations.

It should be noted that, after one week, no professional or volunteer Hydro-Québec firefighter had blood PCB levels higher than concentrations found in the population at large (≤5 ppm of 1242 on average). This is an excellent indication of the low risk to which they were exposed. It should be pointed out that most of the firefighters were wearing a self-contained, positive-pressure breathing apparatus.

6.4.1.2 Exposure to polychlorodibenzofurans

In section 6.2.4 we estimated that a maximum of 125 g of PCDFs could have been formed by the combustion of PCBs (this is an overestimation). Let us take the worst hypothesis, where the 125 g of PCDFs formed gradually over two hours were evenly distributed throughout the high-voltage laboratory (a volume of about 496 219 m^3: 76 x 76 x 61 + 73 x 73 x 27 m) and remained suspended in the air: in this case, the average concentration per m^3 would be equal to the concentration existing after one hour, i.e. 0.125 mg/m^3 [½ x (125 g ÷ 496 219 m^3 of air)]. After two hours, a firefighter would have absorbed a maximum of 0.25 mg of PCDFs, which is less than the quantity ingested during the poisoning in Japan (between 2.4 mg and 7 mg). Obviously, this hypothetical case is unrealistic as the PCDFs, during their formation, were trapped in the dense, oily smoke which deposited them on the ground and walls. The PCDF content of oily soot bears witness to this fact; thus, their concentration was much lower.

Moreover, wearing a self-contained, positive-pressure breathing apparatus reduced this risk by a factor of roughly 3 000 or more. At IREQ, even in the worst hypothesis, a firefighter wearing a mask could have inhaled a maximum of 83.3 n g of total PCDFs, of which only 3 percent were tetrachloroisomers according to the analyses made at IREQ (the other isomers are less important from the standpoint of toxicity).

These observations enable us to conclude that, during the burning of several hundreds of litres of PCBs, the risk of firefighters displaying symptoms of Yusho disease is negligible where preventive measures are taken.

As a precaution, we recommend:

- that anyone intervening in a fire in equipment insulated with askarel be sure to wear a self-contained, positive-pressure breathing apparatus and a coverall to protect the skin;

- that emergency intervention procedures be established for all areas where this type of equipment is located, and that unauthorized persons be denied access to the site during and after the fire.

6.4.2 Health risks during decontamination

This risk will be assessed in a general manner, using comparisons with the quantities of PCDFs which caused Yusho disease. It should be remembered that the total quantity of PCDFs in samples collected near the seats of fires in San Francisco, Binghamton and at IREQ were 289 mg/g, 438 mg/g and 39 mg/g of soot, respectively. In other words, to absorb a quantity of PCDFs equivalent to that in Japan, i.e. between 2.4 mg and 7 mg, workers involved in decontamination procedures would have only had to absorb several milligrams of soot (see Table 12) by inhalation, contact or ingestion.

Although PCDFs are not very volatile and soot is very oily and sticks to surfaces, it is to be noted that risks they present to health are very high. For this reason, it is important to take extraordinary precautions to prevent any contact with soot and any contamination of workers' clothing.

Risk was highest at Binghamton, because of two factors: the presence of tetrachlorobenzene, and the high chlorine content of AR 1254 as opposed to AR 1242 which was present in the other two cases. Risk in Binghamton was even

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TABLE 12: Quantities of Soot to be Absorbed to Attain the Amount of Total PCDFs Which Caused Yusho Disease

FIRE	QUANTITY OF SOOT TO BE ABSORBED (mg)
San Francisco	8.4 to 24
Binghamton	5.5 to 11
IREQ	60 to 175

higher than it appeared to be, as the relative quantities of tetra-CDF and 2,3,7,8-TCDF (the most toxic congeners of the PCDF family) were higher than in the other two cases.

6.5 Fire in Equipment Containing Mineral Oil Contaminated with PCBs

During the past decade it has been discovered that transformers insulated with oil built since the early 1950s are often contaminated with PCBs. On the basis of inventories, it is estimated that between 10 percent and 15 percent of such transformers contain concentrations of more than 50 ppm of PCBs, and less than 1 percent contain concentrations of more than 500 ppm.

For this reason, the public at large and especially workers in the electrical industry and firefighters, fear the consequences of fires involving this type of equipment. Does the presence of PCDFs during combustion of contaminated oils create a risk, and can such risk be estimated?

6.5.1 Quantities of PCBs present in contaminated oils

Oil contaminated at 50 ppm contains 50 mg of PCBs per kilogram: 1 mg of PCBs per kilogram of oil corresponds to each part per million (ppm) of PCB contamination.

A transformer containing 300 kg of mineral oil contaminated with 100 ppm of PCBs by weight contains a total of 30 g of PCBs (100 mg of PCBs/kg of oil x 300 kg of oil), i.e. a factor of 10^4 times less PCBs than in a transformer insulated with PCBs alone.

6.5.2 Health risks posed by PCDFs formed in burning contaminated oil

Let us turn again to the example of a transformer containing 300 kg of oil contaminated at 100 ppm, i.e. a total of 30 g of PCBs. If we assume that during the fire the PCBs are destroyed under conditions similar to those of the IREQ fire, we are entitled to think that our estimate of the formation of a maximum of 500 mg of PCDFs/kg of PCBs is still valid. This would lead to a maximum total of 15 mg of PCDFs resulting from the combustion of the PCBs present (100 molecules of PCBs dispersed through 999 900 molecules of oil).

Let us imagine that the 300 kg of contaminated oil burn up in about 30 minutes, a fairly realistic estimate. This would result in the emission of an average total of 8.3 µ g/s of PCDFs over the entire surface of the burning oil at a given moment. If we take into account the chimney effect of the fire, which would tend to draw particles upward, the effect of wind dispersal and the effect of dilution over distance, it is easy to imagine that the concentration of PCDFs per unit of volume would be very small. Moreover, account must be taken of the fact that PCDFs are not very volatile (they tend to fall back to the ground).

If we combine the average human rate of inhalation (2 m^3/h) with the duration of the fire (≤ 1 hour), it appears rather unlikely that a person will absorb significant quantities of PCDFs. Through preventive measures (having firefighters use self-contained breathing apparatus and keeping bystanders away from the fire), health risks related to PCDFs become virtually nil in such a case.

A recent laboratory study (Alan et al., 1985, Southern California, Department of the Environment, International Symposium on PCBs, Seattle) showed that by burning 25 gallons (112.5 L) of oil contaminated at 9.9 ppm according to a mathematical model of dispersion of PCDFs emitted, the probability of a person in the neighbourhood of the fire absorbing a dose equivalent to that set by the EPA for lifetime exposure (risk equivalent to 1/1 000 of NOEL) was below 1 in 7 000 000.

It seems clear that these data, even based on estimates, indicate that health risks in the event of fires in contaminated oil are not nearly as pronounced as in fires involving askarels. Research must, however, be pursued to clarify

this risk. In the meantime, we advise caution: firefighters should wear appropriate protective equipment during the fire. Afterwards, even when risk of contamination by PCDFs is low, great care should be taken and the advice of experts sought before cleaning and rebuilding operations are launched.

6.6 Decontamination Criteria

IREQ's decontamination criteria comprise long-term levels of exposure to PCDDs and PCDFs currently in force in Québec. These criteria were established with the participation and consent of the Commission de la santé et de la sécurité du travail du Quebec (CSST).

TABLE 13: Toxicological Criteria on Residual Levels of PCDDs and PCDFs in the Air and on Surfaces

PCDDs AND PCDFs	CONCENTRATION IN THE AIR	SURFACE CONCENTRATION
2,3,7,8-TCDD equivalent	10 pg/m^3	25 ng/m2

These criteria correspond to those established by Kim and Hawley (1984), experts attached to the New York state health department, in agreement with an advisory board set up after the Binghamton, New York fire. These experts consider safe for humans a daily exposure dose below 1/500 of the daily dose for which no toxic effects have been detected among laboratory animals, which in turn corresponds to 1/500 of the Non Observed Effect Level (NOEL).

Without outlining in detail the approach employed to establish the tolerable concentrations of PCDDs and PCDFs in the air and on surfaces to ensure that the safe exposure dose is not exceeded, it might be useful to offer a general idea of the origin of these criteria.

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First, tolerable concentrations of PCDDs and PCDFs are reduced to 2,3,7,8-TCDD equivalents, with a view to adjusting the concentration of various congeners of PCDDs and PCDFs measured in relation to the concentration of 2,3,7,8-TCDD required to produce the same toxic effect in animals. A weighting factor has been established for all isomers of PCDDs and PCDFs relative to 2,3,7,8-TCDD. This concept is based on the approach suggested by Hutzinger et al. (1981, 1982).

Second, the minimum daily dose of 2,3,7,8-TCDD for which no effect is observed in laboratory animals (NOEL) was established by R.J. Kociba et al. in 1979, who stated, in agreement with the National Cancer Institute (NCI), that no toxic or carcinogenic effects or effects on reproduction were detected in rats and mice for doses below 1 ng/kg/day of 2,3,7,8-TCDD. A safety factor of 500 was proposed by the Binghamton experts and was included in Kim and Hawley's calculation.

Consequently, a lifetime daily exposure dose of 2 pg/kg/day of 2,3,7,8-TCDD equivalent was adopted for humans, corresponding to 100 pg/day for a person of average weight (50 kg). Assuming that a person breathes an average of 10 m^3 of air in the course of a working day, Kim and Hawley set the tolerable concentration in air at 10 pg/m^3 of 2,3,7,8-TCDD equivalent (100 pg/d/person ÷ 10 m^3 of air = 10 pg/m^3). They established the tolerable surface contamination criterion at 25 ng of 2,3,7,8-TCDD equivalent/m^2, using a mathemetical model of absorption by the skin, bearing in mind the body surfaces exposed, i.e. arms and hands.

It should be noted that all of these criteria were complied with at IREQ once buildings had been decontaminated.

7. PCBs AND THE ENVIRONMENT

7.1 Quantities of PCBs Produced in North America

It is estimated that more than 610 million kg of PCBs were sold in the United States between 1930 and 1978; about 60 percent appear to have been used in closed-circuit systems such as transformers, capacitors and hydraulic equipment, and the remaining 40 percent for open-circuit uses such as plastics, paints, glues, waxes, inks and so on. According to a report prepared for the EPA in 1983, approximately 13.5 percent of PCBs sold, i.e. 82 million kg, have apparently been released in the environment. The remaining PCBs are either still being used (60 percent), or have been buried or put in storage centres.

Figure 10, taken from this report (EPA, 1983), indicates the annual production of PCBs in the United States between 1962 and 1978 and contains an estimate of the quantities of PCBs released in the environment every year.

An analysis of this figure reveals that:

- production of PCBs was highest between 1963 and 1970, when more than 20 million kilograms per year were manufactured;
- PCB production in the United States ceased in 1978;
- in 1973, the use of PCBs in open-circuit applications was halted on a voluntary basis, with the result that discharges in the environment ceased after that year;
- since 1962, some PCBs have been produced involuntarily, especially in mineral oil used as an insulator in electrical. A study effected in 1981 by the Chemical Manufacturers Association in the United States (cited in the 1983 EPA study) revealed that prior to 1981, 6200 kg of PCBs per year came from oil contaminated at less than 50 ppm. At present, new oil used in electrical equipment in the U.S. may not contain more than 2 ppm of PCBs (2 mg/kg of oil).

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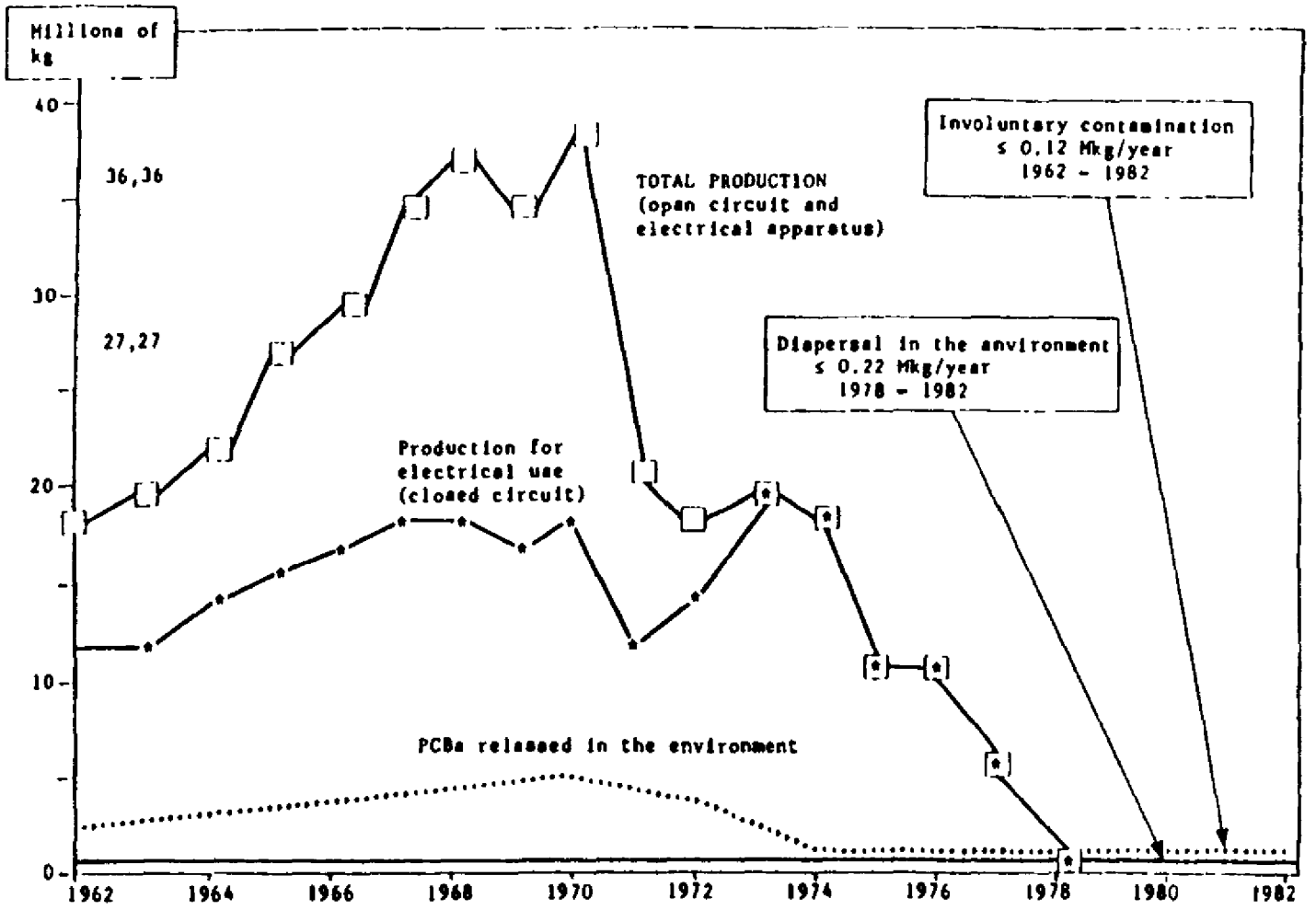


FIGURE 10: Annual Production of PCBs in the United States and Estimate of the Quantity Released in the Environment

The most widely sold commercial products prior to the 1970s were mixtures containing less chlorine (AR 1221 and AR 1242). Figure 11 indicates total production of PCBs by family of homologous compounds between 1930 and 1978; it reveals that more than 35 percent of such PCBs were trichlorinated biphenyls, followed in descending order of importance by tetras (22 percent), pentas (16 percent) and dichloros (11.6 percent). As we will see in section 7.7, in the environment the PCBs which are degraded the fastest are the mono-, di- and trichlorinated isomers, whose half-life is several months at the most. It may, therefore, be assumed that a considerable proportion of the PCBs which were released in the environment have now disappeared.

7.2 Quantities of PCBs Still in Use in the United States and Canada*

7.2.1 Electrical equipment insulated with askarel

Of a total of 610 million kg of PCBs sold in North America, about 356 million kg are still in use, i.e. 341 million kg in the United States and 15.5 million kg in Canada (including 6.36 million in Ontario, 5 million in Québec and the remainder in the other provinces).

In Québec alone, some 5 014 900 kg appear to be in use, distributed as indicated in Table 14; of this amount, approximately 1 million kg, or roughly 20 percent, belongs to Hydro-Québec (see Table 15 for the distribution of PCBs in its transformers and capacitors insulated with askarel).

7.2.2 PCBs in contaminated transformer oil

In Canada, we do not have any information from the federal Environment department on the quantities of PCBs in transformer oil. However, at Hydro-Québec it is estimated, on the basis of an initial evaluation, that about 13 million L of transformer oil are contaminated at more than 50 ppm. The average contamination of the oil is about 200 ppm, which means that this substantial quantity of contaminated oil contains no more than 2600 kg of PCBs overall, i.e. 0.13 percent of the total quantity of PCBs found in Hydro-Québec electrical equipment. The PCB content of these thousands of transformers insulated with oil is equivalent to the quantity of PCBs found in three transformers insulated with askarel.

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* According to the inventory prepared by Environment Canada (June 27, 1985).

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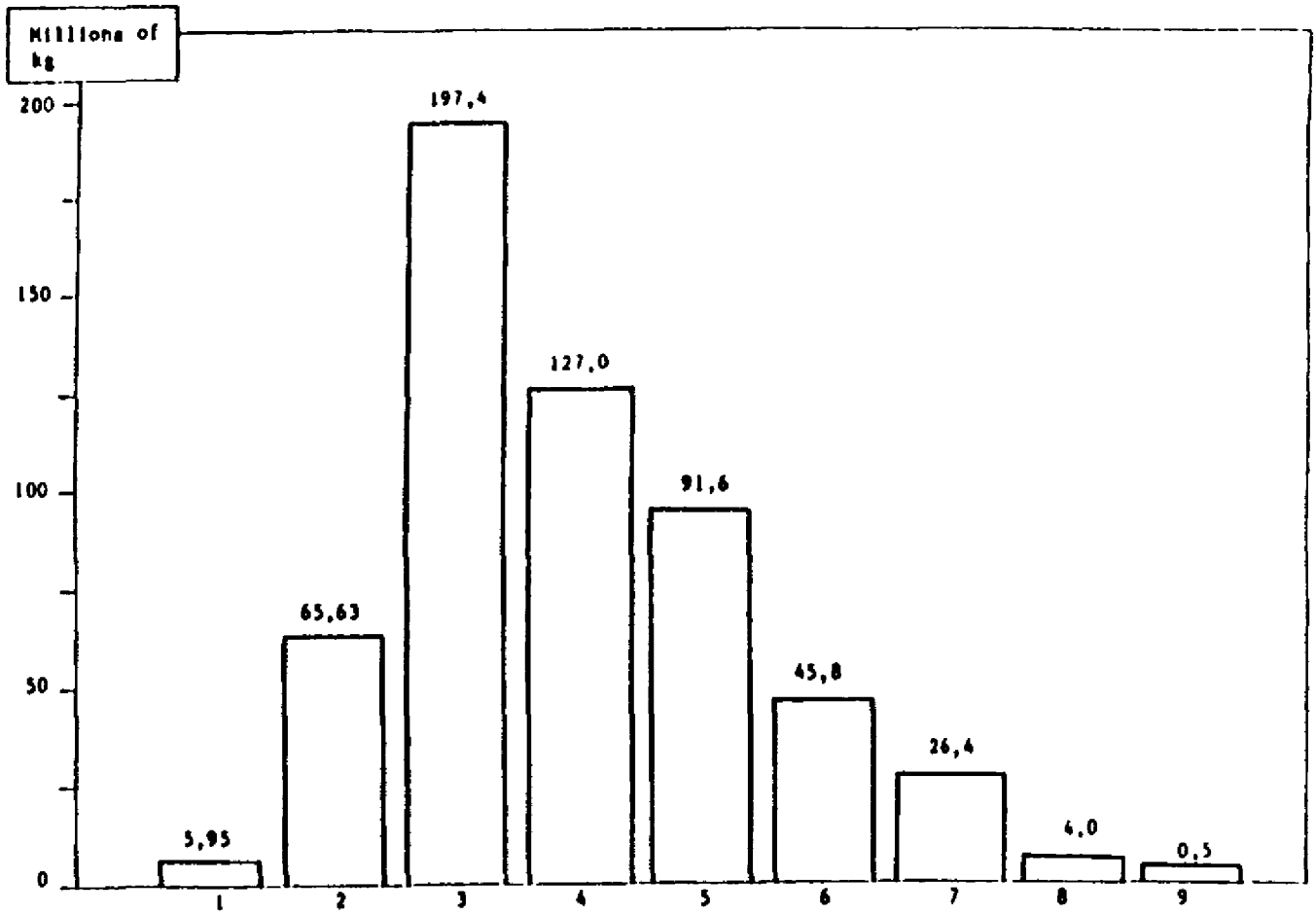


FIGURE 11: PCBs Sold in the United States between 1930 and 1978

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TABLE 14: Inventory of the Distribution of PCBs in Québec by Type of User

CATEGORY OF USER	QUANTITY (1000 kg)	(%)
Public services	1392.2	27.8
Forestry/Pulp and paper	667.0	13.3
Government facilities	509.0	10.1
Mines and smelting	459.0	9.2
Iron and steel industry	260.0	5.2
Hospitals and schools	185.0	3.7
Commercial warehousing	170.4	3.4
Textiles	107.1	2.1
Metal refining	87.1	1.8
Chemical products	83.2	1.7
Food industry/distilleries	77.6	1.5
Rubber products	46.9	0.9
Electrical equipment manufacturers	40.7	0.8
Cement industry	35.4	0.7
Oil refining	24.1	0.5
Other	869.6	17.3
TOTAL	5,014.9	100.0

TABLE 15: Distribution of PCBs at Hydro-Québec

TYPE OF EQUIPMENT	NUMBER OF APPARATUS	QUANTITY OF PCBs IN LITRES*
Transformers	115	140 000*
Capacitors	± 52 000	490 000*

* The density of PCBs is about 1.5 greater than that of water.

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From an environmental standpoint, the difference between askarels and contaminated oils is very important: the risk of contamination is much lower in the case of the latter. For example, if for contaminated oils we apply a limit of 1 g/day of PCBs (Environment Canada Chlorobiphenyls Regulations No. 3 (Release), 1985) which can be deliberately discharged in the environment in any region of Canada which is not a body of water, during the operation, repair, maintenance, breakdown, transportation or stocking of any electrical equipment, admissible discharges can be considerable where concentrations of PCBs are low, as Table 16 indicates.

It should be stressed that, at present, in Québec any fluid containing more than 0.15 ppm of PCBs (0.15 mg/L of fluid) is deemed to be hazardous waste and, from an environmental viewpoint, must be treated as though PCBs alone were involved. On the other hand, according to Environment Canada, it is at levels of 50 ppm or more that contaminated oil is considered a hazardous waste. The Ontario Department of the Environment and the EPA in the United States also use the same criterion.

In light of the foregoing, it appears obvious that environmental protection standards, to be truly effective, should be aimed above all at askarels, then at the most contaminated oils.

TABLE 16: Ratio of the Concentration of PCBs and the Volume of Oil Containing 1 g of PCBs

CONCENTRATION OF PCBs IN OIL (ppm)	VOLUME OF OIL CONTAINING 1 g OF PCBs (L)
0.15	6 666
2	500
10	100
50	20
100	10
1 000	1

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7.3 Distribution of PCBs in the Environment

The distribution of PCBs in the environment depends upon several factors:

. their entry point;

. their physical properties such as solubility and volatility (vapor tension), which determine their water-ground and water-air distribution coefficient;

. the incidence of bioaccumulation;

. their susceptibility to degradation in various environments where they are found.

According to Moolenaar (EPA, 1983), the total quantity of PCBs discharged in the environment in North America prior to 1975 is estimated at 82 x 10^6 kg, distributed in the following manner: 0.02 percent in the atmosphere, between 0.14 percent and 2.8 percent in the lithosphere (the solid crust of the globe as distinct from air and water) and the remainder in the hydrosphere, in salt water, fresh water and sediments.

The Atlantic Ocean appears to be the main reservoir of PCBs. However, the heaviest concentrations are usually found in lakes and rivers, although it is conjectured that drinking water ecosystems contain only a small proportion of the total quantity of PCBs. In Canada, the contamination of several water bodies has been studied, especially the Great Lakes in Ontario and the St. Lawrence and its principal tributaries in Québec (Eisenreich, 1982; Goulet and Laliberté, 1982).

Eisenrich estimates the total PCB content of the Great Lakes at between 0.44 x 10^6 kg and 0.5 x 10^6 kg, and that 88 percent of the substances are found in sediments (see Table 17).

We do not have data on the total quantity of PCBs in the St. Lawrence or its tributaries. Nonetheless, judging from the concentrations of PCBs measured in fish, palacypods and sediments in the water bodies studied (Goulet, Laliberté, 1982), it may be thought that in certain milieux, the quantities of PCBs per unit of volume approach those of the Great Lakes.

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TABLE 17: Estimated Quantity of PCBs in the Great Lakes (Eisenreich, 1982)

LAKE	PCBs (10^3 kg)
Superior	38
Michigan	57
Huron	69
Ontario	122
Erie	187

7.4 Pathways of PCBs in the Environment

7.4.1 PCBs for short-term use

PCBs for short-term use, or use in open circuits, find quicker, more direct pathways to the environment than those used in closed circuits. The PCBs that were in the past used as dust-control agents on dirt roads and in solvents for spreading insecticides contaminated the surrounding environment for very brief periods. Other products containing PCBs, such as glue, plastics, paint, printing paper and so on, reached the waste stage sooner or later and ended up in dumps, incinerators, waste-water treatment plants, or were discharged directly into the environment. Left in dumps, these products eventually released their contaminants into the ground, water or air. When such waste was burned in the dump or in an incinerator, PCBs were either destroyed completely, or evaporated into the air, depending on the temperature of combustion. A certain quantity of PCBs might be degraded, forming PCDFs which spread into the environment.

7.4.2 PCBs for long-term use

Plants manufacturing PCB-based products for long-term use released waste containing PCBs, either as liquids in effluents, or solids in dumps or burial sites.

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In electrical equipment, heet exchangers end hydreulic machinery, e leek is all it tekes to releese PCBs in the environment. Accidentel spills can elso occur when such equipment is transported to service* or storage sites.

According to the experts who wrote a report for the EPA in 1983, leaks from electrical epparatus accounted for less than 0.25 percent of discherges in the environment. The NAS (1979) feels that the releese of PCBs in the environment on sites where equipment conteining PCBs is stored is negligible.

However, in light of the quentity of PCBs stored every yeer in Canade, it is urgent to develop technologies for deetroying them.

7.5 Dispersel of PCBs in the Environment

Whet happens to PCBs releesed in the environment? Some of them sprsed in the ground and, even though they ere hardly soluble in water, they gradually reech equatic ecosystems. Because of their affinity for particles, they ettach themselves to particles suspended in the water and ultimately settle out es sediments, which account for more than 88 percent of PCBs in aquatic ecosystems. As we will see in section 7.9.3, water rarely contains more than several parts per billion (ppb) of PCBs, while ssdiments contain higher concentrations of them. Through bioaccumulation, e portion of PCBe are integrated into fish and land animals. Tha air also contains limited quantitiea of PCBs; there, they are carried by winds and float until rain, snow or the fellout of etmospheric dust precipitete them. Although the atmosphere contains limited quantities of PCBs, many specialists agree that it does contribute to spreading them. As there is no direct source of PCBs in northern Québec, their presence in fish and animal species can be explained by atmospheric transmission, a feirly efficient dispersal method.

* The new Canadian reguletion adopted in July 1985 prohibits the sale, import and export of electrical equipment containing more than 50 ppm by weight of PCBs, except when such equipment is an integral part of a building, plent or construction offered for sale.

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This hypothesis is highly plausible and, if it proves to be correct, it must be concluded that even were we to completely halt discharges of PCBs in the environment in Québec, we could not prevent releases in the United States and Ontario from reaching the province. The phenomenon is similar to the well-known problem of acid rain.

7.6 Bioaccumulation of PCBs

BPCs tend to accumulate in fish and animal species, including humans, through bioaccumulation, which is favoured by several factors, i.e. their low solubility in water, their great solubility in lipids and chemical reactions related to the catabolism and elimination of BPCs which occur in animals.

Bioaccumulation is defined as the ability of living organisms to concentrate the substances they absorb. For example, in fish, bioaccumulation is directly linked to the ratio of the concentration of a chemical substance in the fish and its concentration in water; this ratio is called the bioaccumulation factor. It depends on the species and the length of exposure to the substance, and is independent of the concentration in the milieu (in this case, water). This means that, for a fish species whose bioaccumulation factor for AR 1242 is 100 000, a concentration in the water of 0.01 ppb (0.01 µ g of PCBs/kg of water) is sufficient for the fish to carry a concentration of 1 ppm (1 mg of PCBs/kg of fish) after a certain time. Among minnows, small fish living in streams, the bioeccumulation factor has been found to be 32 000 for AR 1242 and 307 000 for AR 1254*. The maximum permanent concentration in this species is reached after about eight months of exposure. The bioaccumulation factor is different for each animal species.**

* It is normal for the components of AR 1254 to be bioaccumulated more extensively than those of AR 1242, as the former contains more molecules with high chlorine content.

** The bioaccumulation factor in certain piscivorous birds can apparently reach 10 (Goulet, Laliberté, 1982).

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The bioaccumulation factor also depends on the duration of ingestion of food. It is obvious that the maximum concentration may not be achieved where exposure is sporadic or short lived. This notion is very important in helping us understand the consequences for various living species of absorbing food with high PCB content.

Take humans, for example. Let us put forward the hypothesis that their bioaccumulation factor* of PCBs in fat is equal to 100 and that the time* required to reach the maximum permanent concentration (100 x concentration in the diet), for a given concentration in their diet, is one year. Where food containing an average of 1 ppm of PCBs (1 mg/kg) is ingested every day, lipidic tissues would contain 100 ppm of PCBs after one year. However, sporadically eating food containing 1 ppm of PCBs would produce only a small concentration of this substance in human lipidic tissues.**

The total quantity of PCBs accumulated is diluted in all of the organism's fat tissues; part of the quantity ingested is ultimately eliminated. The rate of elimination depends on the number and position of chlorine atoms in the congeners present in the diet. The lipidic tissues of Canadians contain average concentrations of PCBs of about 0.1 ppm to 1 ppm, although they occasionally eat fish with concentrations of PCBs over 1 mg/kg (1 ppm).

However, among heavy fish eaters, concentrations may be much higher because of bioaccumulation and the contamination of the human trophic food chain (see Figure 12).

A considerable portion of PCBs released in the environment eventually reach aquatic ecosystems. Fish, which have a high capacity to bioconcentrate PCBs, are eaten by piscivorous birds, other fish and animals, which further bioconcentrate PCBs. Humans ultimately absorb some PCBs when they eat such fish, animals and birds.

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* The bioaccumulation factor and the maximum permanent concentration time in humans are not known.

** The Legal Marketing Allowance (LMA) of foodstuffs containing PCBs in Canada is 2 mg/kg (2 ppm).

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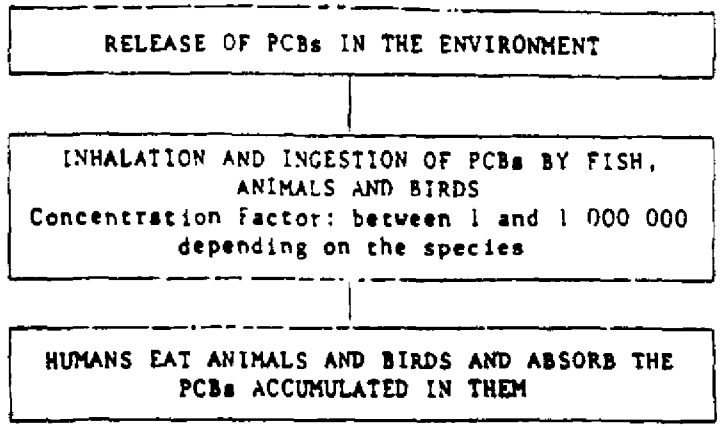


FIGURE 12: Bioaccumulation through Bioconcentration in the Food Chain

7.7 Degradation of PCBs in the Environment

7.7.1 Persistence of PCBs: the eternal question

Do PCBs remain indefinitely in the environment or are they ultimately degraded? If so, at what rate?

For a long time, it was thought that PCBs which reached the environment and living organisms could not be eliminated. This conviction was largely responsible for encouraging legislators to take measures aimed at banning the production of PCBs. Today, thanks to research on the degradation of PCBs in various ecosystems and on the presence of the substances in living organisms, including humans, it has been noted that PCBs are eventually degraded and the mechanisms which contribute to this elimination are better understood. These mechanisms comprise photolysis, aerobic and anaerobic bacterial degradation, hepatic catabolism among mammals and birds, transfer through mother's milk and

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eggs among birds and fish, and so. Generally, congeners containing few chlorine atoms, i.e. three or fewer, tend to be eliminated quickly; those containing many such atoms are degraded much more slowly.

7.7.2 Process of natural elimination of PCBs in the environment

During the 1970s, the Great Lakes were the ecosystem on which researchers most frequently focused their attention. In 1982, Eisenraich presented a summary of studies effected since 1962 on concentrations of PCBs in the environment of the lakes (International Symposium on PCBs in the Great Lakes, Michigan State University). Figure 13 summarizes data on concentrations measured in the water, the flesh of fish in Lakes Superior and Michigan, in the air above these lakes and in the eggs of seagulls nesting on islands in Lake Superior. A net reduction in the concentration of PCBs has been noted since 1974, i.e. one year after sales of the substance used in open-circuit systems were halted. It is, therefore, accurate to say that PCBs are gradually being eliminated from the environment.

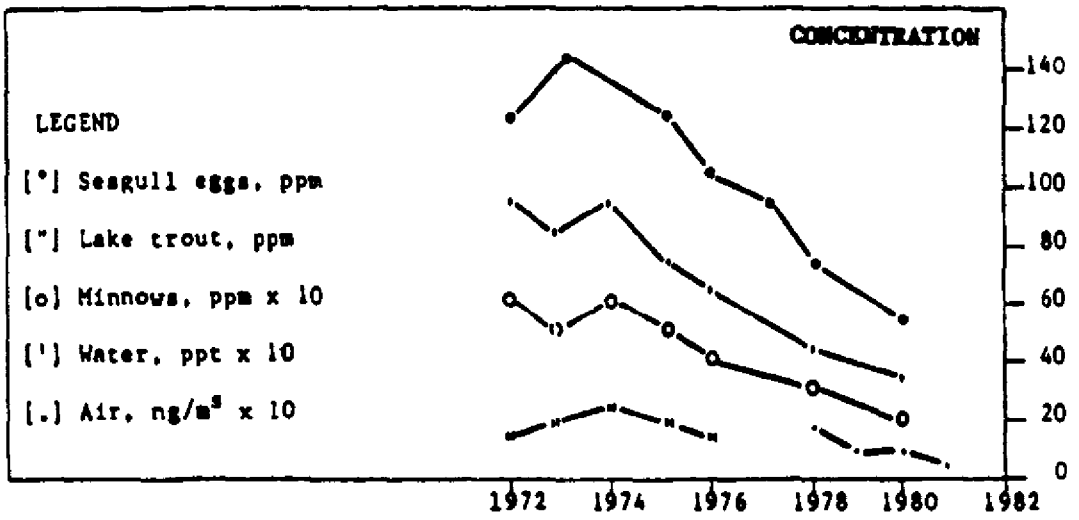


FIGURE 13: PCBs in the Environment of the Great Lakes: Changes over Time

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A more detailed analysis of this figure reveals that the concentration in:

- seagull eggs dropped from 145 ppm to 50 ppm between 1973 and 1980;

- the water fell from 7.8 ppt to 2.5 ppt (parts per trillion) between 1974 and 1980;

- minnows declined from 5.5 ppm to 2.2 ppm between 1974 and 1980;

- lake trout dropped from 20 ppm to 17 ppm between 1974 and 1976; we do not have results subsequent to 1976;

- the air fell from an average of 1.5 ng/m^3 (range of 0.9 to 3.5) in 1978 to an average of 0.3 ng/m^3 (range of 0.1 to 0.6) in 1981.

In light of these figures, it seems obvious that PCBs tend to eliminate themselves from the environment. With recent dumping control measures (July 1985) set out by the government in Regulations 2 and 3 of the Environmental Contaminants Act, it may be expected that the natural elimination process will be accelerated.

In the sections that follow, we will review various factors which affect the degradation of PCBs in different milieux and which, to some extent, enable us to predict long-term changes in the process by which PCBs are eliminated.

7.7.3 Estimated life of PCBs in different systems

7.7.3.1 Biodegradation in living organisms

In living organisms, the elimination rate of PCBs is primarily determined by the rate of hepatic catabolism, although some of the substances can be eliminated through lactation in mammals or egg laying in fish and birds. In certain species, the quantities of PCBs found in the bile and feces remain unchanged. It should also be noted that the elimination rate varies widely from one species to another.

According to studies carried out by J.F. Brown et al. (1976-1983) and Hara (1985) among workers in the transformer and capacitor manufacturing industries, and Chen (1985), among patients in Taiwan, PCBs can be divided into three groups, depending on their persistence: (1) non-persistent congeners which do not accumulate; (2) moderately persistent congeners with a half-life of between six months and five years; (3) highly persistent congeners, i.e. those which do not display a significant elimination rate during the period of observation.

We will study biochemical factors affecting the degradation rate in section 10.3. In lower mammals like rats and mice, PCBs tend to be eliminated more quickly than among humans, although the elimination of non-persistent and highly persistent congeners occurs in proportions similar to those observed in humans. (See Figure 14, taken from the 1983 EPA report, which indicates the excretion rate of PCBs in rat feces for mono-, di-, penta- and hexaphenyls).

It is important to note that the first group of non-persistant congeners accounts for approximately 90 percent of AR 1216 and 80 percent of AR 1242, while the group of highly persistent congeners, which comprises hexa-, hepta- and octachlorinated isomers with chlorine atoms in position 4-4', represent less than 50 percent of AR 1254 and more than 50 percent of AR 1260. This means that the quantity of PCBs accumulated in the organism and their persistence depend largely upon the mixture to which the subject was exposed, as the tendency for congeners with low chlorine content to be eliminated rapidly is general. It may be inferred that over time the PCBs to which we are exposed through food are, above all, congeners in the highly persistent group. Fortunately, all laboratory studies to date seem to show that these congeners have a very low rate of toxicity (Safe, 1984).

7.7.3.2 Biodegradation of PCBs in aquatic ecosystems: aerobic and anaerobic sediments

J.F. Brown et al. (General Electric Research and Development Centre, Schenectady, NY) presented a thorough review of the state of current knowledge in this field at the International Symposium on PCBs in October 1985 in Seattle. A summary follows.

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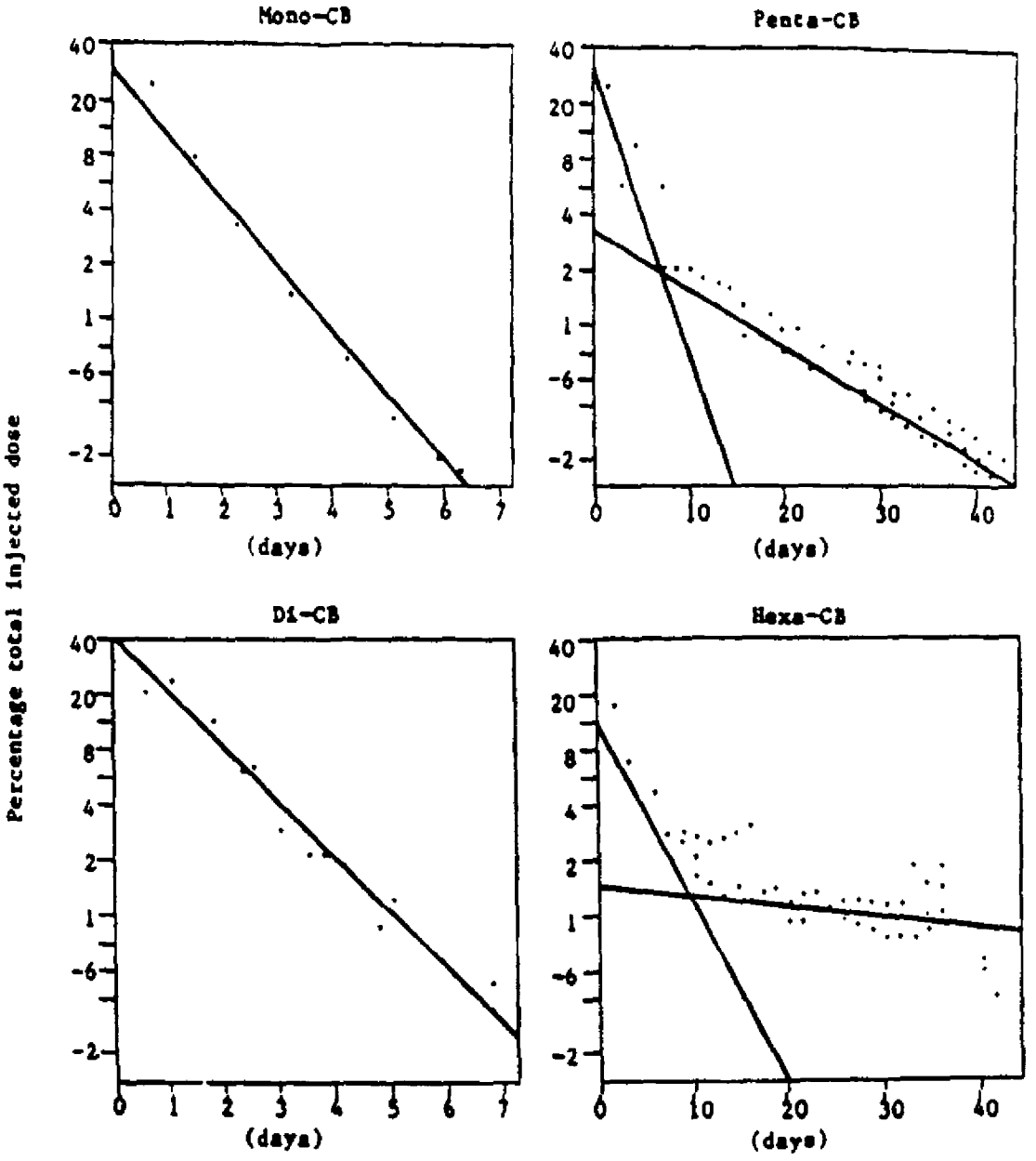


FIGURE 14: Percentage of Radioactivity in Rat Feces 7 and 42 Days after the Injection of 0.6 mg/kg of mono-CB, di-CB, penta-CB and hexa-CB

Aerobic bacterial biodegradation

In aerobic aquatic ecosystems (on the surface of sediments), large quantities of organic matter are metabolized by aerobic bacteria. This complex microbial population is able to metabolize most of the organic chemical components and natural matter present, including PCBs.

Until now, more than a dozen aerobic bacterial species have been isolated in earth and sediments, which degrade PCBs through oxidation. However, the different species identified do not all behave in the same manner in relation to the various PCB congeners. One group essentially attacks mono- and dichlorinated congeners, while others attack tetra- and pentachlorinated ones. Most of them are of the 2,3-dioxigenase type and usually attack congeners with chlorine atoms in positions 4,4', but without chlorine in positions 2,3 or 5,6. A small proportion of the types of bacteria identified are of the 3,4-dioxigenase type and generally attack congeners without chlorine in positions 3,4.

Researchers have noted that the concentration of PCBs in the flesh of fish in the Upper Hudson River fell by half in two years. Moreover, they found that the PCBs present were, above all, AR 1016 and that the bacteria isolated from sediments were of the 2,3-dioxigenase type. After two years, the residual PCBs resembled AR 1242; congeners with low chlorine content were degraded. Obviously, this group of aerobic bacteria has played an important role in the degradation of PCBs in this ecosystem.

Anaerobic bacterial biodegradation

Until very recently, it was thought that no degradation was possible in the deepest layers of sediments in aquatic ecosystems. However, in recent years, Brown et al. (1984) discovered that PCBs in such anaerobic systems are degraded, especially in the sediments of the Upper Hudson River and Silver Lake (Pittsfield, MA). Six different anaerobic bacteria were identified.

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Among those found in the Hudson River, a number of them can remove chlorine atoms in the para- (4) positions and others in the meta- (3 or 5) positions, although none is able to remove the chlorine atom in the ortho- (2 or 6) positions. The net effect has been to convert an appreciable proportion of the AR 1242 deposited over the past 20 or 25 years into mono- and dichlorinated congeners in the ortho- position. On the other hand, bacteria present in Silver Lake can reduce chlorine atoms in the ortho-, meta- and para- positions, with a slight preference for position 4. However, they leave intact several tri- and tetrachlorobiphenyls. The long-term effect has been to convert the AR 1254 and AR 1260 present into a mixture of congeners whose average content is similar to AR 1242 and AR 1248, but with a different distribution of congeners.

It is interesting to note that the congeners with chlorine atoms in position 4,4', usually deemed to be more toxic than other isomers with the same number of chlorine atoms but not possessing this substitution, are those which are eliminated the most rapidly in anaerobic sediments.

7.8 Dynamics of PCBs in the Environment

During the 1950s and 1960s, considerable quantities of PCBs were released in the environment; they dispersed until a certain equilibrium was achieved in different milieux, i.e. water-ground, water-air, water-fish and so on. This balance is shifting constantly according to fluctuations of PCBs added to, or eliminated from, the environment; it depends upon the various distribution coefficients between the different milieux. In light of data studied earlier, it is also possible to predict discrepancies in the distribution of congeners over time. As we noted, since 1973, influxes of PCBs have declined considerably, and elimination has outstripped bioaccumulation. Figure 15 explains this phenomenon by describing the process.

Given that world production of PCBs has ceased and that measures have been taken to control new influxes of the substances in the environment, we can hope that, from an environmental standpoint, the PCB problem is being solved.

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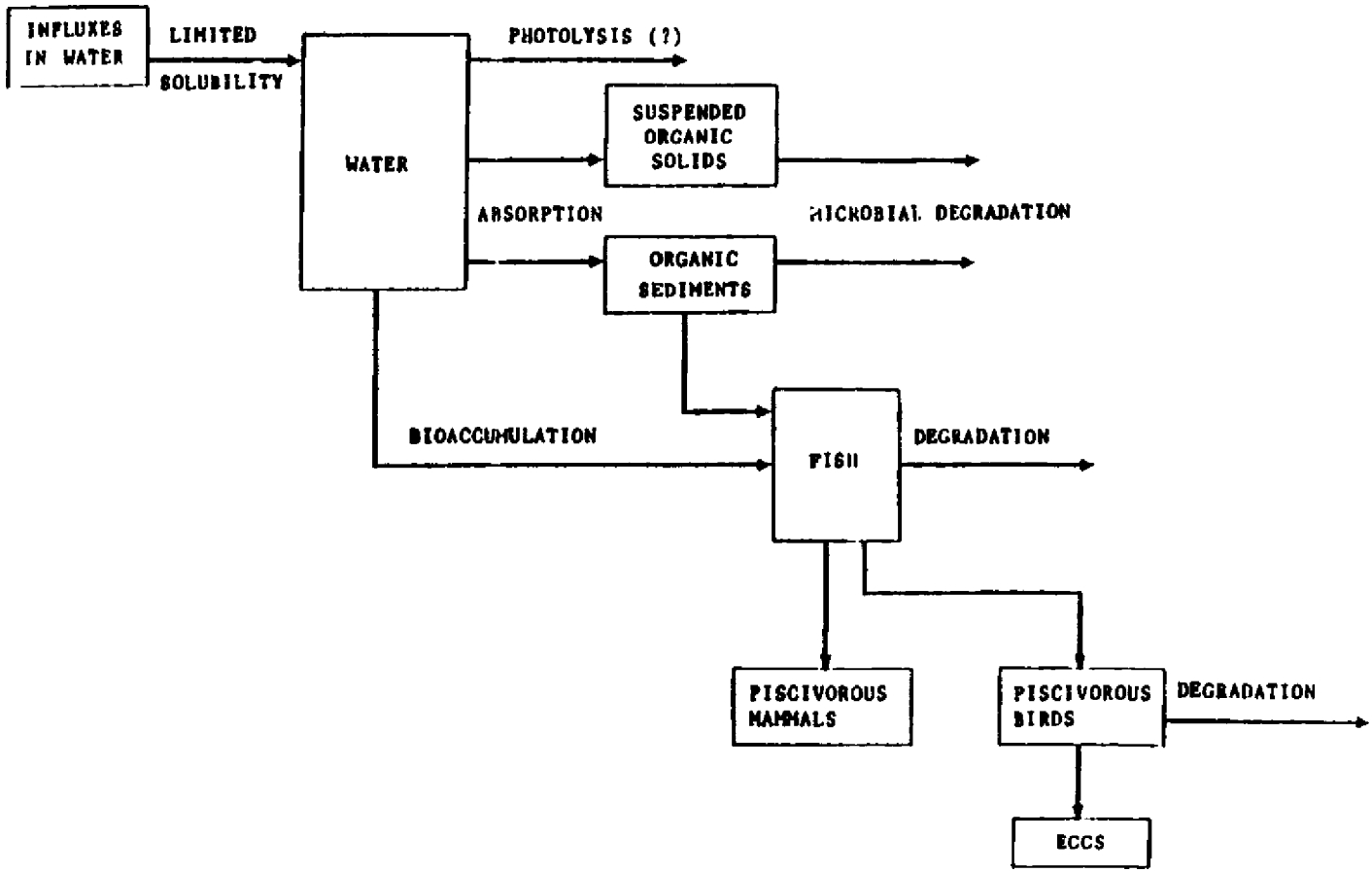


FIGURE 15: Mechanism of the Elimination and Degradation Processes (This Mechanism Prevails over Bioaccumulation)

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7.9 PCBs in the Québec Environment

It is impossible to review all of the studies published on environmental contamination through PCBs in Québec. However, readers who are interested may wish to consult syntheses of them published by J. Bélanger et al. (1977) and Goulet and Laliberte (1982).

Until now, samples have been collected throughout Québec, in the St. Lawrence, the Gulf of St. Lawrence, the principal basins of the river's tributaries, the Atlantic coast and the northwestern region of Québec. Samples have been taken in different milieux such as the water, sediments, fish, shellfish, tissues of birds (gannets, crested cormorants, seagulls, common terns, Arctic puffins, common murre and so on), seagull eggs, tissues of mammals (seals, polar bears, beluga whales and so forth), and in drinking water and milk.

An analysis of these studies reveals that the presence of PCBs in the Québec environment is fairly high, given that in certain zones direct influxes seem quite unlikely.

7.9.1 PCBs in the atmosphere

To date, it appears that the the presence of PCBs in the atmosphere in Québec has not been studied. However, as they have been found in fish and mammals in northwestern Québec, we believe that atmospheric transport is a highly efficient dispersal method.

7.9.2 PCBs in the ground

Here, too, it appears that no studies have been effected concerning the contamination of the ground in Québec. It is nonetheless plausible that the presence of PCBs in the air is an important source of ground contamination, through rainwater, snow, or simple atmospheric fallout of the substances.

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7.9.3 PCBs in water

A report published by Fisheries and Environment Canada (Québec region) mentions four especially important studies. According to the first, conducted in 1977 under the Canada-Québec agreement on the St. Lawrence, concentrations of PCBs varied between 10 ppt (10^{-12}) and 40 ppt (10^{-12}) in the water inlets and outlets of filtration plants in Montréal, Longueuil and Lévis.

The second study analyses untreated and treated water in the Montréal, Longueuil, Lévis, Sainte-Foy, Pierrefonds, Terrebonne, Pont-Viau, Saint-Eustache and Bécancour filtration plants, and was carried out by the Québec Service de protection de l'environnement between June 15 and July 25, 1977. This analysis did not reveal any PCBs (detection limit: 10 ppt).

The third study, dealing with waste water in Val-Cartier municipality, showed that concentrations of PCBs varied between 0.02 ppb (10^{-9}) and 3 ppb (10^{-9}).

Analyses of water from 26 sampling stations located in the St. Lawrence (Canada-Québec 1977) revealed concentrations below 10 ppt in most samples.

These results are hardly astonishing, given that PCBs are barely soluble in water: water is a poor indicator of the presence of the substances in the environment.

7.9.4 PCBs in sediments

An initial studied carried out by the Centre de recherches sur l'eau de l'Université Laval (CENTREAU, 1975) centred on the St. Lawrence, from Québec City to Trois-Pistoles. In 27 sampling sites out of 169, concentrations of PCBs between 20 ppb and 220 ppb were measured in fine sediments. This is equivalent to concentrations in sediments overall of between 4 ppb and 175 ppb, with an average of 70 ppb.

The second study, carried out by the Québec Service de protection de l'environnement, focused on the rivière Richelieu, from the U.S. border to its mouth. Results ranged from undetectable to 91.2 ppb, with an average of 20 ppb.

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7.9.5 PCBs in fish and Mollusca

Samples of shellfish (pelecypods) and fish (black chub and yellow pike) were collected during the summer of 1978 in 55 sites located in 11 of the most polluted basins of tributaries of the St. Lawrence in southern Québec.

Of a total of 202 analyses carried out on fish, the concentrations of PCBs measured varied between 0.063 ppm and 5.9 ppm. Among the 11 river basins studied in southern Québec, quantities measured in one or more sites in the Assomption, Saint-François, Richelieu and Nicolet rivers were equal to or in excess of the established Canadian marketing standard, i.e. 2 mg/kg (ppm) for fish flesh. Contamination was lower in the other basins.

The concentration of PCBs in the 37 pelecypods sampled appears, on average, to be 80 times lower than in fish, with variations of between 20 and 500 times less. All of pelecypods had concentrations of PCBs far below the 2 mg/kg standard; the maximum measured was 0.116 mg/kg (ppm).

7.9.6 PCBs and wildlife

Birds

In 1973, the concentrations of PCBs in the muscles, brains and eggs of piscivorous birds in the Gulf of St. Lawrence, such as gannets, crested cormorants, long-billed gulls and common terns, varied between 2 ppm and 80 ppm (Pearse, Gruchy, Keith).

In Labrador, in Arctic puffins and common murres, concentrations of PCBs were much lower, i.e. between 0.31 ppm and 0.41 ppm.

Mammals

The amounts of PCBs measured in the fat of seals and polar bears revealed that these contaminants are present around the entire periphery of Québec from the St. Lawrence estuary to James Bay, including the Gulf and the Atlantic and Arctic Oceans. In seals, concentrations of PCBs were found to vary (Canada, 1976) between 0.16 ppm and 9.2 ppm. In the fat of polar bears, average levels

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of 7.8 ppm were measured; in beluga whales (Actualité, February 1986), concentrations were about 576 ppm in fat and as high as 1725 ppm in the whales' milk. It should be pointed out that increasingly spectacular quantities of heavy metals, DDT, Mirex and APH (polycyclical aromatic hydrocarbons) are being found in belugas.

In Chapter 11, we will study in detail the concentrations of PCBs measured in humans.

7.10 Is Environmental Contamination Alarming?

7.10.1 Consequences for humans

Until now, no study has suggested that, in the concentrations measured in the population at large, PCBs might be injurious to health. However, heavy eaters of fish have concentrations of the substances close to those of exposed workers who have experienced health problems (see sections 11.1 and 11.3).

This leads us to believe that, had the release of PCBs in the environment not been controlled, several decades from now the levels of contamination reached might have seriously threatened health. In Chapter 11, we study in detail the potential effects of PCBs on health.

7.10.2 Consequences for flora and fauna

Laboratory studies suggest that in certain species of piscivorous birds or a number of animals, the concentrations of PCBs measured might be sufficient to affect their reproduction rate. For this reason, it is important to implement control measures to avoid adverse effects on the environment.

Fortunately, studies on the persistence and degradation of PCBs in the environment (see sections 7.6 and 7.7) show that the rate of elimination now seems higher than the rate of influx.

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8. TOXICITY AND MECHANISM OF ACTION OF PCBs

8.1 Growing Knowledge

Between 1970 and 1973, a number of researchers (Nichizumi 1970, Fujita 1971, Villeneuve 1971, Kimbrough et al. 1972) clearly showed that commercial PCB compounds altered the hepatic function through ultrastructural changes and the induction of a wide variety of enzymes which affect the biotransformation of substances which are exogenous and endogenous to the organism.

In 1973, while studying the effect of Aroclors 1221, 1254 and 1260 on the induction of hepatic enzymes, Chen and Dubois demonstrated that enzymatic activity increased with the chlorine content of the various products for most of the enzymes studied.

Given the considerable heterogeneity of commercial PCB-based products, the results of these studies raise several questions:

(1) Do dichlorobiphenyls produce the same effects as tetra- or hexachlorobiphenyls?

(2) Do all isomers displaying the same chlorination have comparable toxic effects?

(3) Are the toxicological effects observed due to the biphenyl nucleus, the position of chlorine atoms or the number of chlorines present in the molecule?

(4) Does the presence of PCBs stimulate the production of enzymes other than those which have been identified?

Researchers in different parts of the world have succeeded in answering many of these questions; research is intensifying to establish potential effects on humans.

In 1974, Johnstone, Ecobichon and Hutzinger showed that the enzymatic induction of many congeners varies widely. The same year, Hansell and Ecobichon (1974) proved that the effects of congeners on hepatic alteration with respect to ultrastructure or morphology also varied considerably. The degree of alteration was directly linked to the number of chlorine atoms and the presence of chlorine in position 4,4'.

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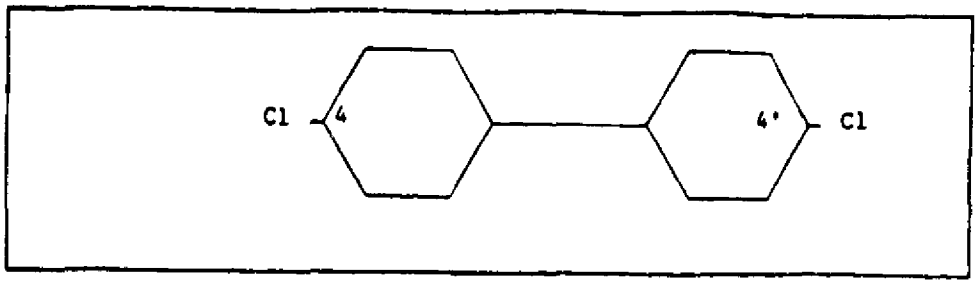


FIGURE 16: 4,4' - Dichlorobiphenyls

For example, the 2,2' dichloro- which has no chlorine in position 4,4' produces few alterations, while the 4,4' dichloro- produces more of them. Researchers have also noted that the 3,4,3',4' tetrachloro- congener is the most active of the congeners evaluated.

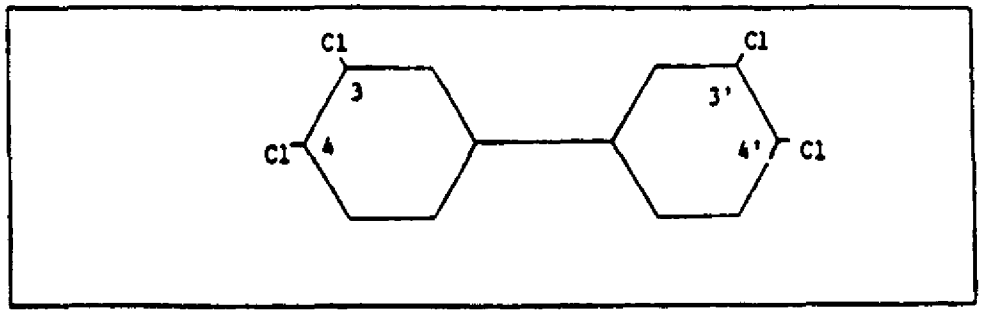


FIGURE 17: 3,4,3',4' - Tetrachlorobiphenyls

In 1975, Ecobichon and Comeau demonstrated that the results noted in the morphology of the liver are in correlation with functional effects. More precisely, PCBs carrying chlorine atoms in 4,4' have a higher enzymatic induction power than the other isomers with the same number of chlorine atoms but without this substitution.

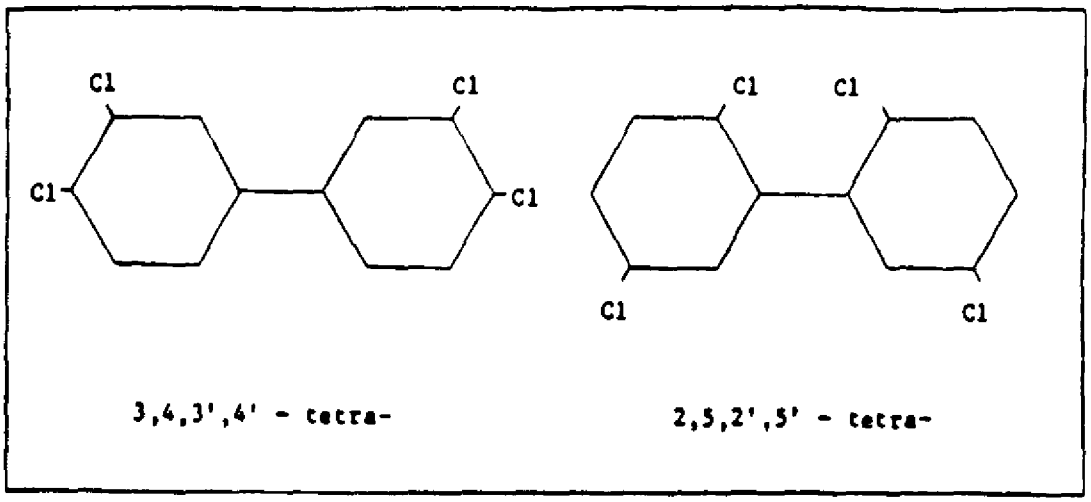


FIGURE 18: An Example of a Molecule Subatituted in 4,4' (on the Laft) and an Example without Subetitution in These Positions

Alvares et al. (1977) showed that AR 1016 activatas the enzymatic system of drug metabolism and the microsomal mixed function oxydase (MFO) in the same way as phenobarbital in humans and in animels. AR 1016 activates the P-450 cytochrome needed for the catabolism and metabolism of endogenous and exogenous substancee, such as phanobarbital and steroids.

This discovery is very important for medacina and toxicology. The induction of microsomal enzymetic activity (MFO) altera the rate of metabolism of drugs, endogenous substances such as staroids, and the degree of toxicity of a wide variety of toxic agents found in the environment. The toxic effect is uaually attenuated when the metabolism of these subatances is activatad.

Alvares concludes that PCBs can play a significant role in the biological response of living organisms exposed to a wida variaty of chemical agents in their environment.

Goldstein (1979) established a correlation between the induction of different hepatic enzymes of MFO by various congeners of PCBs and their toxicity. Here is his explanation: in 1977, Poland et al. showed that P-450 cytochrome could take numerous forms and that the various isomers and congeners of polychlorodibenzodioxins (PCDDs) induced these different forms with varying intensities. Cytochromes can be divided into two main groups:

(1) the phenobarbital (PB) type

(2) the 3-methylcholanthrene (3-MC) type.

The protein form of 450 cytochromes induced by PB-type substances differs from that induced by 3-MC type substances. For simplicity's sake, Goldstein called the 3-MC type cytochrome 448.

PB-type = P-450 cytochrome

MC-type = P-448 cytochrome

He concluded, as is the case with respect to dioxins (PCDDs), that congeners and isomers of PCBs which induce P-448s are much more toxic than those which induce P-450s. It has been shown that:

. some congeners, mainly those with little chlorine (mono-, di- and tri-) are inactive in the presence of inducing cytochromes (P-448 and P-450) and are scarcely or not at all toxic;

. some congeners have a PB-type effect only (or P-450) and are hardly or not at all toxic;

. others, displaying mixed induction (P-448 and P-450), are slightly more toxic than the preceding ones;

. some induce only P-448s and appear to be the most toxic. It is thought that 3,4,5,3',4' pentachlorobiphenyl displays this type of induction (MC-type).

Expanding knowledge has made it possible to demonstrate that substances belonging to the family of halogenated aromatic hydrocarbons produce a similar biological response and toxic effects, although at different doses. All of these substances, i.e. dibenzodioxin, dibenzofuran, azobenzene, naphthalene and polychlorinated biphenyls, have a similar chemical structure. Research suggests that they have a common mechanism of action: the model proposed by the scientific community appears in Figure 19 (Goldstein, 1979; Poland, 1979; Safe, 1984; Vickers, 1985; and so on).

Enzymatic induction occurs through the intermediary of one or more receptors (Ah receptor in the cytoplasm and the nucleus) genetically controlled by a simple locus called the Ah locus. This locus controls and modulates the expression of a battery of genes, including the P-448 cytochrome and aryl hydrocarbon hydroxydase (AHH), an enzyme associated with it which participates in the metabolism of halogenated hydrocarbons.

Affinity for the Ah receptor is a function of the structure of toxic substances.

8.2 Properties of the Ah Receptor

The Ah receptor appears to be an anomaly of adaptation in the evolution of living organisms (Vickers et al., 1985). Its structure and mechanism are analogous to those of steroid receptors; however, no steroid or other known endogenous compound can bind with this receptor.

Wide variations exist in the number and binding affinity of receptors between various tissues from the same animal and between various species. This may at least partially explain the appreciable differences in the toxicity of the same product in different tissues of the same animal or different species. For example, the greater susceptibility of guinea pigs and rats to thymus atrophy compared with mice and hamsters may be explained by the existence of eight times more receptors in the first two species.

The most powerful inducer of P-448 cytochrome (MC-type) currently known is 2,3,7,8-tetrachlorodibenzodioxin (TCDD) (see Figure 20). Its power of induction is 30 000 times greater than that of 3-MC. This substance is the most toxic in the family of polychlorinated aromatic hydrocarbons (dioxins, furans, PCBs, azo(oxy)benzenes, naphthalenes, quaterphenyls and so on).

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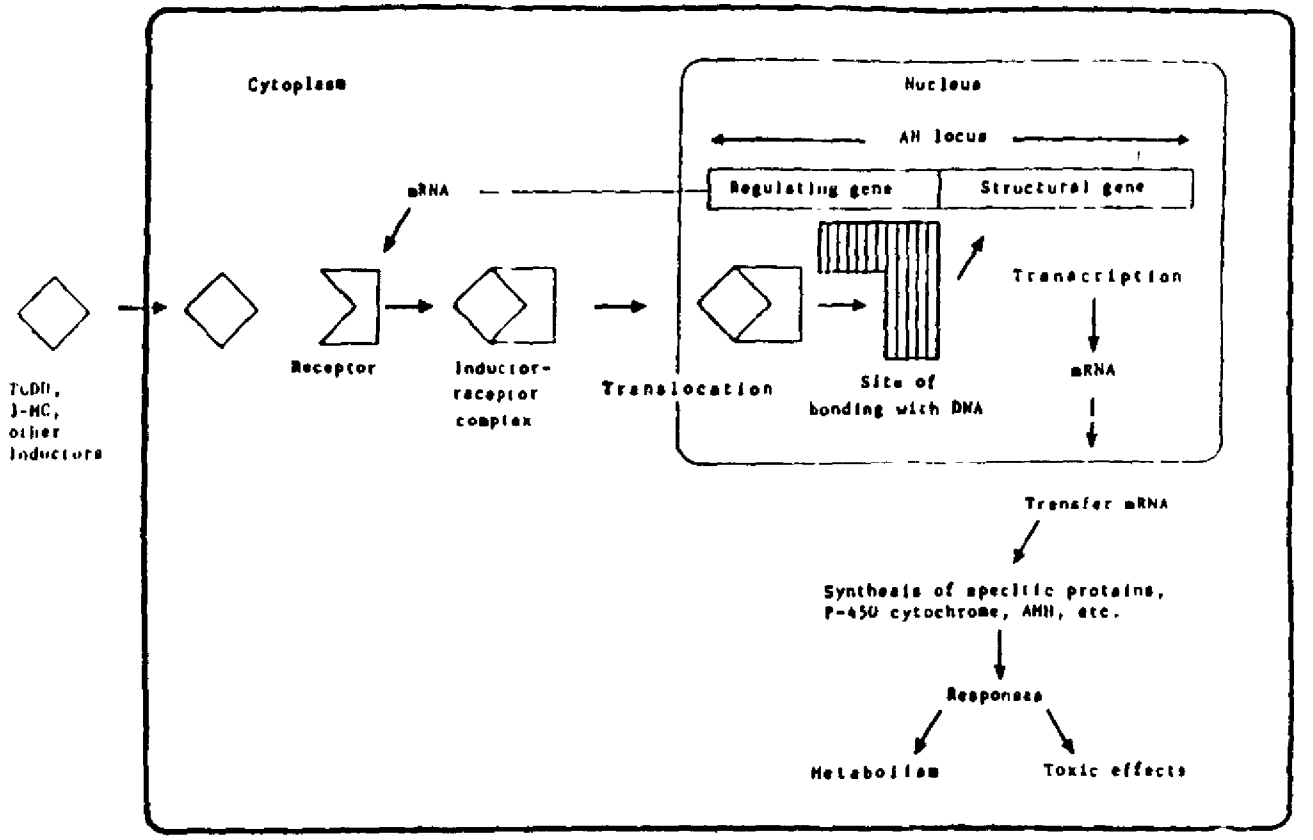


FIGURE 19: Model of the Mechanism of Action of Aromatic Halogenated Hydrocarbons at Cell Level

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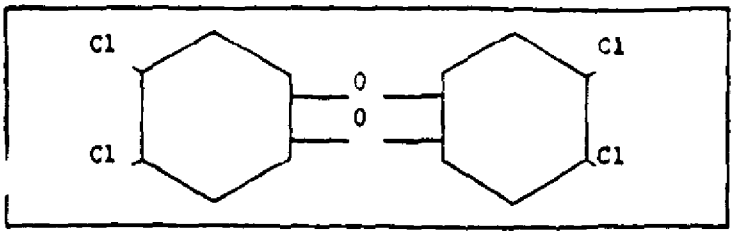


FIGURE 20: 2,3,7,8-(TCDD)

A series of studies (summarized by Safs et al. for the EPA in 1983 and for the Centre for Disease Control (CDC) in 1984) demonstrated that MC-type but not PB-type inducing substances had serious effects on animels, in particular:

. weight loss;

. atrophy of the thymus and spleen;

. lipid accumulation in the liver and hepatomegaly;

. porphyria.

It was also shown that:

(1) Different substances have widely varying effects: those of dioxins are greater than those of furans and much greater than those of PCBs.

(2) For the same substance, there are major variations (in the order of 3000) in toxic effects from one species to another.

(3) Congeners of dibenzodioxins other than 2,3,7,8-(TCDD) are all between 1 and over 30 000 times less toxic than it is.

(4) Congeners of dibenzofurans other than 2,3,7,8-tetrachlorodibenzofuran (TCDF) are between 1 and 30 000 times less toxic than it is.

(5) The biological effects of PCBs vary widely according to the different congeners. Researchers all seem to conclude that the most toxic of the polychlorinated biphenyls are:

3,3',4,4'-tetra;

3,3',4,4',5-penta-; and

3,3',4,4',5,5'-hexa.

These three substances are all inducers of MC-type cytochrome, like 2,3,7,8-(TCDD), but to a much less degree, about 20 times less. It has been shown that their coplanar structure resembles 2,3,7,8-(TCDD) and 2,3,7,8-(TCDF). Fortunately, these three PCBs are absent from commercial products, except for 3,3',4,4'-tetra-, traces of which are occasionally present. Chen found that even though 3,3',4,4' accounted for approximately 1 percent of the total PCBs in the rice oil responsible for the poisoning in Taiwan, this substance was not found in the blood of most of the patients, nor in the intestinal lipids of a patient who died in an accident two months after the poisoning. This suggests, in accordance with animal experiments, that the organism metabolizes and excretes this type of PCB fairly rapidly.

The three PCBs (tetra, hexa and penta) in Figure 21 have the following traits in common:

(1) cholorinated substitutes in position 4 - 4' (para- position);

(2) at least one substitution for each benzene nucleus in meta- positions (3,3' or 5,5');

(3) no substitution in ortho- positions (2,2' or 6,6').

If we modify these structures, either by removing chlorine atoms from para- (4,4') or meta- (3,3',5,5') positions, or both, or by adding chlorine atoms in ortho- (2,2',6,6') positions, toxicity declines. A number of these new congeners are inactive, while others are of the phenobarbital (PB) type, and the remainder are mixed (PB + MC) with toxicity higher than the PB type but lower than the MC type alone.

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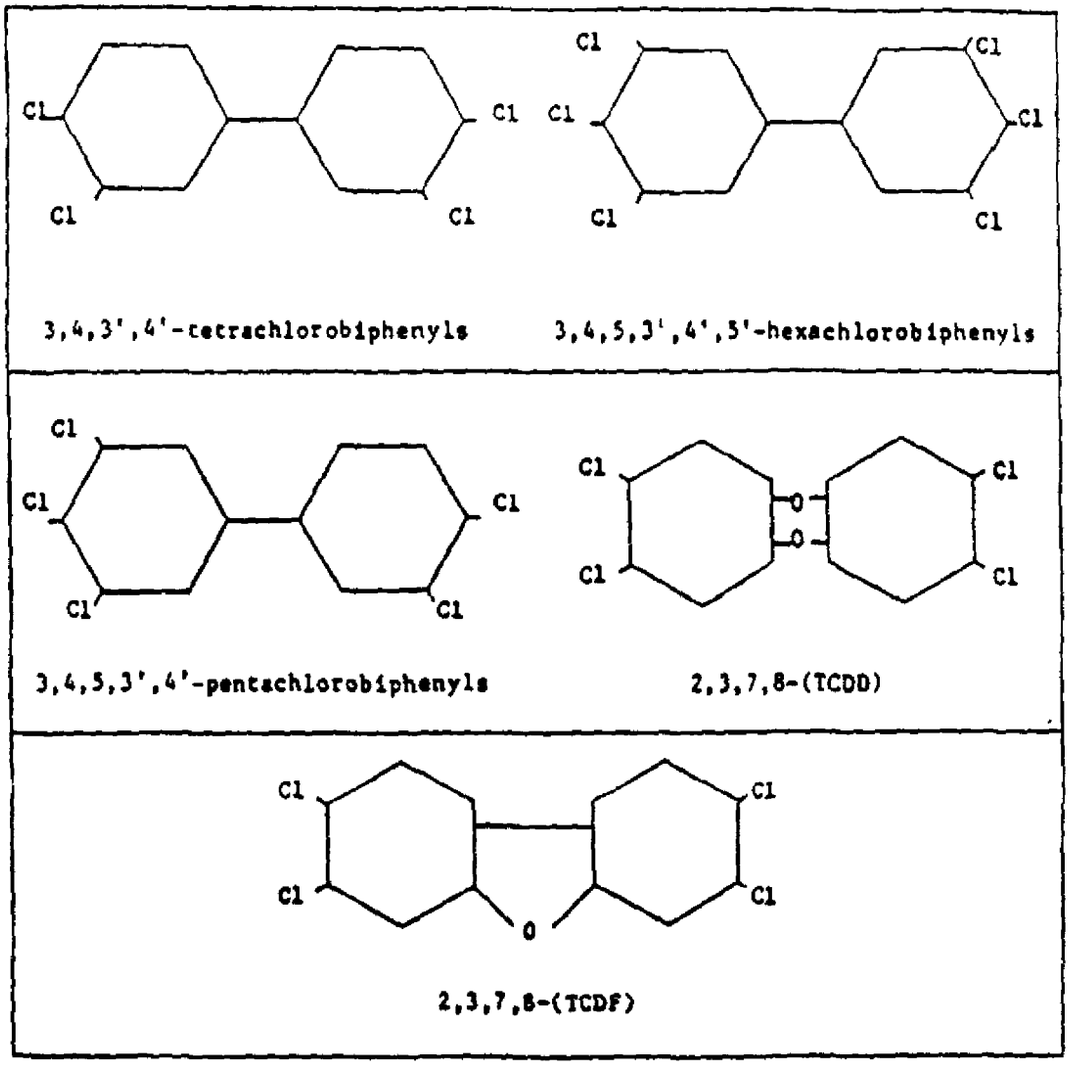


FIGURE 21: Related Structure of Several Molecules Belonging to the PCB, Dioxin and Furan Families

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At present, for most of the 209 congeners of PCBs, it is possible to establish a correlation between the structure (number and position of chlorine atoms) and relative biological and toxicological effects in rats (Safe, 1984).

8.3 Other Mechanisms of Action

We know that there are other mechanisms of action of toxicity. Sono et al. (1985) have shown that the metabolism of porphyrine, in addition to being affected by the P-448 cytochrome and AHH, is also affected by another mechanism which interferes with the catabolism of uroporphyrines.

It appears that the fact that responses vary according to the animal species cannot be explained by the variation in the number of Ah receptors alone. It is currently difficult to show that such responses are attributable to greater cellular reaction to the activation of the receptor in a number of species.

8.4 Enzymatic Activity in Certain Organs and Tissues

Dioxins, furans and several PCBs induce enzymatic activity in tissues other than the liver, i.e. the lungs, kidneys and skin, although to a lesser extent. This may partially explain toxic effects on these organs. Once again, it must be stressed that response varies from one species to another.

8.5 PCBs, an Antidote for 2,3,7,8-TCDD

The preliminary findings of recent research conducted by Stephen Safe at the University of Texas are now available (interview with the author, April 1986). The researcher and his team tried to find an antidote for 2,3,7,8-TCDD in the event of acute poisoning. The best known antidote is AR 1254; it is about 800 times less toxic than 2,3,7,8-TCDD in rats. When AR 1254 is given to an animal into which 2,3,7,8-TCDD had been injected, the Aroclor acts competitively on the same receptors as those affected by the 2,3,7,8-TCDD, resulting in the inhibition of the latter's toxic effects.

8.6 Mechanism of Action and Future Prospects

More extensive knowledge of the mechanism of the toxicity of TCDDs will enable us to better evaluate the effects on health of toxic halogenated aromatic hydrocarbons and will help determine whether humans are sensitive or resistant to these substances.

To accurately assess the risk of toxic effects to which we would be exposed by accidentally absorbing substantial quantities of PCBs, current research must, in addition to qualitatively and quantitatively evaluating the different congeners present in the organism, make it possible to estimate the toxic potential of the dose of PCBs absorbed, bearing in mind the relative toxic potential of each congener, and without overlooking differences in their degree of persistence.

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9. PCBs AND ANIMAL TOXICITY

With respect to the effect of halogenated hydrocarbons on animals, the enormous difference in toxicity noted between the species remains an enigma. Guinea pigs are the most sensitive, followed by monkeys, rats, rabbits, mice, dogs and hamsters. The sensitivity of guinea pigs to TCDD, the most widely studied substance, was 5000 times greater than that of rats. The discrepancy in toxicity is even greater still between the different isomers of dibenzodioxins ($\geq 10^4$). Studies of the toxicity of furane and PCB MC-type (P-448) enzyme inducers suggest that their effects on various animal species are similar to those of dioxins, provided, however, that the dose administered is adequate. It should also be noted that the impact on each organ varies from one species to another.

9.1 Acute Toxicity

The LD50 (lethal dose to 50 percent) in mice and rats of different PCB-based commercial products varies from 2 g/kg to 20 g/kg on average.

Bruckner (1973) showed that the oral LD50 for rats, with doses spread out over 14 days, was on average 4.25 g/kg, with a confidence interval of 95 percent between 2.80 g/kg and 6.38 g/kg. It should be noted that for doses between 200 mg/kg and 1 g/kg, no sign of acute toxicity was noted.

Signs observed four hours after absorption of the dose:

- diarrhea;
- sluggishness;
- reduced response to pain;
- slight chromodacryorrhea;
- unusual behaviour and stance, characterized by arching the back and raising the rear part of the trunk.

During the next 24 hours, the rats' condition deteriorated:

- profuse diarrhea, chromodacryorrhea, adipsia, oliguria and anorexia;
- absence of response to pain;
- weakness;
- ataxia, followed by coma and death.

9.2 Chronic Toxicity

A considerable number of studies have been conducted on this subject, although it is impossible to cite all of them. We might note in passing that when doses are adequate, PCBs can produce multiple toxic responses in animals, summarized below.

1. Functional and structural damage to the liver 2. Weight loss 3. Atrophy of the thymus and spleen	Most widespread toxic effects in various species.
4. Suppression of the humoral immunological system	Not found in all species.
5. Cutaneous lesions on hair follicles and meibomian glands	Dilation of hair follicles and keratinization. Atrophy of the sebaceous glands. Syndrome resembling chloracne, in monkeys.
6. Porphyrias	Observed in monkeys and rats; probably found in all species.
7. Damage to intestinal mucosa (metaplasia)	Found in rats and monkeys.
8. Effects on reproduction	In rats, monkeys and mice: PCBs are not teratogenic but fetotoxic. Drop in fertility. Increase in the number of abortions and stillbirths. Reduction in post-weaning survival rate.

9. Effects on lipid metabolism (peroxidation)

10. Some studies suggest possible alterations:
    - in thyroid function (Barsano, 1981)
    - of the hematopoietic system (Beran, 1983)
    - of the pituitary and adrenal glands (Dunn, 1983)

11. Carcinogenic effects

As the carcinogenic effects of PCBs and their effects on reproduction are ov far the problems which create the most anxiety in the public at large, we will pursue these questions further.

9.3 Animal Experimentation and Cancer

Through experimentation, it has been shown that hundreds of products can cause cancer in one or several animal species. However, some products are so common that it is impossible to keep them out of the environment. Moreover, no carcinogenic effect on humans has been demonstrated for products such as isoniazid and saccharine.

We must display considerable caution before accepting as valid for humens positive or negative results observed with respect to animals. Experimentation and epidemiology must complement each other.

Furthermore, we must not overlook the fact that humans are never exposed to a single carcinogenic agent, but to many potentially carcinogenic physical or chemical agents. It is likely that most human cancers result from the repeated action of a single factor, such as cigarette smoke, asbestos and so on.

All chemical or physical carcinogenic agents have a common property, i.e. they can alter DNA molecules; this alteration is a key factor in carcinogenesis.

At present, in the realm of animal experimentation, the following theory is usually accepted to explain the onset of cancer: the birth of a cancer is the result of a long process, requiring several modifications of the cell. At least two phases can be discerned in animals:

- Initiation, a phase during which the cell undergoes one or more mutations capable of causing cancer. The mutated cell retains its ability to reproduce, despite the functional change.

- Promotion, which can occur much later. It takes place when the cells of the tissue containing the ''initiated'' cell are subjected to the action of an agent which, among other possible effects, provokes a proliferation of initiated cells, forming clones of mutated cells. The mechanisms regulating pro-

motion are not well-known; we are aware of products with an inhibiting effect on the sequence of phases necessary for the onset of a cancer.

However, the distinction between initiation and promotion is somewhat artificial, as we know that some allogenic factors can be both initiators and promoters of cancer, e.g., ionizing rays. This distinction helps us understand how some substances can intervene in the process of carcinogenesis.

9.3.1 Do PCBs have initiator effects?

9.3.1.1 Mutagenicity

Three types of tests designed to detect the mutagenicity of PCBs on DNA have been effected, i.e. the Ames Test, Micronucleus Test and Unscheduled DNA Synthesis Test. Results indicate that PCBs do not appear to be mutagenic (G.S. Probst et al., 1981; Watanabe et al., 1981; EPA, 1982, p. 216).

9.3.1.2 Ability of PCBs to induce cancer in laboratory animals

(a) In rodents:

Two studies of male mice (Nagazaki et al., 1972; Ito et al., 1973) have shown a higher incidence of hepatocarcinoma in animals which received Kanechlor 500 in concentrations of 500 ppm in their diet for 32 weeks.

Another study (Kimbrough et al., 1975) has shown a significant increase in hyperplastic nodules (benign lesions) and hepatocarcinoma in female rats given AR 1260 at a concentration of 100 ppm in their diet for 84 weeks.

In 1978, the National Cancer Institute published a study on the possible carcinogenesis of AR 1254 in female and male rats receiving three different doses in their diets (25 ppm, 50 ppm and 100 ppm) for between 104 and 105 weeks. Researchers found:

- hepatomas and hepatic hyperplastic nodules. The incidence of hepatomas is not significant, but the incidence of hyperplastic nodules appears to be related to the administration of PCBs; and

- metaplasias and carcinomas of the gastrointestinal tract, but the incidence of carcinomas is not significant. This was confirmed by Morgan et al. in 1981.

No research has resulted in a significant increase in hepatocarcinomas in rodents fed compounds with lower chlorine content, such as Kaneclor 500 (55 percent) or AR 1260 (60 percent).

(b) Dogs:

No carcinogenic effect has been shown in dogs which absorbed high doses, i.e. more than 100 ppm, of PCBs for two years and were destroyed at the end of the period.

9.3.2 Promoter and inhibitor effects

By exposing animals to carcinogens with known initiation potential and to PCBs, research conducted since 1974 has confirmed that the effect of PCBs on cancer depends on the time they are administered in relation to the time when cancer-inducing substances are administered.

9.3.2.1 Makiura et al. (1974)

This study indicates a high incidence of hepatomas with the administration of three known carcinogens: 3'-Me-DAB (incidence: 65 percent), N-2 FAA (53 percent), and DENA (92 percent). However, if these compounds are administered simultaneously with PCBs, cancer is inhibited (incidence: 0 percent).

9.3.2.2 Kimura et al. (1976)

These researchers reassessed the effect of PCBs on rats which had been given 3'-Me-DAB. No cancer was obtained with PCBs, but a 13 percent incidence of it with 3'-Me-DAB was noted. When PCBs were given before the administration of 3'-Me-DAB, the incidence of cancer remained 13 percent, but rose to 64 percent when they were given afterwards.

9.3.2.3 Nishizumi (1976)

According to Nishizumi, Peraino had shown in 1971 that phenobarbital (PB) inhibited the carcinogenic effect of 2-FAA when given before, and enhanced its effect if given after. Knowing that PCBs and PB have similar effects on the enzymatic activity of the liver, he wanted to compare their carcinogenic effect when combined with DENA, a known carcinogen. His research reveals that the effects of PCBs and PB are similar: when either product is administered after treatment with DENA for 5 or 10 weeks, a significant increase in cancer is noted.

9.3.2.4 J. Hendricks et al. (1977)

This team showed that when PCBs are administered to rainbow trout at the same time as aflatoxin, a powerful initiator of cancer, the incidence of cancer is reduced significantly compared with the onset rate when aflatoxin alone is administered.

9.3.2.5 Ito et al. (1978)

Like Nishizumi (1976), Ito et al. once again proved the similar effect of PCBs and phenobarbital on promoting cancer once the latter is initiated with 2-FAA. Moreover, they showed that when a partial hepatectomy is performed on rats given 2-FAA, there is also a slight cancer-promoting effect. The promoting effect is further accentuated by adding PCBs or phenobarbital.

9.3.2.6 Preston et al. (1981)

Preston et al. wished to verify a hypothesis put forward by several researchers, who thought that the promotion of cancer by PCBs might be due more to its contaminants, especially furans (PCDFs). They compared the effect of purified AR 1254 with that of AR 1254 containing PCDFs. Their research shows that:

- administered alone, AR 1254 and PCDFs do not initiate cancer;

administering AR 1254 or AR 1254 with PCDFs after the administration of DENA had a cancer-promoting effect but the effect of the second compound was more pronounced.

They concluded that PCDF impurities were not the only factors in the promoter effect attributed to AR 1254.

9.4 Remarks on Cancer and PCBs

9.4.1 The results obtained by Kimbrough in rats and Nagazaki and Ito in mice suggest that PCBs have a cancer initiating effect. However, some researchers feel that account must be taken of the fact the cumulative doses of PCBs (1.2 g to 3 g) administered to laboratory animals are sufficient to largely affect their immunological systems (atrophy of the thymus and spleen, suppression of the humoral immunologic system) and cause weight loss, thereby appreciably altering the animals' resistance to cancer. The incidence of hyperplastic nodules and hepatomas in animals in control groups was not zero; we cannot, therefore, exclude the possibility of the promoter effect of PCBs in predisposed animals.

9.4.2 When lower doses are given, gastrointestinal metaplasias and hepatic hyperplastic nodules appear, creating serious doubt among researchers, as a number of them believe that such lesions could be pre-cancerous.

9.4.3 PCBs seem to be both a promoter and an inhibitor of liver cancer, depending on when they are administered in relation to the administration of carcinogens known to initiate cancers. Moreover, this effect is comparable to that of other known enzymatic inducers, such as phenobarbital.

9.4.4 For an animal study on cancer to be valid, an adequate number of animals must be evaluated over their entire lives. For example, dogs live for between 10 and 14 years; it is, therefore, hardly surprising that no cancers have been found in them after two years.

9.4.5 It seems extremely hard to draw conclusions on the impact of these results on humans. Research should be pursued to further clarify this question.

9.5 Effects on Animal Reproduction

9.5.1 Estrogenic potential of PCBs and consequences

It is increasingly apparent that PCBs alter the metabolism of sexual hormones, through 450-type cytochromes, enzymes which play a role in the cellular metabolism of steroid hormones such as estrogens and progesterone. We know that certain congeners activate the MFO system through P-450 cytochrome, which suggests they have an estrogenic potential.

It has been shown (Allen et al., 1979; Barr et al., 1979; Sager et al., 1983; Barlow 1982 and so on) that by administering certain doses of PCBs to laboratory animals, effects are produced on the reproductive function, especially an alteration of the menstrual cycle (irregularities, lengthening of the cycle, increase in the duration of menstruation, alteration of the rate of bleeding and, occasionally, inhibition of ovulation), leading to a reduction in the fertility rate, an increase in the number of abortions and stillbirths, a drop in birth weight and an increase in the rate of post-natal deaths. However, these studies were unable to show that PCBs have a teratogenic effect, i.e. no effects were demonstrated on morphology.

9.5.2 Dose-effect relationship in certain species

A Non Observed Effect Level (NOEL) has not been defined for animals. The wide variation in response from one species to another does not simplify the task of researchers who want to define one.

In rhesus monkeys, Allen et al. (1979) noted that nursing babies whose mother had ingested 5 mg of AR 1248/kg of food during her eight-month pregnancy displayed important signs, i.e. a drop in weight and retarded growth at birth, acne, edema of the eyelids and skin pigmentation, and neonatal edema. The total quantity the mother ingested during the eight months of pregnancy corresponded to 210 mg of PCBs; the babies consumed PCBs during the first eight months of their lives through their mother's milk, which contained between 154 µ g of PCBs/L and 397 µ g of PCBs/L. However, for a diet containing between

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0.5 mg and 1 mg of AR 1248/kg of food (total consumption during the eight months of pregnancy: 60 mg), Allen noted very few effects, i.e. a slight drop in birth weight, and poor pigmentation during the lactation period, with subsequently normal development.

According to a study carried out by Spencer (1982), AR 1254 had harmful effects on reproduction in rats which consumed between 25 ppm and 900 ppm of PCBs (mg of PCBs/kg of food). A rat with an average weight of 250 g which consumed 20 g of food per day ingested, on average, 2.5 mg of PCBs/kg of body weight per day. At 25 ppm, no effects on the attachment and embedding of the fertilized ovum in the endometrium were noted on day 12, but the survival rate fell as the dose increased above this level. At 100 ppm, birth weight was significantly reduced ($P \leq 0.05$).

Thus, in rats, the NOEL for reproduction is less than 2.5 mg of PCBs/kg of body weight per day, which is clearly higher than the NOEL which can be assumed in rheaus monkeys, as we just noted, because a diet for the latter of 1 mg of AR 1248/kg of food (1 ppm) over eight months corresponds to about 20 µ g of PCBs/kg of body weight ingested, on average, per day. Therefore, it seems that the NOEL in the rhesus monkey is below 20 µ g of PCBs/kg of body weight per day.

An analysis of studies conducted on mammals and birds reveals the effects on reproduction potential in some species when their diet contains between 1 ppm and 100 ppm of PCBs. As we know that piscivorous birds and fish-eating animals can concentrate PCBs by a factor of between 1 and 10 times greater than the concentration absorbed, it is readily apparent that had we continued to release PCBs in the environment at the same rate as in the 1960s and 1970s, they would have become a serious threat to some species. It is suspected that these substances are seriously threatening beluga whales.

It is acknowledged that PCBs in birds are inert as long as they remain in body fat. However, the situation changes markedly when food consumption decreases, for example, during reproduction and migration seasons and unfavourable periods. When body fat is consumed to provide energy, considerable quantities of organochlorinated compounds are released in the blood and reach other tissues, the liver, brain, gonads and so forth. Toxic effects could appear during these periods in the most sensitive species.

Although these data are incomplete, we believe they are sufficient to justify the implementation of efficient but rational control measures to prevent the dispersal of PCBs in the environment.

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10. PHARMOKINETICS OF PCBs

10.1 Pathways of PCBs

PCBs can enter the organism in three ways:

by ingestion: intestinal absorption ~ 100%

by inhalation: absorption ~ 100%

by skin contact: rapid, intense absorption (Wester et al., 1983)

The properties of skin absorption on the abdomen in rhesus monkeys and guinea pigs are similar to those in humans, which enables us to study the phenomenon in the laboratory. Wester et al. (1983) studied the rate of skin absorption, the elimination rate in urine and the efficacy of washing skin for Aroclors marked at ^{14}C in rhesus monkeys and guinea pigs. Washing was effected with water and acetone. The following conclusions were reached:

- Without washing the skin appears to have a fairly high capacity for absorption. In guinea pigs, 33 percent of the AR 1242 and 56.75 percent of the AR 1254 applied were absorbed. In rhesus monkeys, between 15 percent and 34 percent of the AR 1242 was absorbed. This difference is a function of the quantity of PCBs per unit of surface.

- Washing the skin immediately after the application removed 59 percent of the AR 1242.

- Washing the skin 24 hours after the application had virtually no effect: it removed about 1 percent of the AR 1242 and 20 percent of the AR 1254.

These observations reveal the importance of preventive measures to avoid absorption by contact. This applies to almost pure PCB compounds. However, it should be mentioned that the absorption of PCBs by contact with oil contaminated with PCBs will certainly not be as extensive. The affinity of PCBs for oil will limit such absorption except when the oil itself is absorbed; however, because of its chemical structure (chains of saturated hydrocarbons), oil can hardly be absorbed by the skin.

10.2 PCB Distribution

Once absorbed, PCBs enter the blood and are distributed in the most vascularized tissues, i.e. the liver, heart and muscles. They are subsequently metabolized and eliminated by the organism or redistributed in tissues, especially in lipids. Generally, PCBs with low chlorine content are quickly metabolized and eliminated, while those with high chlorine content (tetra- to octa-) tend to persist longer in the organism. This has been confirmed by animal studies (Matthews et al., 1979) and clinical studies (Chen 1982 and 1985; Hara, 1985, and so on). Shortly after occupational exposure, mono-, di- and tri-CBs are eliminated, while hexa- and hepta-CBs persist for a long time.

The following table shows the distribution of PCBs in the tissues of a Taiwan patient who died in an accident two months after he was diagnosed as having Yu-Cheng disease. The highest concentrations were found in lipids in the intestines, bronchia and heart, while the lowest were in the brain and spleen.

TABLE 18: Concentration of PCBs in the Tissues of a Patient Suffering from Yu-Cheng Disease

Tissue	PCBs (ppm)
Intestinal lipids	10.81
Bronchi	5.56
Large intestine	3.28
Heart	2.48
Stomach	0.66
Liver	0.65
Jejunum	0.27
Kidney	0.25
Lung	0,18
Brain	0.08
Spleen	0.07

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10.3 Elimination of PCBs in the Blood

Hara (1985) followed the PCB levels in the blood of workers exposed to the substances in a capacitor factory in Japan. He took annual blood samples for seven years after the plant closed. One group of workers had been exposed to Kaneclor 300 (43 percent chlorine), while another had been exposed to both Kaneclor 300 and Kaneclor 500 (55 percent chlorine). Kaneclor 300 contains, above all, isomers of tri- and tetra-CB, while Kaneclor 500 mainly contains isomers of penta- and hexa-CB. The results are outlined below.

In workers exposed to Kaneclor 300 and 500, the PCB level in the blood decreased an average of 27.5 percent over seven years; during the same period, it fell 61.1 percent among those exposed to Kaneclor 300 alone.

Table 19, taken from a study by Chen et al. in 1985 to assess the rate of elimination of PCBs in the Taiwan patients, indicates the differences in the rate of elimination of various congeners identified in patients' blood.

An analysis of this table reveals that not only do PCBs with high chlorine content (hexa-CB) tend to persist longer, but that the position of the chlorine atoms plays an important role. It is even possible to identify the structural properties which promote or delay elimination. For example, the molecules without chlorine in the adjacent meta- and ortho- positions (positions 2,3 or 2',3' or 5,6 or 5',6'), but with chlorine atoms in positions 4 and 4', are eliminated more slowly.

In the same study, using preliminary data, Chen was able to extrapolate the half-life of the two penta-CBs which had the highest concentrations in the poisoned patients, as Table 20 indicates. He also noted that the elimination time for the same congener varied greatly from one person to another.

TABLE 19: Comparative Rate of Elimination of the Principal PCBs in the Blood of Taiwanese Patients (cf. Chen et al., 1985)

Peak No	Chlorine Substitution	Elimination Rate	Adjacent Unsubstituted Carbon Atoms
9-4-a	2,4,5,4' (?)	+++	2',3';5',6'
9-4-b	2,5,3',4'	+++++	3,4;5',6'
9-4-c	2,4,3',4'	++++	5,6;5',6'
10-4	2,3,3',4' (?)	+++++	4,5,6;5',6'
11-5-a	2,5,2',3',5'	++++	3,4
11-5-b	2,5,2',4',5'	++++	3,4
11-5-c	2,4,2',4',5'	++	5,6
12-5-a	2,3,2',4',5'	+++++	4,5,6
12-5-b	2,5,2',3',4'	+++++	3,4;5',6'
12-5-c	2,4,2',3',4'	++++ (?)	5,6;5',6'
13-5	2,3,6,3',4'	+++++	4,5;5',6'
15-5	2,4,5,3',4'	+++	5',6'
16-5	2,3,4,3',4'	+++	5,6;5',6'
16-6	2,4,5,2',4',5'	+	none
18-6	2,3,4,2',4',5'	+	5,6
19-6	2,3,4,2',3',4'	++	5,6;5',6'
20-6	2,3,4,5,3',4'	+	5',6'
21-7	2,3,4,5,2',4',5'	+	none
22-7	2,3,4,5,2',3',4'	+	5',6'

+: extremely slow ++: very slow +++: slow ++++: less rapid
+++++: rapid

10.4 PCBs in Breast Milk and the Placenta

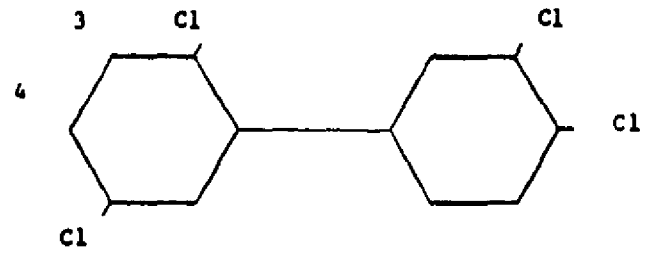
Kuwabara et al. (1978), Yakushiji et al. (1978, 1984), and Kodoma et al. (1980) have shown that mothers eliminate some of the PCBs in their tissues by breastfeeding; the PCBs are thus transferred to their babies. PCB elements in mother's milk are the same as those in the blood and adipose tissues. Hara (1985) showed that there is a linear relationship between the level of PCBs in blood and milk. The concentration in the milk was about 10 times that in the blood (for example, if the blood concentration was 10 ppb, it was 100 ppb in the mother's milk), although it was below the concentration in adipose tissues as mother's milk contains only 1 percent to 4 percent lipids (see sections 9.5.2 and 11.1.2).

The placental concentration of PCBs is about 5 to 10 times less than the blood concentration. The placental barrier appears to be effective.

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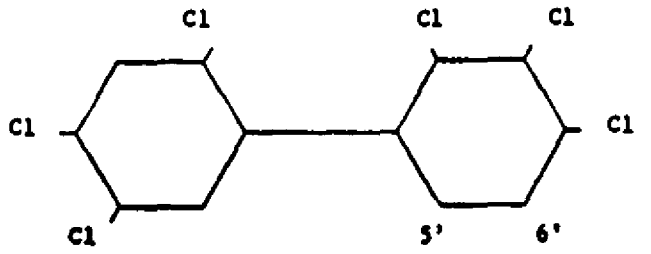
TABLE 20: Average Half-life of Two Penta-CBs

Average half-life (in months)	Penta-CB congeners
9.8 ± 5	2,4,5,3',4'
6.7 ± 2.5	2,3,4,3',4'



2,5,3',4'

Rapid Elimination
(positions 3,4 free)



2.4,5,2',3',4'

Slow Elimination
(positions 5',6' free)

FIGURE 22: Examples of Rapidly and Slowly Eliminated PCBs

11. PCBs AND HUMAN TOXICITY

At present, fish is the principal source of PCBs absorbed by the population at large. However, as PCBs were used in open systems, such as paints, solvents, inks and so on, until 1976, it is not an exaggeration to state that we have all been exposed to the substances to a greater or lesser degree.

Exposure can occur in three ways:

. environmental exposure through surroundings, air, water and food;

. accidental exposure in the event of specific contamination accidents, as was the case in Japan, or accidental dispersal in drinking water supplies and so on;

. occupational exposure occurs among workers who handle equipment containing PCB-based compounds.

11.1 PCB Levels in the Population and Effects on Health

11.1.1 PCB levels in the blood

According to data from the Centre for Disease Control, the average level of PCBs in the blood in the United States is about 5 ng/mL to 7 ng/mL (ppb). However, some people have higher PCB levels without having experienced occupational exposure to the substances.

Kreiss et al. (1982) evaluated the PCB level in the blood of 458 residents of Triana, Alabama, a fishing village. The average level varied between 3.2 ppb and 157.9 ppb, with an average of 17.2 ppb. Within the group:

98 people (21.4 percent) had a level greater than 30 ppb;
60 people (13.1 percent) had a level greater than 40 ppb;
41 people (9.0 percent) had a level greater than 50 ppb; and
 7 people (1.5 percent) had a level greater than 100 ppb.

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Medical tests have shown an association between PCB levels and the following parameters: γ GT, cholesterol rate, and diastolic blood pressure. However, it should be noted that some of the people had low blood pressure and high PCB levels and vice versa.

In Canada, we have no statistical data about the level of PCBs in human blood. A limited study suggests that PCB levels in the blood of Hydro-Québec office employees and manual workers are more or less the same as those found in the general population throughout Canada and the United States.

11.1.2 PCB levels in mother's milk and effects on health

Wickizer et al. (United States, 1981) examined samples of mother's milk collected from 1057 breastfeeding mothers.* All of the samples contained PCB residues, varying from traces to concentrations of 5.1 mg of PCBs/kg of lipids found in mother's milk, with an average of 1.5 mg of PCBs/kg of lipids (1.5 ppm).

Of this number, 49 percent had concentrations between 1 mg/kg and 2 mg/kg of lipids; 17.4 percent, between 2 mg/kg and 3 mg/kg of lipids; and 6.4 percent, more than 3 mg/kg of lipids. The average lipidic content of the milk was 34.8 g/L (3.48 percent); consequently, an average of 1.5 mg of PCBs/kg of lipids is equivalent to 52.5 μ g of PCBs per litre of milk. In the first eight months of their lives, the children consumed an average of 9.4 mg of PCBs, of which 8 mg remained in the organism. Wickizer et al. concluded that in 1981, the milk of about 50 percent of mothers in Michigan had a PCB concentration higher than the limit allowed by the Food and Drug Administration for cow's milk intended for drinking (1.5 mg/kg based on lipidic content). However, a group of experts under the direction of B.W. Weil recommended to Michigan women who had not experienced occupational exposure to PCBs to continue breastfeeding. A study conducted by Jacobson et al. (1983) among 313 newborn babies and their

* This study was carried out in Michigan. Samples were collected in three Grand Rapids hospitals and in one in Muskegon (located on Lake Michigan). Grand Rapids is the most important metropolitan region bordering on Lake Michigan and is located near the Grand River, which is very popular for salmon and trout fishing.

mothers in the region studied by Wickizer (1981) did not reveal any effects among the babies or their mothers.

In Canada (Atkinson, 1979), a reduction in the average PCB level in mother's milk among Ontario women was noted: it fell by a factor of 2, from 40 µ g/L between 1970 and 1974 to 20 µ g/L in 1978. No toxic effect resulting from PCBs among newborn babies has been reported in Ontario in the past.

Both studies show that in 1978 the PCB level in mother's milk in Ontario (20 µ g/L) was 2.5 times lower than the average rate observed in 1981 in Michigan women (52.5 µ g/L).

In light of the analysis on the degradation of PCBs (see section 7.7), it may be expected that the average PCB level among Canadians will be lower still in 1986.

11.1.3 PCBs and lipids

According to studies conducted by the Centre for Disease Control in the United States and Environment Canada (1976), the average PCB level in the lipids of Americans and Canadians is between 0.1 ppm and 1 ppm (1 mg/kg of fat). Some people, especially heavy fish eaters living in the most contaminated regions, may have lipidic concentrations of PCBs exceeding 10 ppm. Generally, it is thought that this concentration is, on average, between 150 and 200 times the concentration of PCBs in the blood.

11.1.4 Effects on health in the population

The PCB levels of people who have not experienced accidental or occupational exposure to the substances does not seem to cause serious health problems, even in those groups most likely to be contaminated, such as fishermen.

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11.2 The Yusho and Yu-Cheng Poisonings

11.2.1 Description

The first massive PCB poisoning occured in 1968 in Japan, where more than 1780 people, mainly residents of Fukuoko and Nagasaki, were poisoned after consuming rice oil contaminated by PCBs, furans and queterphenyls for six months. The Japenese called the resulting illness Yusho (''rice oil'') disesse. The morbidity rate is not known. The oil contained more than 2000 ppm of Kaneclor 400; it is thought thet the consumption of PCBs varied between 0.5 g and 2 g, and that of furane, from 2.4 mg to 7 mg.

Eleven years later, the same type of poisoning occurred in Taiwan, where the epidemic affected tha cities of Tai-Chung, Hsincho, Miaoli and Chang-Hua. According to the records of the Taiwan provincial health department, 2061 cases were reported. Although victims displayed the same symptoms as those suffering from Yusho disease, the Taiwanese called it Yu-Cheng disease. The rice oil in Taiwan contained Kaneclor 400 and Kaneclor 500, furans and quaterphenyls; more or less the same quantities of PCBs and furans were consumed in Taiwan and Japan. In the cities mentioned, the morbidity rate per 100 000 inhabitants varied between 56.56 and 1348.81. Symptoms usually appeared bstween 1.5 months and more than 6 months after ingestion began, with an avarage of 3 or 4 months.

With respect to human toxicity, these two events have contributed the most to furthering understanding of the effects of PCBs and their contaminants, mainly dibenzofurans and quaterphenyls.

Between 1968 and 1973, the official diagnosis of Yusho disease focused almost exclusively on symptoms involving the mucose membranes and skin. In addition to such symptoms (by far the most common), patients complained of fatigue, loss of appetite, headaches, weakness and edemas of tha extremities. Nausea and vomiting were also reported; at the time, they were considered minor complications. Laboratory findings also showed such anomalies as increased triglycerides, anemia, lymphocytosis, hypoalbuminamia and reduced neuro-sansorial conducting speed.

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It was only five years later, in 1973, that PCBs were identified in the blood samples of patients suffering from Yusho disease, at very low concentrations (5.9 ± 4.5 ppb and 6.7 ± 5.3 ppb on average). These concentrations in the blood were much lower than those measured later in workers exposed to PCBs in transformer and capacitor factories. Indeed, we now know that such concentrations were very high, reaching as much as 1319 ppb (Maroni, 1981, Italy, concentration between 88 ppb and 1319 ppb; Wolff, 1982, United States, 1 ppb to 546 ppb; Hara, 1985, Japan, 121 ± 76 ppb). However, these symptoms of Yusho disease were much more serious than those displayed by industry workers.

Later, with more sophisticated analysis methods, it was possible to measure furans and quaterphenyls in the contaminated oil; concentrations of these substances were by no means negligible, which raised a number of questions.

. Were the symptoms of Yusho disease actually caused by PCBs? Might not dibenzofurans be the principal cause?

. What are the real hazards of PCBs?

The Taiwan poisoning provided the answers to these questions, as both accidents are similar: the oils were contaminated with PCBs, furans and quaterphenyls in both instances and the quantity of these contaminants ingested was also the same.

Less than a year after the symptoms appeared, the PCB level in the blood of Yu-Cheng patients varied from 3 ppb to 1156 ppb, with an average of 99 ppb. Although these levels are equivalent to those in workers experiencing occupational exposure, the severity and persistence of the symptoms of Yu-Cheng disease were considerably more pronounced.

After 1979, polychlorinated dibenzofurans were identified with certainty in the blood of patients suffering from Yu-Cheng disease and in the tissues of those with Yusho and Yu-Cheng diseases. Some of these substances, such as 2,3,4,7,8-pentachloro-DF, are deemed to be highly toxic. It has been shown that this furan accumulates in the livers of humans, monkeys and rats and is very toxic to animals (for example, it causes atrophy of the thymus, weight loss and hypertrophy of the liver with marked enzymatic induction at low doses, i.e, between 1 µ g/kg and 10 µ g/kg). On the basis of epidemiological studies conducted in Japan and Taiwan, the average consumption of PCDFs has been estimated at about 3,4 mg, which corresponds to 68 µ g/kg of body weight for a person weighing 50 kg.

These data, supported by animal experiments, have led the scientific community to conclude that most of the effects observed at the time of both poisonings were attributable primarily to the ingestion of polychlorinated dibenzofurans.

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11.2.2 Symptoms observed in patients with Yusho and Yu-Cheng diseases

Peri-ocular signs (earliest symptoms of Yu-Cheng disease, at an average plasmatic concentration of 41 ppb of PCBs).

- accentuated conjunctive pigmentation;
- abnormal cyst formations in meibomien glands;
- secretions;
- eyelid edema.

Cutaneous lesions (mild to severe)

- grade I: circumscribed pigmentation;
- grade II: comedones;
- grade III: mild acne eruptions;
- grade IV: extensive distribution of acne eruptions with purulent infections.

These symptoms regressed in time, but in some patients suffering from Yusho disease, they persisted after more than 10 years.

Hepatic lesions

These lesions ranged from the disturbance of liver parameters (such as γ GT, SGOT and SGPT) with no clinical signs, to hepatomegaly and jaundice.

General impairment

Weakness and fatigue.

Rise in triglyceride and cholesterol rates

Neurological lesions

At the onset of the disease, some patients displayed decreased peripheral sensitivity.

Effects on reproduction

- no teratological effect;
- lower average birth weight;
- some babies born with hyperpigmentation and chloracne.

These symptoms disappeared after two or three years.

Effects in nursing babies

Some nursing babies whose mothers were poisoned displayed symptoms of Yusho disease, with the appearance of dermatological problems such as chloracne. The symptoms disappeared after two or three years.

Immunological lesions (studied in Taiwan)

- increased IgM and IgA;
- normal IgG;
- reduction of T cells, active T cells and helper T cells;
- increased cutaneous and respiratory infections.

Three years later, a marked improvement in these immunological parameters and a drop in the infection rate were noted.

11.3 Occupational Exposure and Effects on Health

11.3.1 Fischbein et al. (1979)

This clinical study of 326 workers exposed to PCBs showed a correlation between the level of exposure, dermatological problems and SGPT level. Other objective and subjective symptoms and current biological blood parameters were not linked to PCBs.

11.3.2 Maroni (1981)

The study of 80 workers in a capacitor factory revealed that the PCB level in their blood varied between 41 ppb and 1319 ppb. Fifteen of them had cutaneous diseases, including four cases of chloracne. An association was also noted between PCB levels and the increase of γ GT, SGOT and SGPT.

11.3.3 Smith (1982)

This study, conducted for the National Institute of Occupational Safety and Health (NIOSH) in three groups of Monsanto workers, revealed:

- a positive correlation between exposure to PCBs and subjective symptoms of skin and mucous membranes irritations and systemic illnesses. No clinical anomaly attributable to PCBs was observed;

- a positive correlation between exposure to PCBs and SGOT, γ GT and plasma triglycerides;

- an inversely proportional correlation with HDL-cholesterol.

11.3.4 Colombi et al. (1982)

This study showed a slight prophyrinogenic effect in workers at a capacitor factory by measuring an increase in total urinary porphyrine. However, the porphryia did not reach the stage of clinical signs.

11.3.5 Kuwabara et al. (1978)

The study of PCB levels in the blood of women exposed to the substances in the workplace and in their children from 0 to 13 years old made it possible to evaluate the effects of such exposure on their health. It was observed that the PCB level in the blood of these women and their children was higher than among patients suffering from Yusho disease and that levels measured in the children increased with the duration of the lactation period. None of the children displayed signs of PCB poisoning.

TABLE 21: Concentration in the Blood in u g/L (ppb) in Women Exposed in the Workplace, Their Children, Japanese Patients and Non-exposed Women

	EXPOSED WOMEN	CHILDREN OF EXPOSED WOMEN	JAPANESE PATIENTS	NON-EXPOSED WOMEN
SAMPLE	n = 20	n = 39	n = 12	n = 28
RANGE	8.3 to 64.5	0.8 to 93.2	1.8 to 8.6	0.8 to 5.7
AVERAGE ± STANDARD DEVIATION	38.6 ± 21.5	14.8 ± 18.1	4.2 ± 1.9	2.6 ± 1.2

11.3.6 Sahl (1985)

Only one study has been published on the potential effects of PCBs among electrical company workers. It was conducted recently at Southern California Edison (Sahl, October 1985, International Symposium on PCBs, Seattle). It centred on 220 employees in positions with high potential for exposure between 1979 and 1984 (maintenance workers and installers), who were compared with a group of office workers not subject to risks of exposure and a group of new employees who underwent a pre-hiring examination.

The length of exposure in the group of high-risk workers varied from 1 to 37 years, with an average of 14 years. The PCB level in their blood varied from 1 ppb to 15 ppb, with an average of 3 ppb and a standard deviation of about 4 ppb. These levels correspond to those measured among 738 new employees and the 1058 employees already working for the utility, whose PCB levels displayed the same proportions (average: 5 ppb; standard deviation: 4 ppb). Thus, there was no difference in the PCB level in the blood of the three groups studied.

Sahl concluded that working for this firm did not result in greater accumulation of PCBs than in the population at large. According to him, at such levels, nothing indicated that there were health risks for workers as other studies in the industrial milieu had not showed significant effects on the health of workers whose blood contained much higher PCB levels, i.e. between 50 ppb and 1300 ppb.

11.4 Remarks on Health Risks in the Workplace

11.4.1 No study in the workplace has dealt with the effects of PCBs on the reproductive system; consequently, we cannot conclude that no such effects exist. On the other hand, Kuwabara did not note any health problems in the children of women who gave birth after exposure in the workplace.

11.4.2 Although the concentration of PCBs in the blood of workers is higher than the rates observed in Japan and Taiwan, it has been noted that the health problems of workers are much less pronounced. With the exception of mild cutaneous disorders and variations in hepatic parameters without symptoms, no clinical signs have been associated with PCBs found in workers.

11.4.3 Researchers have noted that, once exposure to PCBs was halted, signs and symptoms were quickly reversible (after less than one year), while symptoms persisted in victims of Yusho disease even after 10 years. They explain this phenomenon by the presence of PCDFs in the tissues of Japanese patients; this shows that PCDFs continue to produce toxic effects beyond a 10-year period.

11.4.4 As Yusho and Yu-Cheng diseases are attributable primarily to polychlorodibenzofurans, we now have a reference point on the quantities of furans which can produce toxic effects in humans. Between 2.4 mg and 7 mg of total PCDFs were sufficient to produce the signs and symptoms observed at the time of both massive poisonings.

11.4.5 According to the NIOSH study (Criteria for a recommended standard occupational exposure to PCB's, 1977), chloracne usually appears in exposed workers having blood PCB levels above 200 ppb. Chloracne seems to be the first symptom to appear in workers in the electrical equipment industry.

11.5 PCBs and Cancer in Humans

Only three epidemiological studies have been conducted until now to determine whether PCBs cause cancer.

11.5.1 Brown (1981)

In 1977, at the request of NIOSH, Brown (1981) carried out a retrospective study of mortality among 2567 workers in two PCB manufacturing plants in the United States.

There were relatively few deaths (163), which seriously limits the study's statistical value. Moreover, follow-up lasted only 15 years. A latency period of between 20 and 30 years is often necessary to observe a fatal cancer. More than 50 percent of the sample had been exposed for two years or less.

Greater mortality levels than anticipated were noted for rectal cancer (4 cases observed for 1.19 expected) and liver cancer (3 cases observed for 1.07 expected) was noted. However, if both populations are combined, no excess is statistically significant (probability less than 0.05). The study continues, although no other results have been published since 1981.

11.5.2 Behn et al. (1976)

In the course of this study of a group of 72 workers in a petrochemical plant in the northeastern United States, three cases of melanomas were found, two in a group of 31 men who had handled AR 1254 in the past and were extensively exposed, and the third in a group of 41 R&D employees, less exposed to PCBs. In the first group, the incidence was significantly higher than expected: 0.06 cases observed for 0.04 expected, with probability less than 0.001.

However, the sample studied was relatively small and the workers had also been exposed to other chemical products and to the sun. We know that prolonged exposure to the sun can cause melanomas.

11.5.3 Bertazzi et al. (1981)

This retrospective study of mortality covered 1310 employees (290 men and 1020 women) who had worked for at least six months in a capacitor factory in Italy between 1946 and 1970. The researchers found 8 cancer deaths among the men (4558 person-years) for 3.3 expected with p = 0.04. The excess was attributable primarily to cancer of the digestive tract and the lymphatic and hematopoietic system (ganglions and blood). Six cancers (for 2.3 expected, p not significant) were observed in the women (16 088 person-years), including two cancers of the lymphatic and hematopoietic system. Bertazzi concluded that the limited number of deaths and the fairly low average age of the population made it difficult to draw a valid conclusion. This study continues; to our knowledge, no other data have been published.

11.6 Remarks on PCBs and Cancer in Humans

11.6.1 An epidemiological study of cancer can be defined as a statistical study of the health of populations which consists of evaluating and comparing the frequency of various cancers in populations with different characteristics such, for example, as exposure or non exposure to PCBs. Dool (1981) and the International Agency for Research on Cancer (IARC, 1980) have defined criteria to determine the validity of the results obtained. The main criteria are:

- The excess of cancer observed in the exposed group must be higher than what is statistically probable;
- it must be possible to establish a relationship with dose and duration of exposure;
- there must be no biases or confusing factors;
- the association between exposure and cancer must be observed several times under different circumstances.

By applying these criteria to the studies mentioned, we find that data available until now and the results obtained do not allow us to conclude that PCBs have a carcinogenic potential in humans.

11.6.2 Statistically, all three studies are limited and do not enable us to establish a cause-and-effect relationship between the cancers observed and exposure to PCBs. Moreover, a number of methodological weaknesses may have led to mistakes, i.e. considerable variability in exposure time and the age of exposed persons, exposure to other products, occasionally very short latency period, information drawn from death certificates and subject to error, and so on.

11.6.3 A study which does not show a statistically significant increase in cancer is called a negative study or a test with a negative result. In light of such studies, one is tempted to conclude that the substance studied is not carcinogenic, although this conclusion is not necessarily fair. Many reasons can explain why a cancer study is negative. Often, this results from the poor sensitivity of the method or its lack of power: number of samples, exposure time and the relationship between exposure and dosage, and so on, as in the case of studies cited earlier, on the basis of which we can neither confirm nor deny a carcinogenic effect. However, given the rather high number of samples in studies conducted by Brown and Bertazzi, i.e. 2567 and 1310 workers, respectively, we can at least confirm that if PCBs have a carcinogenic potential, it appears to be relatively small.

12. APPLICATION OF STANDARDS AND REGULATIONS AND HEALTH AND ENVIRONMENTAL PROTECTION

12.1 Do PCBs Belong to the Category of Chemical Substances Which Threaten Health and the Environment?

First, let us clarify what we mean by a chemical substance which threatens health and the environment.

Direct hazard: direct contact with the product may produce acute or chronic effects in humans or animals, or have a destructive effect on wildlife.

Indirect hazard: products do not pose a direct threat, although once they are released in the environment they can, in the long run, be prejudicial to ecosystems and indirectly threaten health. Contamination by such products can reduce the number of animal or plant species, slow down important economic activities, cause irreversible ecological changes and, consequently, prevent humans from benefiting from nature.

Theoretically, the most hazardous chemical products have some of the following characteristics. They can:

. spread in very high concentrations in the environment;

. persist for a long time in the environment and accumulate there indefinitely;

. accumulate in living tissues and move through the food chain;

. cause biological or geochemical changes, even in small concentrations;

. be impossible to recover once dispersed in the environment;

. enter the environment in several ways, from many sources.

In light of the preceding chapters, it can be concluded, with some qualification, that PCBs have, in part, some of the following characteristics:

. relatively high concentrations of them have been found everywhere in the environment, as a result of uncontrolled dumping over three decades;

. some congeners with high chlorine content persist for a long time in the environment (half-life of more than two 2 years), but not indefinitely;

. they accumulate in living tissues and move through the food chain;

. they produce biological changes in the most sensitive species for concentrations in the order of mg/kg of body weight. According to laboratory data, some animal species could be affected at concentrations currently found in the environment;

. they cannot be recovered once they are dispersed in the environment;

. since their use in open syatems was halted, sources of infiltration in the environment have been appreciably reduced; PCBs tend to be eliminated slowly from the environment, as tha degradation rate seems to surpass the influx rats.

Nonetheless, we believe that all of these characteristics of the behavior of PCBs in the environment are sufficient to justify measures to control the risk of spills.

12.2 Are PCBs a Direct Threat to Health?

We have seen that PCBs have caused health problems in workers of both sexes exposed to them in industrial workplaces, especially in plants manufacturing transformers and capacitors using these substances. Such problems, affecting mainly the skin and liver, occur after the accumulation of considerable quantities of the substances in the organism when PCB concentrations in the blood and lipids reach levels generally above 200 ppb and 40 ppm, respectively. At the concentrations found in the population at large, no notable effects have been observed.

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1.2.2.1 Exposure in the workplace

12.2.1.1 Exposure to askarels

PCB-based fluids called askarels, found in electrical equipment, can be absorbed by inhalation or direct contact.

By inhalation

In Québec, the following occupational exposure standards are currently in force (Regulation respecting the quality of the work environment):

AR 1260	0.5 mg/m^3	x	[8] houre
AR 1254	0.5 mg/m^3	x	[8] hours
AR 1242	1 mg/m^3	x	[8] hours

Were they stringently applied, these standards would result in an average accumulation by inhalation of 5 mg/day for AR 1260 and AR 1254, and 10 mg/day for AR 1242. Based on all of the information we heve at our disposal concerning occupational exposure, we might well expect symptoms after 3 or 4 months' exposure. This standard seems ineffective to us, but as it will be impossible to experience such exposures in the future, it is now inapplicable.

By contact

As askarels contained in electrical equipment seem to be relatively absorbable by the skin, it is important to provide workers handling such equipment or fluids with effective skin protection.

12.2.1.2 Exposure to oils contaminated by PCBs

Is contact with contaminated oils hazardous for workers or the public, in the event of an accidental spill?

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This question is complex, as the risk depends upon the type of exposure, the quantity of PCBs in the oil, the frequency of exposure, the degree of absorption by the skin, the surface exposed and so on. A concentration of 50 ppm is often cited as a risk threshold. It should be pointed out that this concentration is the permissible limit of PCB content for fluids to be governed by the same environmental protection legislation as pure PCBs. We know that PCBs have an affinity for oil: for the 50 ppm contained in the oil to be absorbed by the skin, the oil would have to be completely absorbed. This means, for example, that a person must have daily contact with 10 mL of oil contaminated at 50 ppm; in this instance, a maximum of 50 µ g of PCBs would be absorbed, provided the 10 mL of oil were completely absorbed by the skin. However, as the skin absorbs very little of this type of oil (see section 10.1), the risk certainly does not exceed several micrograms of PCBs per day. If, in addition, workers adopt hygienic and protective measures, such as wearing gloves and washing thoroughly when oil is found on the skin, the quantity of PCBs absorbed becomes negligible.

This perhaps explains the results obtained by Sahl among workers at Southern California Edison, where he found that installers and maintenance workers exposed to PCBs for several years did not have blood levels of the substances higher than those in the population at large.

12.2.2 Exposure through food

The Legal Marketing Allowance (LMA) for food in Canada is 2 mg/kg (2 ppm).

As we noted earlier, consumers of fish are the most exposed. In Canada, those at highest risk appear to be consumers of fish caught in the Great Lakes and certain tributaries of the St. Lawrence. The average consumption of fish in Canada is 125 g per week; consequently, a person eating this quantity of fish contaminated at 2 ppm ingests an average of 250 µ g of PCBs per week.

MONS 223288

12.3 Indirect Threat to Health, Flora and Fauna

See Chapter 7.

12.4 Are Measures to Combat PCB Pollution Effective?

There is no doubt that measures taken in the early 1970s to prohibit the use in food processing plants of equipment containing PCBs and to halt the use of the substances in open circuits led to a substantial reduction in the threat to health and the environment.

In the late 1970s and in early 1985, legislation prohibiting the production of new equipment containing PCBs and improved measures designed to prevent accidental PCB spills while equipment is being transported or handled have certainly enhanced the efficacy of earlier measures.

MONS 223289

CONCLUSION

Our analysis of available data respecting, on the one hand, the toxicity of PCBs, their contaminants and combustion products in humans and, on the other, their persistence in the environment, leads us to make the following observations.

PCBs can produce toxic effects in animals and humans; these effects are closely related to the quantity of the substances absorbed by the organism. However, because of widely varying responses to PCBs and related products from one animal species to another, it seems very hard to apply the data obtained with laboratory animals to humans in order to quantify toxicological risks and risks of cancer in humans.

However, the data obtained using laboratory animals are very useful. They provide very precise information on the type of response which PCBs produce in mammals, and indications of the potential risk which some species in the environment are facing. They also explain the differences in toxicity and persistence of various commercial PCB-based compounds in humans, and shed new light on the mechanism of action and the metabolism of PCBs in living organisms.

For a long time, it was thought that PCBs were highly toxic, because of the severe clinical signs displayed in 1968 by Japanese poisoned by rice oil contaminated with PCBs and other substances which were unknown at that time (furans, quaterphenyls and methylsulphones). Through intensive follow-up among victims and studies conducted among workers exposed to PCBs in the workplace, and others carried out following the Taiwan poisoning in 1979, the scientific community concluded that the seriousness of the symptoms of Yusho disease were attributable primarily to furans found in the rice oil and not to PCBs. This conclusion is supported by a considerable body of research on humans and laboratory animals.

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When comparing the symptoms of industry workers among whom plasma and lipid concentrations of PCBs were equal to or higher than those measured in victims of Yusho and Yu-Cheng diseases, researchers noted marked differences. In populations exposed to PCBs in the workplace, the substances have caused a number of cases of chloracne, other types of dermatosis and a slight alteration of hepatic and lipidic functions, although these signs disappeared several months after exposure ceased. Among patients suffering from Yusho and Yu-Cheng diseases, the symptoms were generally much more severe and persisted in some of them 15 years later. Recent research has shown that these effects are due to the presence of furans in the patients' tissues.

At the concentrations usually observed, no toxic effect has been noted in the population at large. Even in populations of fishermen who consume substantial quantities of contaminated fish, no important effects due to PCBs were observed. It should be noted that in some fishermen, plasma concentrations of PCBs were higher than the average concentration measured in patients in Japan and Taiwan.

With regard to potential effects on reproduction, no laboratory study has shown that PCBs have a terstogenic effect, i.e. they do not appear to cause deformities. However, in laboratory animals there is a threshold beyond which the substances are fetotoxic (increased abortions, reduced birth weight) and probably have a contraceptive effect (reduced fertility rate).

It may be thought that such effects might occur in all living organisms. However, the absence of documentation on women exposed in the workplace to high doses of PCBs during the 1950s, 1960s and 1970s prevents us from estimating the exact dose at which such problems would occur.

At present, one source of anxiety in the population at large is the potential effect of PCBs in mother's milk. Studies have been conducted in Japan among children between 0 and 13, who were born to mothers exposed to PCBs in the industry and breast fed, and in the United States and Ontario in the Great Lakes region, among women and their nursing babies. These studies did not

reveal any effects among these babies during breast feeding and childhood. American specialists have concluded that women should not avoid breast feeding despite the presence of traces of PCBs in mother's milk.

Data on the incidence of neoplastic diseases in populations exposed to PCBs do not enable us to establish a cause-and-effect relationship. Although the data do not entirely exclude the possibility that PCBs are potentially carcinogenic, we can conclude that this potential is certainly very limited. In our opinion, to significantly confirm or deny a cause-and-effect relationship, studies would have to deal with much broader population samples that those in studies conducted until now, for example, by grouping together several exposed populations. However, such studies seem hard to conduct as there are no longer any highly exposed populations in the industry since the manufacture of PCBs has been prohibited.

The greatest health risk is fire in equipment insulated with PCBs (askarels), incomplete combustion of which leads to the formation of substances deemed to be extremely toxic, primarily furans and, where there is an associated solvent (polychlorobenzenes), dioxins. It is, therefore, essential that preventive measures be taken to reduce these risks to a minimum in the event of a fire. We feel that risk is higher in workers responsible for decontaminating and rebuilding sites than it is in firefighters. For the latter, a positive-pressure breathing apparatus, gloves and a coverall make health risks negligible.

Exceptional protective measures must be taken among workers responsible for decontaminating and rebuilding sites, to prevent effects similar to those produced by Yusho disease. After the fire, care must be taken to ensure that only authorized personnel enter the site of the fire and only after experts have advised them on protective measures. Obviously, high priority should be accorded to replacing electrical equipment insulated with askarel in zones where contamination risks are high.

We have also shown that in the event of a fire in equipment insulated with oil contaminated by small concentrations of PCBs (several hundred ppm), firefighters face very limited risks, even without protective equipment; if they wear positive-pressure breathing apparatus and coveralls, risk is virtually eliminated. Of course, for greater safety, we recommend that such measures be taken.

Before the site is cleaned up, an assessment must be carried out to determine the concentrations of toxic products present and establish what measures are needed to decontaminate it.

Protective measures must be adopted for industry workers called upon to handle, maintain or transport equipment containing askarels, to avoid skin contact with PCBs. Where temperatures are high, for example, or ventilation is poor, it may be necessary to wear a self-contained, positive-pressure breathing apparatus. To avoid absorption of PCBs through the skin, splashes must be immediately and thoroughly washed with soap and water. The risk of absorbing contaminated oil by contact is much more limited than with askarel, because of the small concentration of PCBs in the oil and the low absorption rate by the skin of the type of oil used in such applications.

The environment is widely contaminated and such contamination reaches the trophic food chain of humans. All living organisms carry traces of PCBs. In some ecosystems, contamination is such that over time, some species could very well have disappeared had not the uncontrolled dumping of PCBs in the environment been halted.

Studies carried out in the Great Lakes since 1970 tend to show that, since the production and sale of PCB-based compounds for open-circuit applications were halted in 1973, the quantity of PCBs which are eliminated in the environment exceeds new influxes. Researchers have noted a significant reduction in concentrations of PCBs in sediments, water, the air and in fish and birds.

Although production of PCBs has been halted throughout the world, because there are still large quantities of the substances in use in electrical equipment, it remains essential to maintain measures governing dumping, through legislation and regulations. Such measures will accelerate the process of eliminating PCBs from the environment, which has already begun.

Current knowledge on the risks of toxicity and the general behavior of PCBs in the environment allows us to adopt a rational approach to effectively combat PCB pollution, by reducing health and environmental risks, while taking into account the socio-economic impact of the measures adopted, and their applicability.

In our view, to fully eliminate PCBs from the Canadian environment, we will have to install PCB destruction units like those in operation in the United States and Europe. Storing electrical equipment containing PCBs eliminates some risks, such as fire, although the latter risk still exists when this type of equipment is used in an electrical system. On the other hand, storage does not protect us from catastrophes such as earthquakes, which would cause tons of PCBs to be released in one place.

We can find a solution to any pervasive problem which threatens health and the environment. As world-wide production of PCBs has stopped, and as the substances are gradually biodegrading and governments are taking measures to control PCB dumping in the environment, we can hope that within several decades they will no longer pose their current threat.

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