COM-72-10419

Polychlorinated Biphenyls and the Environment

Interdepartmental Task Force on PCBs
Washington D.C.

May 1972

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BIBLIOGRAPHIC DATA SHEET	1. Report No. ITF-PCB-72-1	2.	3. Recipient's Accession No. COM-72-10119
4. Title and Subtitle PCBS AND THE ENVIRONMENT			5. Report Date 20 Mar. 72 6.
7. Author(s)			8. Performing Organization Rept. No.
9. Performing Organization Name and Address Interdepartmental Task Force On PCBs			10. Project/Task/Work Unit No. 11. Contract/Grant No.
12. Sponsoring Organization Name and Address Departments of Agriculture, Commerce, Health, Education, and Welfare, and Interior; and Environmental Protection Agency; plus other participating agencies.			13. Type of Report & Period Covered Final 14.

15. Supplementary Notes

16. Abstracts

This report is the product of a six month review of the chemicals known as PCBs--polychlorinated biphenyls--by five Federal agencies, with participation by other agencies. The Interdepartmental Task Force on PCBs had as its goal the cobrdination of the scientific efforts of the Government aimed at understanding PCBs and the strengthening of the Government's ability to protect the public from actual or potential hazards associated with them. The task force made nine findings, conclusions, and recommendations, primarily pointing out that PCBs should be restricted to essential or nonreplaceable uses which would minimize the likelihood of human exposure or leakage to the environment. Supplementing the 20-page report are eight appendices detailing current knowledge about various aspects of PCBs, including their use and replaceability; occurrence, transfer, and cycling in the environment; occurrence and sources in food; and PCBs effects on man and animals.

17. Key Words and Document Analysis. 17a. Descriptors

*Pollution,
*Chlorine aromatic compounds,
*Biphenyl/chloro,
Environmental surveys,
Insulating oil,
Economic factors,
Government policies,
Toxicology,
Public Health.

17b. Identifiers/Open-Ended Terms

17c. COSATI Field/Group 13B, 6T

18. Availability Statement	19. Security Class (This Report) UNCLASSIFIED	21. No. of Pages 192
	20. Security Class (This Page UNCLASSIFIED	22. Price PC $6.00

FORM NTIS-35 (REV. 3-72) USCOMM-DC 14952-P72

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PREFACE

On September 1, 1971, representatives of several agencies of the Federal Government established an interdepartmental task force to coordinate the scientific efforts of the Government aimed at understanding the family of chemical compounds known as polychlorinated biphenyls (PCBs), and to strengthen the Government's ability to protect the public from actual or potential hazards from PCBs. On September 5 it was announced that the task force would "coordinate a government-wide investigation into PCB contamination of food and other products". On September 13 the task force, made up of qualified specialists from a range of disciplines, held the first of a series of meetings. Appropriate spokesmen on various problems associated with PCBs were assigned to prepare a series of background papers, drawing on the resources of their own and other agencies.

The task force included operating units of five Executive Branch departments: Department of Agriculture; Department of Commerce (Assistant Secretary for Science and Technology and National Oceanic and Atmospheric Administration); Environmental Protection Agency; Department of Health, Education, and Welfare (Food and Drug Administration and National Institute of Environmental Health Sciences of the National Institutes of Health); and Department of the Interior (Bureau of Sport Fisheries and Wildlife).

The report which follows represents the results of the task force's review and reflects the position of the operating agencies of the Federal Government which have major responsibilities concerning such chemicals as PCBs in food and in the environment. The task force had the advantage of some additional sources of information and review on PCBs. For example, during the course of the study, the National Institute of Environmental Health Sciences sponsored an international scientific meeting on PCBs on December 20-21, 1971, at the Quail Roost Conference Center, Rougemont, North Carolina. One hundred persons--from Government, universities, industry, and the press--attended. The proceedings of this conference soon will be published by the Institute. The task force also met from time to time with a group of scientific advisors from outside the Federal Government, which was already at work prior to September 1971 examining a number of hazardous trace substances, one of which was PCBs.

The individuals who served on the task force included: Dr. John E. Spaulding and Dr. Harry W. Hays (Department of Agriculture), Dr. Robert W. Cairns and Dr. William Aron (Department of Commerce), Dr. John Buckley (Environmental Protection Agency), Dr. Lawrence Fishbein, John R. Wessel, and Dr. Albert Kolbye (Department of Health, Education, and Welfare), Dr. Lucille Stickel (Department of the Interior), Dr. Edward J. Burger, Jr. (Office of Science and Technology), and Dr. Terry Davies (Council on Environmental Quality). Many others participated in some of the meetings and lent assistance in a variety of ways including authorship of background papers published as appendices in this report. The task force is grateful for this assistance.

The task force will continue to assess new information that comes to its attention.

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FINDINGS, CONCLUSIONS, AND RECOMMENDATIONS

Polychlorinated biphenyls (PCBs) have been used in the United States and elsewhere over the past 40 years, for many industrial and consumer applications. During the past three years evidence has accumulated to indicate that PCBs are widely dispersed throughout the environment and that they can have adverse ecological and toxicological effects.

The principal uses for PCB fluids are in the electrical industry. PCBs have superior cooling, insulating, and dielectric properties and hence are widely used in various electrical devices. Transformers and capacitors filled with PCBs can be used in inside locations where failures of oil-insulated equipment would present a potential danger to life and property. Because PCBs are relatively nonflammable, apparatus containing them is essentially free from the fire and explosion hazards associated with oil-insulated and oil-cooled electric devices. Stability at high temperatures is another major factor in the attractiveness of these compounds. The principal advantage of PCBs over substitutes is the relative freedom from flammability in some applications that previously had been plagued by serious fires. PCBs also give electrical equipment the critical advantages of reliability, long life, and compactness. PCB impregnated capacitors, for example, are markedly more reliable and long-lived, and 1/6 the size, 1/5 the weight, and 1/4 the cost of comparable oil impregnated capacitors. Small capacitors with PCBs have a use-life expectancy of 10 to 15 years, and large capacitors 20 to 25 years. PCBs in transformers are replaced only every 25 to 30 years.

PCBs have been discovered to have a widespread distribution in the environment, and some environmental occurrences have been associated with adverse effects on certain forms of animal life. Beginning in 1971, the Monsanto Company, the sole U. S. producer, has reported taking voluntary actions to reduce the volume of PCB production and to limit its distribution to industries concerned with the manufacture of electrical apparatus. Similar restrictions have been put into effect by statute in Sweden and voluntarily in Great Britain.

A large use of PCBs had been in carbonless duplicating paper. This use has been discontinued. The Food and Drug Administration and the food industry have increased their surveillance to assure that PCBs are not used in food plants, products, or packaging.

The task force has reviewed all of the available scientific information on various aspects of the PCB problem. It has found much data that it regards as inadequate and many questions that remain unanswered. But on the basis of available information, the task force concurs on the following findings, conclusions, and recommendations:

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1. PCBs should be restricted to essential or non-replaceable uses which involve minimal direct human exposure since they can have adverse effects on human health. There currently are no toxicological or ecological data available to indicate that the levels of PCBs currently known to be in the environment constitute a threat to human health, but additional experiments are underway to evaluate the impact of low level, long-term exposure to PCBs.

2. PCBs have been used so widely over such a long period that they are ubiquitous. Even a total cessation of manufacturing and use of PCBs would not result in the rapid disappearance of the material, and ultimate disappearance from the environment will take many years. The elimination of non-essential uses and prohibition of discharges from essential uses will result in gradual elimination from the environment.

3. PCBs were first identified as potential food contaminants in 1966. Three principal sources or routes of contamination of food have been identified. General environmental contamination has resulted in PCB residues in some fresh water fish. Prohibition of PCB discharges into water will result in the reduction of such residues. Another route occurs from the presence in food packaging materials of PCB residues, some of which migrate into packaged food. The FDA has proposed regulations for food packaging materials and foods to deal with this problem. The third route involves accidental contamination of food from leakage or spillage of PCBs into feed or directly into food. The dietary intake of PCBs is of low order and does not present an imminent health hazard. To date, all of the high levels of PCBs encountered in human or animal foods have been associated with accidents, for which Government agencies have exercised necessary regulation and control to minimize the distribution of contaminated foods.

4. The sole domestic producer of PCBs, Government agencies, and key user industries are taking appropriate steps to cut off further introduction of PCBs into the food supply and to reduce the current levels of PCBs as food and environmental contaminants. The Food and Drug Administration (FDA) has acted, under the authority of the Food, Drug, and Cosmetic Act, to preclude the accidental PCB contamination of food. It has also proposed a prohibition on the use in food packaging materials of pulp from reclaimed and salvaged fibers that contain poisonous or deleterious substances that may migrate into the food if the contamination by such substances is deliberate or avoidable. It has proposed temporary tolerances for unavoidable PCB residues in food packaging materials and in certain foods. The Department of Agriculture has acted under the Wholesome Poultry Act and other statutes to prevent accidentally contaminated foods from reaching the market.

The major gap in the regulatory system to deal with PCBs is the absence of any broad Federal authority to restrict use or distribution of the chemical, to control imports, and to collect certain types of information. The task force believes that such authority is needed. This authority would be provided by the Toxic Substances Control Act proposed by the Administration and now pending before Congress.

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5. Housekeeping is particularly important in the manufacture, use, and disposal of PCBs. Under a program of limitation on the sale of PCBs, the electrical industry will continue to be the principal user of PCBs; it, as well as industries now holding inventories of PCBs, have a special responsibility for monitoring and controlling their wastes. In this connection, the Environmental Protection Agency will restrict industrial liquid discharges of PCBs from PCB users. To keep levels in fish as low as possible, and in any case below FDA's interim action level of 5 parts per million, concentrations in rivers or lakes from all sources should not exceed 0.01 parts per billion.

6. The use of PCBs should not be banned entirely. Their continued use for transformers and capacitors in the near future is considered necessary because of the significantly increased risk of fire and explosion and the disruption of electrical service which would result from a ban on PCB use. Also, continued use of PCBs in transformers and capacitors presents a minimal risk of environmental contamination. The Monsanto Company, the sole domestic producer, has reported voluntarily eliminating its distribution of PCBs to all except manufacturers of electrical transformers and capacitors.

Pending passage of the Toxic Substances Control Act, the Federal Government does not have the legal authority to impose restrictions corresponding to the actions reported by Monsanto. Although some Federal enforcement authority is available, the Federal Government does not have the authority to control PCBs at their source.

7. Most capacitors presumably have been disposed of in landfills. PCB containing material buried in soil is not expected to migrate but should remain in place. In the past, many fluids containing PCBs have been disposed of in sewers. More appropriate means of disposal such as high-temperature (at least 970°C) incineration must be used instead.

8. PCBs are manufactured in countries other than the United States. Importation of PCBs as a chemical or as a component in products remains legally possible because the Toxic Substances Control Act has not yet become law. Electrical products imported from abroad may contain PCBs. The task force looks to international agreements to bring about some multi-national understanding on the sale and use of PCBs globally. Importation of PCBs for uses other than those singled out in the present pattern of voluntary limitations should be avoided by users.

As an additional measure, the United States has asked the Organization for Economic Cooperation and Development (OECD) through its Environment Committee to make a special review of member states' national policies concerning PCBs and also to identify products moving in international trade which contain PCBs. OECD, whose membership includes all major Western industrialized states plus Japan and Australia, has been giving priority attention to the problem of PCBs over the past year.

9. More scientific information about PCBs is needed, and several Government agencies are seeking it through research. The task force recognizes that the scientific basis of much of our knowledge must be

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strengthened through research. The total exposure of a human being to a given substance from all sources--air, water, and food--must be considered, and interactions of PCBs and other substances within and outside the body must be evaluated. Similar consideration must be given to the other body organisms.

Current scientific knowledge gained from laboratory animal experiments is often inadequate to allow reliable interpretation of the data in terms of possible effects on man. The scientific basis for interpreting such tests must be improved.

The situation regarding PCBs is not significantly different from the problem of other toxic susbstances which cause concern when they come into contact with man, his food, and his environment. Continuing vigilance on the part of Government agencies, industry, universities, and many other agencies both within and outside the Government will be necessary to achieve an effective system for assessing and controlling the hazards of toxic substances, including PCBs.

The task force, by reviewing research needs and the present Federal research effort, has helped to insure that these efforts of the agencies are well planned and coordinated. Certain Government laboratories as well as a number of non-Government scientists recently have embarked on additional research on PCBs, and the results will be communicated to the scientific public completely and promptly through normal channels such as meetings and journals.

I. PRODUCTION, DISTRIBUTION, AND USE OF PCBS

Polychlorinated biphenyls (PCBs) were first manufactured commercially in 1929. By virtue of their unusual chemical and physical properties, they achieved widespread use in a variety of applications. PCBs are now manufactured in Great Britain, France, Germany, the USSR, Japan, Spain, Italy, and Czechoslovakia, as well as in the United States.

In the United States, PCBs have been manufactured by a single producer, the Monsanto Company, and marketed under the tradename "Aroclor". Table 1 gives a breakdown, by category of use and by type of PCB, of the total U. S. production, domestic sales, and U. S. export sales from 1957 to the present. Figure 1 and Figure 2 summarize these data for the years 1963 through 1971.

Both production and domestic sales of PCBs roughly doubled between 1960 and 1970. If one assumes a constant rate of growth of domestic sales since 1930, the cumulative sales in North America by 1970 would be of the order of 500,000 tons. (1) Corresponding data on production and use of PCBs outside the United States are not available. Current estimates suggest that total U. S. production represents roughly one-half of the total world production.

As can be seen in Table 1, the majority of the PCB material produced in the U. S. was marketed domestically. Between 1963 and 1971, the proportion of the production which was exported averaged 13 percent. In 1971, the Monsanto Company reportedly undertook a variety of voluntary restrictions on the distribution of PCBs to various categories of industries. Both

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TABLE 1

PCB MANUFACTURING AND SALES
DATA FROM MONSANTO INDUSTRIAL CHEMICALS CO.
1957 THROUGH 1971
(Thousands of Pounds)

	1957	1958	1959	1960	1961	1962
TOTAL PRODUCTION (For Domestic Sales)(1)				37919	36515	38353
DOMESTIC SALES	32299	26061	31310	35214	37538	38043
DOMESTIC SALES BY CATEGORY						
Heat Transfer	-	-	-	-	-	157
Hydraulics/Lubricants	1612	1549	2685	2523	4110	3915
Misc. Industrial	704	755	1569	1559	2114	1681
Transformer	12955	5719	5984	7921	6281	7984
Capacitor	17028	14099	16499	16967	15935	15382
Plasticizer Applications(2)		3939	4573	6244	9098	8924
Petroleum Additives	-	-	-	-	-	-
Total	32299	26061	31310	35214	37538	38043
DOMESTIC SALES BY PCB GRADE						
Aroclor 1221	23	16	254	103	94	140
Aroclor 1232	196	113	240	155	241	224
Aroclor 1242	18222	10444	13598	18196	19827	20654
Aroclor 1248	1779	2559	3384	2827	4023	3463
Aroclor 1254	4461	6691	6754	6088	6294	6325
Aroclor 1260	7587	5982	6619	7330	6540	6595
Aroclor 1262	31	184	359	326	361	432
Arcolor 1268	-	72	102	189	158	210
Total	32299	26061	31310	35214	37538	38043

NOTE: (1) Production amounts prior to 1960 are not available.
(2) Amounts for plasticizer applications prior to 1958 are not available.

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TABLE 1 (cont.)

	1963	1964	1965	1966	1967	1968	1969	1970	1971	Prospect 1972
U.S. PRODUCTION	44734	50833	60480	65849	75309	82854	76387	85054	40471	25-30 m
DOMESTIC SALES (LBS.)	38132	44869	51796	59078	62466	65116	67194	73061	37635	25-30 m
U.S. EXPORT SALES	3647	4096	4234	6852	8124	11231	10624	13651	9876	?
U.S. DOMESTIC SALES BY CATEGORY										
Heat Transfer	582	929	1237	1766	2262	2529	3050	3958	3480	-
Hydraulics/Lubricants	3945	4374	4616	4258	4643	5765	8039	7403	1643	-
Misc. Industrial	1528	1692	1841	1779	1426	1283	1079	1627	578	-
Transformer	7290	7997	8657	8910	11071	11585	12105	13828	11528	25-30 m
Capacitor	15606	19540	23749	28884	29703	29550	25022	26708	17305	25-30 m
Plasticizer Applications	9181	10337	11696	13481	13361	14404	16460	19537	3102	-
Petroleum Additives	-	-	-	-	-	-	1439	-	-	-
U.S. DOMESTIC SALES BY PCB GRADE										
Aroclor 1221	361	596	369	528	442	136	507	1476	1600	300
Aroclor 1232	13	13	7	16	25	90	273	260	211	300
Aroclor 1242	18510	23571	31533	39557	43055	44853	45401	48588	21000	4000
Aroclor 1248	5013	5238	5565	5015	4704	4894	5650	4073	261	-
Aroclor 1254	5911	6260	7737	7035	6696	8891	9822	12421	5800	6000
Aroclor 1260	7626	8535	5831	5875	6417	5252	4439	4890	1750	600
Aroclor 1262	414	446	558	768	840	720	712	1023	-	-
Aroclor 1268	284	190	196	284	287	280	300	330	-	-

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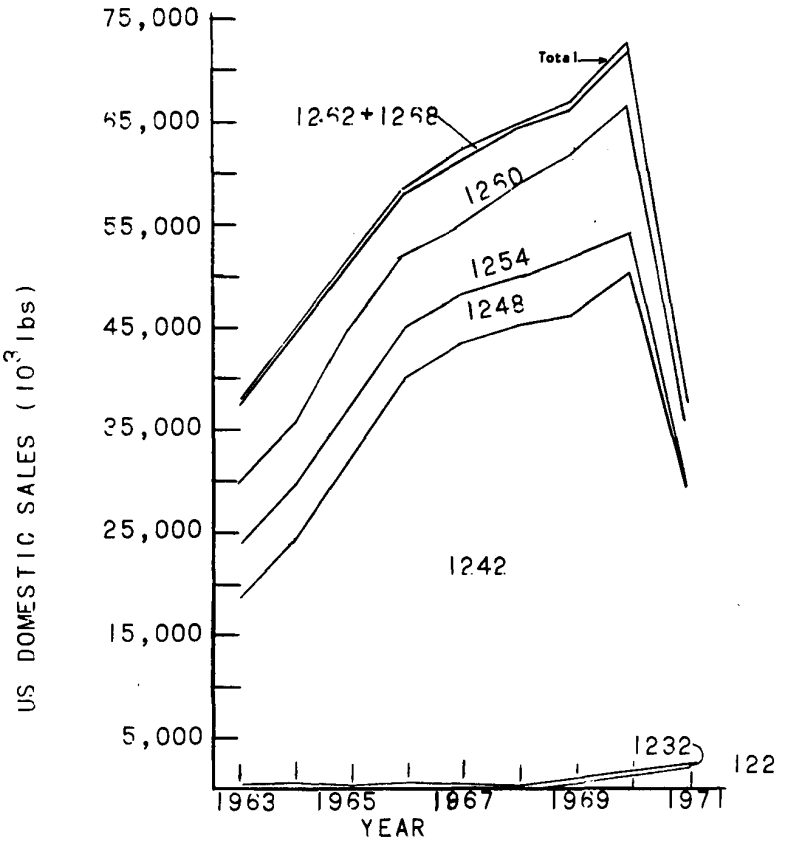

FIGURE 1. US DOMESTIC SALES OF PCBs BY GRADE The uppermost curve represents the total sale

From Nisbet, I.C.T., and Sarofim, A.F. (1)

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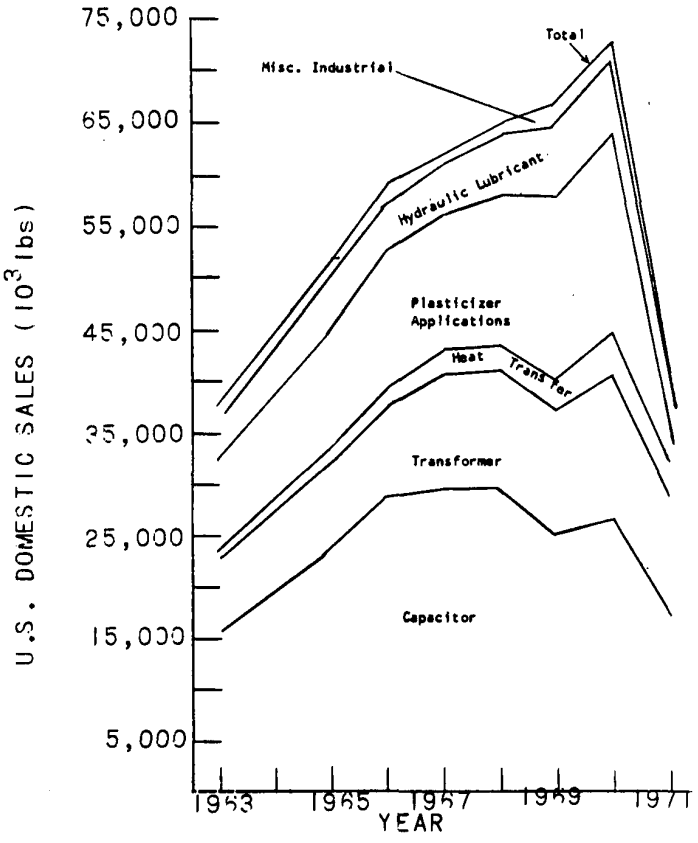

FIGURE 2. US DOMESTIC SALES OF PCBs by Category (The uppermost curve represents the total sales.)

From Nisbet, I.C.T., and Sarofim, A.F. (1)

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production and sales figures for 1971 were roughly half of those for 1970, when these volumes were at their peak (Table 1 and Figures 1 and 2). Projections for 1972 indicate an even lower volume.

Prior to 1971, about 40 percent of the PCB material in the United States was used in applications where containment was difficult and losses into the environment were probable. These uses included plasticizers, hydraulic fluids and lubricants, surface coatings, inks, adhesives, pesticide extenders, and microencapsulation of dyes for carbonless duplicating paper. The remaining 60 percent of domestic sales was used mainly in electrical applications (transformers and capacitors). In 1971, this fraction is expected to have reached approximately 90 percent of the total use, only about half of the total use in 1970.

In terms of the grade or family of PCB manufactured, the lower chlorinated species have generally made up the majority of the products produced. From the figures in Table 1 it can be seen that Aroclor 1242 and grades with lower percentages of chlorination characteristically composed one half or more of the total production between 1963 and 1970.

The largest categories of use of PCBs have been in capacitors and transformers and in certain "plasticizer" applications including carbonless duplicating paper. A large percentage of the production of Aroclor 1242 went into these three categories of products. (2) The major uses for PCBs prior to 1970 (in the order of importance as a reflection of the volume of material used) were:

Capacitors
Plasticizer applications
Transformer fluids
Hydraulic fluids and lubricants
Heat transfer fluids

II. CHEMICAL AND PHYSICAL PROPERTIES AND IMPURITIES

Chemical and Physical Properties of PCBs

Theoretically, there are 210 possible PCB compounds, but only about 100 are likely to occur in commercial products. The degree of chlorination determines the chemical and physical properties of the Aroclors; the first two digits of the numbered Aroclor represent the molecular type, the last two digits the average weight percent of chlorine. Their physical state thus varies from colorless, oily liquids to more viscous and increasingly darker liquids to, in the higher series, yellow and then black resins. The PCBs are not readily biodegradable. They resist breakdown by water, acids, and alkalis and have boiling points ranging from 278 to 475°C.

Analytical Techniques

Whereas in the past it was difficult to identify PCBs in the presence of other organochlorine compounds such as DDT and DDE, they can now be separated from interfering compounds and identified and measured by means of thin layer and gas liquid chromatography at levels less than 1 part

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per million in food and at significantly lower levels in air and water. Confirmation of their presence and molecular structure is possible by mass spectrometry. Various chromatographic columns and GLC detectors have been most useful in the analyses. Increased precision of residue detection in biological materials has also been made possible through the choice of chlorine specific detectors such as the microcoulometric detectors.

Contaminants and Impurities

The starting materials used in synthesis of PCBs determine to a large degree the type of impurity or contaminant in the commercial product. The contaminant variation, of course, renders some divergence in the LD 50 values or other toxicologic response of the PCBs. Fractionated samples of some PCBs of foreign manufacture have shown them to contain as contaminants the tetra- and pentachlorodibenzofurans, the hexa- and heptachloronaphthalenes. Further work is needed to ascertain whether additional impurities or contaminants are present in the various U. S. and foreign PCB products. Also, variance in biological response to the various PCB products should be correlated with analytical data obtained on the actual or likely presence of contaminants.

III. BENEFITS, UTILITY, AND ESSENTIALITY

The task force reviewed the several categories of uses to which PCBs had been put in the past to determine what was known of their utility and to ascertain if alternate or substitute materials were available or whether any of the present applications were essential.

The four major types of applications examined were:

1. Dielectric fluids for capacitors and transformers.
2. Industrial fluids for hydraulic, gas turbine, and vacuum pump uses.
3. Heat transfer fluids.
4. Plasticizers and miscellaneous uses.

This review of utility was undertaken by the National Bureau of Standards. The review was materially aided by information from the National Industrial Pollution Control Council and from certain professional independent testing and evaluation associations.

A major value of the PCB liquids is that those with four or more substituted chlorines per molecule are nonflammable as are their decomposition products, both vapors and arc-formed gaseous products. Thus they can be used as fluids at temperatures up to 700°F without the danger of explosions and fire. The major disadvantage of the PCBs is their toxicity and environmental contamination. The other comparable class of non-flammable fluids is the fluorocarbons, which typically have a lower vapor pressure and lower boiling point than the chlorinated compounds.

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Electrical Uses

PCBs are used in fluids (known as askarels) for electrically insulating and cooling transformers when the transformers are used in or near buildings. Being virtually free of fire and explosion hazards, PCBs can be used where failures of oil-insulated transformers would present a potential danger to life and property. PCBs also are superior to oils in reliability, in making small equipment possible, and in assuring long life and reliability to equipment. Table 1 shows the flammability ratings of two PCBs compared to five other common materials.

Table 1

Underwriters' Laboratories Flammability Ratings

Fluid	Flammability Rating
Ether	100
Gasoline	90-100
Ethyl Alcohol	60-70
Kerosene (100° F.P.)	30-40
Mineral Oil	10-20
Aroclor 1242 and MCS 1016	2-3

PCBs are used in transformers wherever fire protection is particularly important--for about 5 percent of all transformers.

Most of these transformers are located inside public, commercial, or industrial buildings--or on the roof tops of, or in close proximity to, such buildings--and require no special enclosures other than those necessary to prevent accidental hazardous mechanical or electrical contact of persons with the equipment.

The amount of Aroclor used in various types of transformers ranges from 40 to 500 gallons (516 to 6,450 pounds) with an average of about 235 gallons (3,032 pounds). During 1968, the last complete "normal" year for the electrical industry, the total amount of PCBs used in new transformers or as replacement fluid was approximately 1.3 million gallons (8.4 thousand tons).

The only present alternatives to Aroclor-insulated transformers are mineral oil-insulated transformers or dry-type transformers (either those open to the atmosphere or those that are gas-filled and sealed). Mineral oils are the preferred fluids when fire does not create a hazard. Dry transformers also can be used when space is available to install them. Fluorocarbon liquids require a special transformer design.

PCBs are used in more than 90 percent of the electric utility (large power) type and smaller industrial type capacitors made today. They are needed for safety, reliability, and long life, and to achieve sizes compatible with equipment and installation requirements.

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Almost 80 million PCB-impregnated capacitors are manufactured annually, most of them for first-time use. The principal types are high voltage power capacitors used primarily for power factor correction in the distribution of electric power; low voltage power capacitors installed in industrial plants at the load (typically large motors); ballast capacitors to improve the efficiency of lighting systems; and small industrial capacitors for power factor improvement in such equipment as air conditioning units, pumps, fans, etc.

Capacitors used in lighting and air conditioning applications contain 0.0005 to 0.09 gallons of PCB per unit. The largest power capacitors contain about 6.7 gallons of askarel. The National Electrical Code requires that any installation of capacitors in which any single unit contains more than 3 gallons of combustible liquid shall be in a vault like that required for transformers. During 1968, the last complete "normal" year for the electrical industry, the amount of PCBs used in capacitors was approximately 14.4 thousand tons.

Possible alternatives to PCB-impregnated capacitors are capacitors impregnated with mineral oil or certain other liquids. Flammable fluids in capacitors used in buildings are not allowed by insurance companies and building codes.

If codes did allow flammable materials in this use, replacement of PCBs in capacitors and transformers would require considerable time and money for re-engineering, manufacture, and application of substitute equipment, and lack of availability of PCBs for this equipment would cause a major and lengthy disruption in the nation's electrical system.

Industrial Applications

PCBs have been useful in hydraulic systems where leakage onto hot metal surfaces could cause fire, but substitute fluids are available. Gas turbines require lubrication at high temperatures. PCBs can be used but tend to be corrosive. Phosphate ester lubricants seem better in this respect. Chemical stability is more important than non-flammability for high temperature lubricants. PCB fluids are useful in diffusion booster pumps, but non-flammability is not especially important for diffusion pump liquids, and alternative liquids are available.

Heat Transfer Materials

Flammable heat transfer fluids present a fire hazard if they leak into a furnace or onto hot surfaces. The use of PCBs prevents this danger. In some cases water is a suitable substitute at moderately high temperatures, and other satisfactory heat transfer fluids are commercially available and in use.

Plasticizers

The PCBs are good plasticizers for use with adhesives, textiles, surface coatings, sealants, and copy paper. In some cases the PCBs act as fire retardants. There are no unique properties of PCBs for plasticizer uses, and equally effective alternatives are generally available (e.g., phosphate esters are often used as fire retardants).

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IV. OCCURRENCE, TRANSFER, AND CYCLING IN THE ENVIRONMENT

Given the diversity of uses of PCBs and their chemical characteristics (greater stability in the higher chlorine species), it is not surprising that the residues are widespread. While satisfactory quantitative estimates of the contribution of various pathways into the environment are not possible with existing data, there are enough data to be certain that PCBs do reach the environment at least from the following sources:

-- Open burning or incomplete incineration (at usual temperatures) of solid wastes, municipal and industrial. Incineration at 2000°F or above for two seconds will destroy PCBs, but poorly operated incinerators or open burning may result in PCBs being released to the atmosphere unchanged.

-- Vaporization from paints, coatings, plastics, etc. (Nisbet and Sarofim, 1) estimate that as much as 20 percent may be vaporized.

-- Municipal and some industrial sewers (PCBs present in treated as well as untreated wastes).

-- Accidental spills or improper wastes disposal practices.

-- Formerly, direct application to the environment as ingredients of pesticides or as carriers for pesticides (such uses of PCBs are now prohibited).

-- Dumping of sewage sludge, municipal and industrial solid waste, and dredge spoil at sea.

-- Sewage sludges disposed of on land.

-- Migration from surface coatings (paints, etc.) and packaging materials into foods and feeds.

Probably the largest amounts of PCBs circulating in the environment reach it through industrial and municipal discharges to inland and coastal waters.

The recommendation by the task force that "more scientific information about PCBs is needed" is illustrated by the sparsity of knowledge about PCBs in the environment. Only general statements can be made about how PCBs reach the environment, how they reach target organisms, and how much is present.

Nisbet and Sarofim (1) estimate that the total loss of PCBs into the U. S. environment over the last 40 years would approach 30,000 tons to the atmosphere, 60,000 tons to water and 300,000 tons to dumps. Of this total, remaining residues might be 20,000 tons from the air (which would be distributed on land or water), 30,000 tons in water, and perhaps 250,000 tons in dumps.

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Thousands of environmental and biological samples have been analyzed for the presence of PCBs. One or more of the PCB compounds have been detected in all environmental media, and in many organisms.

Water

The water environment is probably the principal sink and transport mechanism for PCBs. Calculations based on measured occurrences in municipal and industrial outfalls, in the receiving waters, and in the downstream reaches of the waterways demonstrate transport through the aquatic system. Measured residues in fishes from various environments suggest accumulations at the downstream ends of the drainageways.

There are few data on removal, disappearance, and sequestering of the substances in soils or bottom sediments of rivers, lakes, estuaries, or the ocean.

Organisms Other Than Man

PCBs being, like many of the organochlorine insecticides, fat soluble, are stored in the lipids of animals. Also like the chlorinated hydrocarbon insecticides, they resist metabolic changes, and tend to be concentrated at succeedingly higher levels in animals higher in the food chain. The higher chlorine PCBs are the most stable.

Occurrence and Sources of PCBs in Food

The identification of PCBs as a potential food contaminant was first reported in 1966. Subsequent investigations, including the development of analytical procedures for PCBs in foods and their incorporation in Federal programs for monitoring the nation's food and feed supply for pesticide residues and other chemical contaminants, established several sources from which foods may become contaminated with PCBs.

Environmental Contamination of Food

PCB residues in fresh water fish appear to be widespread geographically as a result of the environmental contamination of lakes and streams. Depending upon the location of sampling and the species of fish, PCB levels generally range from 1 to 10 parts per million. Foods of animal origin, such as meat, poultry, eggs, and milk contain, in some instances, low background levels of PCBs that may be attributable to environmental contamination.

Industrial Accidents

The widespread industrial uses of PCBs have resulted in a number of identified isolated accidents involving the direct contamination of animal feeds, which, in turn, caused human food to become contaminated.

-- Poultry and eggs became contaminated as a result of the leakage of PCB heat transfer fluid during the pasteurization of fish meal (poultry feed component).

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-- PCB residues in milk have resulted from the use of PCB in certain coatings on the inside walls of silos, which, in turn, contaminated the dairy feed silage.

-- The use of spent PCB transformer fluid as a herbicide spray vehicle allegedly contaminated dairy cattle grazing areas thereby causing residues in milk.

-- The grinding of bakery products along with their PCB-containing wrappers for use as poultry feed is suspected to have caused contamination of fowl.

These incidents, as well as others during the past several years, represent localized sources of contamination. Federal, State, and industry actions prevented most of the contaminated foods from being marketed.

Food Packaging

A significant percentage of food paper packaging materials contains PCBs and has resulted in the migration of low levels to the packaged food. This source of food contamination was identified in 1971. The origin of PCBs in packaging materials is not fully understood. Recycled waste paper containing PCB carbonless "carbon" papers is the prime source of PCB in paperboard product. Virgin paper products, however, have also been shown to contain PCB residues, probably as a result of the paper manufacturing processes. Data on current production of food packaging materials indicate that the levels are decreasing and are controllable so that the potential for PCB contamination of packaged foods can be minimized.

Dietary Intake

National monitoring data, and in particular FDA's total diet studies, indicate that the human dietary intake of PCBs is of a low order. For example: the dietary intake expressed as mg/kg body weight/day, and based on food consumption approximately twice as high as the normal diet, was less than 0.0001 in FY 1971 and 0.0001 in the first-half of FY 1972. As a point of reference, from 1965 to 1970 dietary intake of DDT was 0.0007 mg/kg body weight/day. Other investigations further disclose that except for unavoidable background levels in certain foods, the PCB contamination of food can be significantly reduced or eliminated through appropriate controls.

Man and the Ecosystem

In air and water away from immediate sources of waste discharge, levels of PCBs are low -- a few micrograms per cubic meter (parts per trillion; ppt) in air and less than a part per billion (ppb) in fresh water; soil or bottom sediments contain a few parts per billion, up to several hundred parts per million (ppm) near some industrial outfalls; from tenths of a ppm to tens of ppm in fish and up to hundreds of ppm in some fish and birds near the top of the food chain. To illustrate these relationships, 1/8 of an inch is about

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one trillionth of the distance to the moon; and a part per million is about 5 steps on a walk from Washington to San Francisco.

Man, who is at the top of a food chain, may also have PCB residues in his body fat. Analyses of tissue residues from 688 persons from three States showed two-thirds to have detectable residues, but only one-third contained residues of 1 part per million or more.

PCBs have been shown to accumulate in fish and aquatic invertebrates to levels of 75,000 times that present in the water, and to be accumulated from concentrations as low as 0.06 parts per billion (the lowest concentration for which experimental data are available). Thus, to keep levels in fish as low as possible, and in any case from reaching the 5 parts per million established by FDA as an interim action level for safety as food, concentrations in water should be less than 0.07 part per billion, or to allow some safety factor, 0.01 ppb. This level in water should be sufficiently low that fish and shellfish are not themselves adversely affected.

Existing data suggest that the principal route of PCBs through the environment is from waste streams into receiving waters, downstream movement in the waterways in the water and on sediments, accumulation from the water by aquatic organisms, and transfer to birds and mammals (including man) through residues in fish that are eaten.

Another route to man is through migration of residues to foods from PCB containing packaging materials. A third route to man may have been absorption through the skin from handling carbonless carbon paper. These exposures are being rapidly diminished since PCBs are no longer used in carbonless carbon paper. Presumably most of the PCB residues in paper made from waste paper also came from used carbonless carbon paper, which is no longer used in making food packaging material.

V.A. BIOLOGICAL EFFECTS ON MAN (METABOLISM, TOXICOLOGY, AND RESULTS OF HUMAN EXPOSURES)

Human beings occasionally have been exposed to high levels of PCBs. Some exposure has been the result of occupational experience or of accidental concentrations in food as in the accidental contamination of rice oil in Japan ("Yusho" episode). As far as can be determined, the number of exposed persons in these cases has been limited. Another source of contamination and exposure (although at a lower level) has been fish containing PCBs.

Samples of human fat have been examined to a limited extent for the presence of PCBs. As a result of this limited sampling, it has been concluded that some persons carry a body burden of PCB in their fat tissue. In contrast to the ubiquity and levels of DDT and its metabolite, DDE, in humans in this country, the current levels of PCBs do not appear to be as uniform in distribution. At the levels in which they are found, PCBs do not appear to present an imminent hazard.

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The acute toxicity of commercial PCBs in experimental animals appears to be low. In the case of the human exposure with the Yusho episode, the average dose to that exposed population was calculated as 2 gm. From this incident, it was estimated that the minimum dose necessary to produce positive clinical effects was 0.5 gm.

With a sufficiently high dose it is possible to distinguish a number of toxic actions of PCBs and their contaminants in mammals. Alterations in the functioning of the liver have been observed in a number of species, and these are attributed to PCBs. It is likely that other conditions, such as chloracne (severe skin eruptions), liver damage, and hydropericardium (accumulation of fluid in the sac which surrounds the heart) may be caused by a contaminant, chlorinated dibenzofuran.

The limited mammalian chronic studies of the Monsanto Company indicate no evidence for carcinogenicity. The possibilities of embryotoxicity and mutagenicity, however, are poorly studied and, hence, are ill-defined.

Because of the possibility of human exposure to PCBs, the task force recommends the following additional studies:

1. Toxicological evaluation of a select number of representative, purified PCB isomers as well as purified trace contaminants such as the chlorinated dibenzofurans.

2. Definitive mammalian elaboration of the kinetics, absorption, distribution, metabolism, and excretion of the technical PCBs as well as a number of key isomers and the chlorinated dibenzofurans.

3. Elaboration of the subcellular and intracellular actions of the technical PCBs as well as a number of representative isomers and chlorinated dibenzofurans.

4. More definitive epidemiological studies of the PCBs with both more representative population sampling and standardization on the basis of lipid content of the tissues.

V.B. BIOLOGICAL EFFECTS ON ANIMALS OTHER THAN MAN

The significance of PCBs to wild animals depends primarily upon the sublethal physiological effects of these substances rather than upon their lethal toxicity. They have accumulated in all portions of the natural environmental complex in a manner predictable from their high solubility in fat and their resistance to degradation.

PCBs can be lethally toxic to some fish and aquatic invertebrates when concentrations in the water are parts per billion or less. They are metabolized and excreted very slowly by these organisms.

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They are only moderately toxic to birds and mammals; lethal levels are similar to those of DDE. PCBs may have contributed to direct mortality of some adult birds in the field, but not to an extent to affect populations.

In sublethal exposure, PCBs are physiologically active and induce enzyme activity. Direct effects on reproduction have been shown for chickens, but not for ducks, quail, or doves. Some studies tentatively suggest the possibility of subtle behavioral effects and of interactions with disease organisms or other environmental chemicals.

Full evaluation of actual or potential effects in the environment is hampered by the complex nature of the mixtures that compose PCBs, and by the inclusion of contaminants in these mixtures. As experimental studies have been conducted with the unaltered products, as sold, the results may not properly reflect the effects of the components as they exist in the environment.

Although fully conclusive data are not available, the evidence for toxic and physiological effects indicates that the PCBs must be viewed as potential problems at present environmental levels.

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FOOTNOTES

1. Nisbet, I.C.T., and A.F. Sarofim, "Rates and Routes of Transport of PCBs in the Environment". Paper delivered at International Scientific Meeting on PCBs sponsored by the National Institute of Environmental Health Sciences, and held at the Quail Roost Conference Center, Rougemont, North Carolina, December 20-21, 1971. (Environmental Health in Perspective (in press) 1972.)

2. Data supplied by the Monsanto Company.

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Chemical and Physical Properties of PCBs

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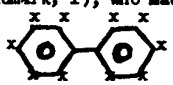
APPENDIX A

Chemical and Physical Properties of PCBs

I. Chemical and Physical Characteristics

The PCBs (trade name Aroclors in the United States, unknown in Soviet Union, Phenoclor in France, Colphen in Germany, and Kanachlor in Japan) are manufactured in the United States primarily by Monsanto Chemical Company. There are also suppliers in Europe and Japan. Because of their unique chemical and physical properties the PCBs have been widely used in paints, electrical transformers, condensers, non-inflammable oils, adhesives, plasticizers, heat transfer systems, hydraulic fluids, caulking materials, printing inks, and many other uses where the nonflammability and heat resistance properties are useful. Monsanto Chemical Company, reacting to the concern about extensive environmental contamination by the Aroclors, have recommended discontinuance and curtailed uses in equipment or products which inadvertently lead to contamination of the environment (food products, potable water supplies, and as an air pollutant). Presumably the history of the polychlorinated biphenyls started in 1930 when an industrial use of non-flammable oils was introduced for electrical transformers, condensers, and paint. It was probably not until the mid-sixties that an awareness of the environmental contamination came about with the realization that after 40 years use of the Aroclors, there was as extensive a problem of contamination as with the organochlorine pesticide DDT. Swedish scientists drew attention to the fact that PCB has been found in fish and birds in their surveillance in 1966, and soon this alerted investigators in other countries to the problem. Surveillance of total diets by FDA failed to report any PCB in fruits and vegetables.

Perhaps the accumulation of PCBs in environmental substrates or living organisms can be associated with the particular chemical and physical properties of these series of synthetic chlorinated aromatic compounds. For example, the nonenvironmental degradability or biodegradability accounts for it as a residue in many media and for its persistence. Like DDT, closely related structurally, it will enter the organism, be stored in the depot fat, and thus be transferred through the food chain at increasing concentrations. The basic structure of PCBs is shown below where x represents any number of possible positions for chlorination leading to about 210 possible combinations, of which 102 are probable. (Widmark, 1), who made this calculation in developing



criteria for these limitations, noted that the criteria were based on compounds containing five to eight chlorine atoms per molecule and the number of chlorine atoms per ring differing by not more than one.

The Aroclors may consist of chlorinated biphenyls, chlorinated terphenyls, or a mixture of these compounds. Invariably, although a specific numbered Aroclor may represent a molecular type and degree of chlorination or weight percent of chlorine, the product is a mixture of compounds in a series such as 1200, 2500, 4400, and 5400. The first two digits represent the molecular type,

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and the last two digits give the weight percent of chlorine. For example:

1200 - chlorinated bipheynls

2500 - blend of chlorinated biphenyls and chlorinated triphenyls (75:25)

4400 - blend of chlorinated biphenyls and chlorinated triphenyls (60:40)

5400 - chlorinated triphenyls

Thus Aroclor 1242 would be a chlorinated biphenyl containing 42 percent chlorine. These biphenyls produced by Monsanto range from 21 to 68 percent chlorine. To refer back to the existence of many possible isomers, it is of interest that Aroclor 1260 would show the presence of 11 isomers; five containing six chlorine atoms, five containing seven chlorine atoms, and one containing eight chlorine atoms (2). Bagley, et al, (3) studying Aroclor 1254, observed 18 distinct compounds. However, as previously stated, the number of compounds present or identified by mass spectrographic studies is much less than theoretically deduced.

The polychlorinated biphenyls which are chemically inert were described in the literature back in 1881 by Schmidt and Shultz (4) but achieved by Swann Company in 1930 and fully developed for industrial use in that year. During the 40-year era, 1930-1970, it was discovered that Aroclors could be used to extend the effectiveness or lethality of organochlorine insecticides such as chlordane, aldrin, and dieldrin in pesticidal formulations, thus enhancing the possibilities for proliferation into the environment (5). Additionally, there were indications that PCB had a synergistic effect on lindane (6).

The Aroclors, as previously stated, are chemically inert, making them ideally suited for certain industrial uses. They are not hydrolyzed by water and resist alkalis, acid, and corrosive chemicals. Since they are not volatile, their boiling points range from 278 C for Aroclor 1221 to 451°C for Aroclor 1268. They are stable to long heating and can be distilled at ordinary pressure without any carbonization or decomposition. As might be expected, since they are insoluble in aqueous media, they are quite soluble in hydrocarbon solvents. It is believed that PCBs are more stable than DDT and metabolites and, accordingly, resistant to biodegradability or biological decomposition. The proximity of the aromatic rings probably accounts for the lack of degradation of transformation as with DDT, which has an ethane linkage intervening the aromatic nuclie.

PCBs and formulations thereof have a wide range in viscosity and physical state, from colorless mobile-like oily characteristics to yellow-green clear mobile oil, to light yellow viscous oil, to light yellow soft sticky resin, to white powder, to light yellow clear brittle resin, to yellow transparent sticky resin, to clear yellow-amber brittle resin, to a black opaque brittle resin. Other properties such as low solubility in water and high dielectric constants make them versatile for industrial uses. Many of these

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PCB preparations have strong adhesive properties, and it is this use in bonding of paper and cardboard in certain packages for food, plus reuse of printing paper in manufacture of grayboard, that has led to a few problems of contamination of certain food products. A rather complete compilation of these properties is listed in the attached table from a report by Monsanto Company.

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Table 3

GENERAL PHYSICAL PROPERTIES OF THE

AROCLOR CHLORINATED COMPOUNDS

	Arochlor 1221 Colorless mobile oil	Arochlor 1232 Practically colorless mobile oil	Arochlor 1242 Practically colorless mobile oil	Arochlor 1248 Colorless to light yellow-green, clear, mobile oil	Arochlor 1254 Light yellow viscous oil	Arochlor 1260 Light yellow soft sticky resin
Form						
Color	SO Max. (APHA)	SO Max. (APHA)	SO Max. (APHA)	SO Max. (APHA)	SO Max. (APHA)	SO Max. (APHA)
Acidity - Maximum (mgs. KOH per Gm.)	0.014	0.014	0.010	0.010	0.010	0.014
Average Coefficient of Expansion....cc/cc/°C	0.00071 (15°-40°C)	0.00073 (25°-100°C)	0.00068 (25°- 65°C)	0.00070 (25°-65°C)	0.00066 (25°-65°C)	0.00057 (20°-100°C)
Typical Density Specific Gravity Pounds per gallon - 25°C (77°F)	1.182-1.192 (25°/15.5°C) 9.85	1.270-1.280 (25°/15.5°C) 10.55	1.381-1.392 (25°/15.5°C) 11.50	1.405-1.415 (65°/15.5°C) 12.04	1.495-1.505 (65°/15.5°C) 12.82	1.555-1.566 (90°/15.5°C) 13.50
Distillation Range - ASTM D-20 (Mod.) Corr. °C	275°-320°	290°-325°	325°-366°	340°-375°	365°-390°	385°-420°
Evaporation Loss - % - ASTM D-6 Mod. 165°C.....5 hrs. 100°C.....6 hrs.	- 1.0 to 1.5	- 1.0 to 1.5	3.0 to 3.6 0.0 to 0.4	3.0 to 4.0 0.0 to 0.3	1.1 to 1.3 0.0 to 0.2	0.5 to 0.8 0.0 to 0.1
Flash Point - Cleveland Open Cup °C °F	141°-150° 286 -302	152°-154° 305 - 310	176°-180° 348 - 356	193°-196° 379°-384°	None	None
Fire Point - Cleveland Open Cup °C °F	176° 349°	238° 460°	None*	None	None	None
Pour Point - ASTM D-97 °C	Crystals at 1°C	-35.5°	-19°	-7° 19.4°	10° 50°	31° 88°
°F	Crystals at 34°F	-32°	2°			
Softening Point - ASTM E-28 °C °F	- -	- -	- -	-	-	-
Refractive Index - D-1ine - 20°C	1.617-1.618	1.620-1.622	1.627-1.629	1.630-1.631	1.639-1.641	1.647-1.649
Viscosity - Saybolt Universal 210°F (98.9°C) Sec. (ASTM - D-88)	30-31	31-32	34-35	36-37	44-48	72-74
130°F (54.4°C)	35-37	39-41	49-56	73-80	260-340	3200-4500
100°F (37.8°C)	38-41	44-51	82-92	185-240	1800-2500	-
*ABOVE indicates-"No fire point up to boiling temperature						
Oral LD_{50}-rate (approx.) - mg/kg	3980*	4470*	8650*	11,000*	11,900*	10,000**
Skin MLD - rabbits - mg/kg	>2000*	>1260*	>794*	>794*	>1260*	>1260**
administered	<3160*	<2000*	<1260*	<1260*	<2000*	<2000**

*Undiluted **administered as 50% soln. in corn oil # 331/3% in corn oil 20% soln. in corn oil. ### 10% soln. in corn oil.

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Aroclor 1262 Light yellow sticky clear resin	Aroclor 1268 White to off-white powder	Aroclor 4465 Light-yellow, clear, brittle resin	Aroclor 5442 Yellow Transparent sticky resin	Aroclor 5460 Clear, Yellow-to-amber, brittle resin	Aroclor 2565 Black, opaque brittle resin
50 Max. (APHA)	1.5 Max. NPA (molten)	2 Max. NPA (molten)	2 Max. NPA (molten)	2 Max. NPA (molten)	-
0.014	0.05	0.05	0.05	0.05	1.4
0.00054 (25°-65°C)	0.00067 (20°-100°C)	0.00061 (25°-46°C)	0.00123 (25°-99°C)	0.00179 (25°-124°C)	0.0066 (25°-65°C)
1.572-1.583 (90°/15.5°C) 13.72	1.804-1.811 (25°/25°C) 15.09	1.670 (25°/25°C) 13.91	1.520 (25°/25°C) 12.24	1.670 (25°/25°C) 13.91	1.734 (25°/25°C) 14.44
395°-425°	435°-450°	230°-320° at 4 mm. Hg.	215°-300° at 4 mm. Hg.	260°-335° at 5 mm. Hg.	-
0.5 to 0.6 0.0 to 0.1	0.1 to 0.2 0.0 to 0.06	0.2 to 0.3 0.0 to 0.02	0.2 0.01	0.03 1.5 to 1.7 (at 260°-5 hrs.)	0.2 to 0.3 -
None	None	None	247° 477°	None	None
None	None	None	> 350° >662°	None	None
35°-38° 95°	- -	- -	46° 115°	- -	- -
-	150°to 170° (hold pt.) 202°to 210° (hold pt.)	60°to 66° 140°to 151°	46°to 52° 115°to 126°	98°to 105.5° 208°to 222°	66°to 72° 149°to 162°
1.6501-1.6517	-	1.664-1.667	-	1.660-1.664	
86-100	-	90-150 (260°F or 130°C)	300-400	-	-
600-850 (160°F or 71°C)	-	-	-	-	-
-	-	-	-	-	-
11,300**	10,900**	16,000**	10,600**	19,200	6,110
≥1260**	≥2510	2000**	≥1260**	≥744	≥2000
≤3160**		≤3160**	≤2000**	-	≤3160

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II. Problems in Analytical Chemistry - Comparison of Methods

II. 1 Separation

One of the primary and early problems in the identification of the polychlorinated biphenyls (PCBs) was that of possible interference in the GLC determination of organochlorine pesticides, where the PCB occurred along with the pesticide residue. During 1969 and 1970, various investigators worked on methods for separating PCB from pesticides (7-14). An excellent treatment of the subject of PCB as a contaminant in the environment, as related to its detection in the presence of other compounds, is covered by Jensen (15).

Jensen (15), in his review, has shown that some of the earlier studies of DDT and DDE in human fat by GLC must have been inordinately high. He first identified some GLC peaks in wildlife in 1964-66 that were not reconcilable but later identified chromatograph peaks from human fat that were not attributable to DDT or metabolites of DDT, which were more nearly comparable to peaks ascribed to PCB. The possibility that these peaks could be attributed to naturally occurring constituents in fish eaten by man was discarded when it was suggested that these same peaks could come from environmental pollutants.

To further establish the then uncertain composition of the chemicals causing the false DDT peaks, Jensen (15) separated out one of the chemicals by GLC and subjected this fraction to mass spectrometry. The mass spectrometry data showed compounds with molecular weights of 324, 358, 392, and 426. The molecular weight differences or differences of 34 mass units suggested one less chlorine atom per position on a carbon atom. In essence, through calculation, he deduced that these unknown chemicals could only be polychlorinated hydrocarbons, in this case having 5, 6, 7, and 8 chlorine atoms in the molecule. He verified his conclusion by introducing a synthetic or known PCB into the mass spectrometer. He also found that PCB standard chromatogram matched those with the same retention times as observed in various samples he analyzed, i. e., eagles, fish, or other wildlife.

It is interesting to point out that failure to recognize such interferences led to spurious results and data and obviously led to the overestimation of DDT in our environment, in man, in wildlife, and aquatic organisms. Jensen (15) referred to PCB as a new pollutant, but, as stated earlier, PCB was with us in the environment since 1930 and thus it would be an old pollutant only recently correctly identified by appropriate analytical chemistry.

In earlier work in the identification of some organochlorine pesticides such as p,p'-DDT; o,p-TDE (DDD), lindane, dieldrin, aldrin, heptachlor, and lindane, some unidentified spots and peaks in TLC and GLC interfered with the detection of these pesticidal chemicals. Jensen (15) in his work extracted the compounds from biological samples and, by appropriate cleanup of some contaminants, identified the pesticides and PCBs by thin layer and gas liquid chromatography followed by mass spectrometry. In 1964, Jensen used TLC to separate the fat soluble chlorinated hydrocarbons from the rest of the sample.

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For pesticide analysis, the common detector is the electron capture detector. For sensitivity, one can determine levels at the picogram range. However, this detector, not specific for chlorine, also gives an answer for oxygen containing compounds. Although the response here is much lower, this can be counter balanced if the level of oxygen containing impurity is somewhat higher. This system has certain inherent disadvantages in that two different compounds can have the same retention time and thus be detected as one compound or the recorded peak does not need to contain chlorine since, as indicated above, the detector is not that specific (15). However, by using different columns with variant properties, one can inject a sample into the instrument and thus secure a good separation.

To further demonstrate whether a compound which is responsible for a certain peak contains chlorine, one can concentrate the sample and analyze by a less sensitive chlorine specific detector such as the microcoloumetric detector. This, therefore, enhances the specificity in this residue analysis.

When Jensen (15) analyzed human fat for DDT by use of two columns, he was able to separate from human fat the two "DDT" peaks into four peaks. Two of these peaks were similar to those for o,p and p,p'-DDT, but two of the peaks were unidentified. As indicated earlier, he thought these peaks were due to naturally occurring components, which would be found in the fish consumed, such as pike. He was able to rule out this premise by treating the sample with alcoholic KOH and H_2SO_4 which would chemically alter DDT but not the more stable or inert polychlorinated biphenyl. Jensen simultaneously noted that samples of pike from a certain region, containing the then unidentified component, were correlated or associated with an area of extreme pollution. Similarly, eagles that fed on pike showed this unidentified component in their tissue. The compound in the eagles had the same retention time as the pike, thus confirming that this was a problem of biological magnification rather than the mere occurrence of a natural biochemical entity.

Another speculation was that metabolites of DDT could explain the appearance of unidentified peaks. Interestingly enough, these same peaks were found in an eagle sample collected in 1943 (prior to use of DDT in Sweden), thus implying that the DDT metabolites were not the potential unknowns. In addition, mercurials were considered, but the occurrence in water, fish and pheasants did not correlate or show a consistent pattern.

A method for the separation of polychlorinated biphenyls from DDT and its analogs has been developed by Armour and Burke of FDA (16). Identification by means of a combination of high resolution gas chromatography and mass spectrometry has been carried out by three independent laboratories in Sweden (17), Holland (2) and the United States (3) in addition to those named above.

There is a need to measure PCBs at extremely low levels of the order of 1 part in 10 or less. Accordingly, the method must be both specific and highly sensitive. Hence gas chromatography, using electron capture detection, has been extensively used with occasional identification and structure confirmation by gas chromatography mass spectrometry. This involves such steps as (a) sample preparation, (b) extraction of PCBs, (c) removal of interferences, (d) concentration, and (e) final gas chromatography and measurement.

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Some biological samples require more extensive pretreatment than for water or effluents from industrial plants. The measurement of PCB concentration of effluents is a simple procedure involving chromatogram of sample and then chromatograms of standard samples developed from known PCBs. For more accurate analysis, calibration graphs can be developed from known specific peaks in the standard samples (15).

In the following charts (Figures 1-6) some chromatograms of the various representative chlorobiphenyl Aroclors are presented according to Armour (18).

Some additional points might be made relevant to separation and quantitation of PCBs. First of all, in separation, the methods used prior to quantitation of residues in samples containing PCBs and chlorinated hydrocarbons fall essentially into two categories: (a) those which necessitate destruction or alteration of one or more of the compounds and (b) those which do not. Peakall and Lincer (19) have described in detail the procedures used for separation.

Briefly, the first group requires nitration ($HNO_3 + H_2SO_4$) treatment at 0°C for 5 minutes, which destroys or alters many of the organochlorine pesticides; thus PCB is left unaffected along with lindane and BHC. Some workers such as Armour and Burke (1) reported that complex chromatograms resulted after nitration, which could not be related to the unreacted DDT-PCB mixture. Thus, nitration was not pursued as a practical means of separating DDT and PCB for further analyses.

Saponification with alcoholic NaOH or KOH will dehydrochlorinate some pesticides such as Perthane, Toxaphene, DDD and DDT to their respective olefinic compounds (20).

Perhaps the more desirable technique allows for special separation of many chlorinated hydrocarbon pesticides from PCB. This involves using various columns and solvents. Another procedure is to use a series of differing polarity columns in the gas chromatograph at the time of determination (21).

II.2 Quantitation

Koeman, et al (2) measured residues in Japanese quail by using one of the peaks in a phenachlor DP6 mixture as standard. Risebrough (22) quantitated relative levels of PCBs by assuming that each PCB compound produced the same peak height with the electron capture detector as the same amount of weight by p,p'-DDE. After adding the heights of the individual peaks, the total was multiplied by a factor derived from measurements of standard solutions with EC and MC detectors. Jensen and co-workers (23) reported concentrations of PCB as the sum of all PCB components and based the estimation on several detection systems such as mass spectrometry and EC and MC detectors. In spite of all these techniques, these investigators consider the method approximate and correct only within a factor of 2.

There are other modifications of techniques, but with all the quantitation methodology available, relative estimates of concentrations of PCB are

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GAS CHROMATOGRAPHIC CONDITIONS

Figures 1 - 6
Columns: (1) 10% DC-200 on Chromosorb W HP

(2) 15% QF-1/10% DC-200 on Chromosorb W HP

6 ft x 4mm id.

200°C

120 ml N_2/min.

Detector: electron capture (tritium)

200°C

one nanogram heptachlor epoxide causes one half scale recorder deflection

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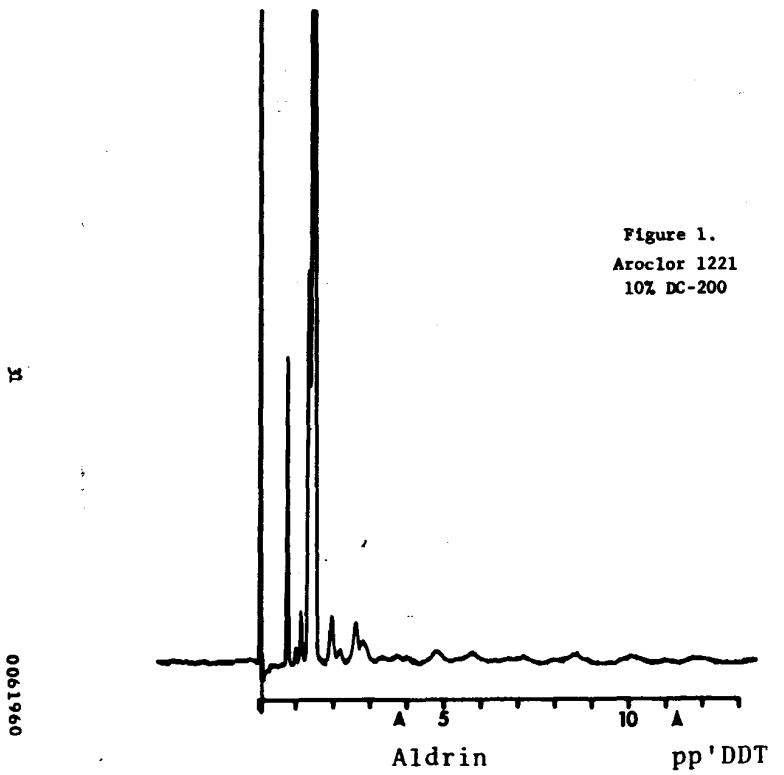

Figure 1.
Aroclor 1221
10% DC-200

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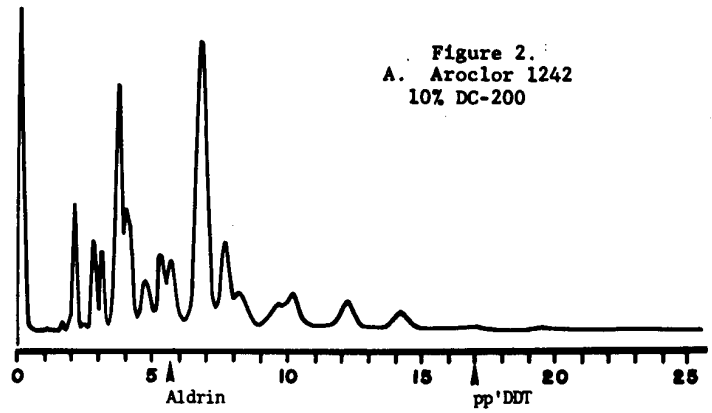
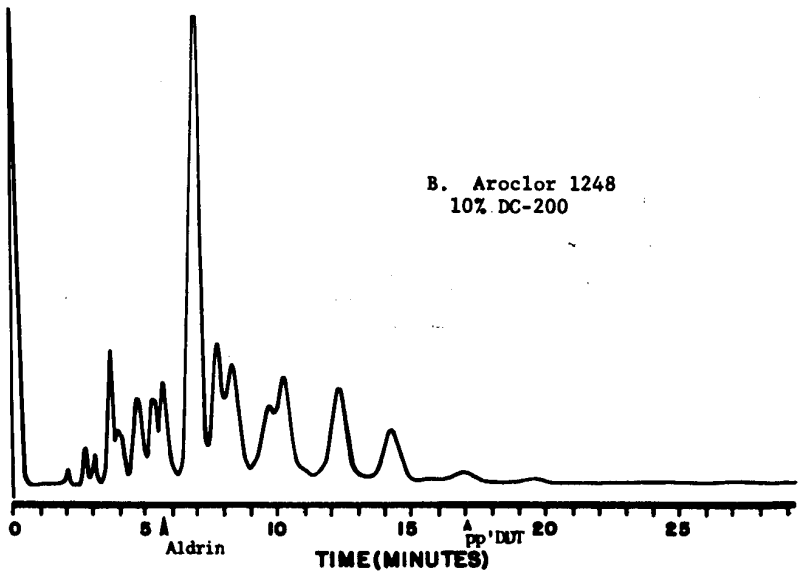


Figure 2.
A. Aroclor 1242
10% DC-200



B. Aroclor 1248
10% DC-200

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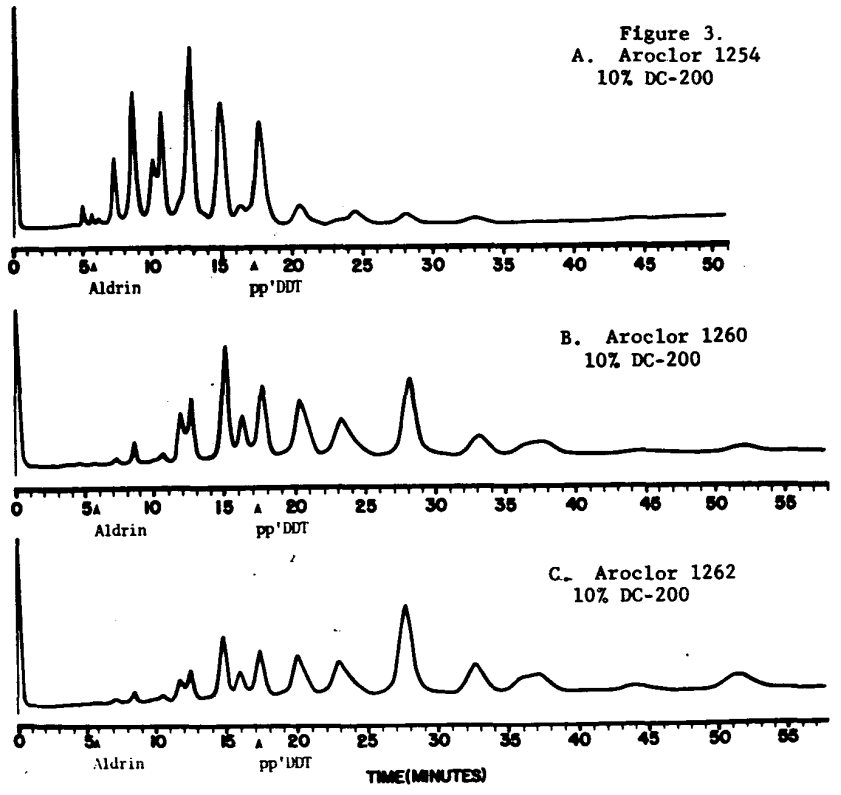

Figure 3.

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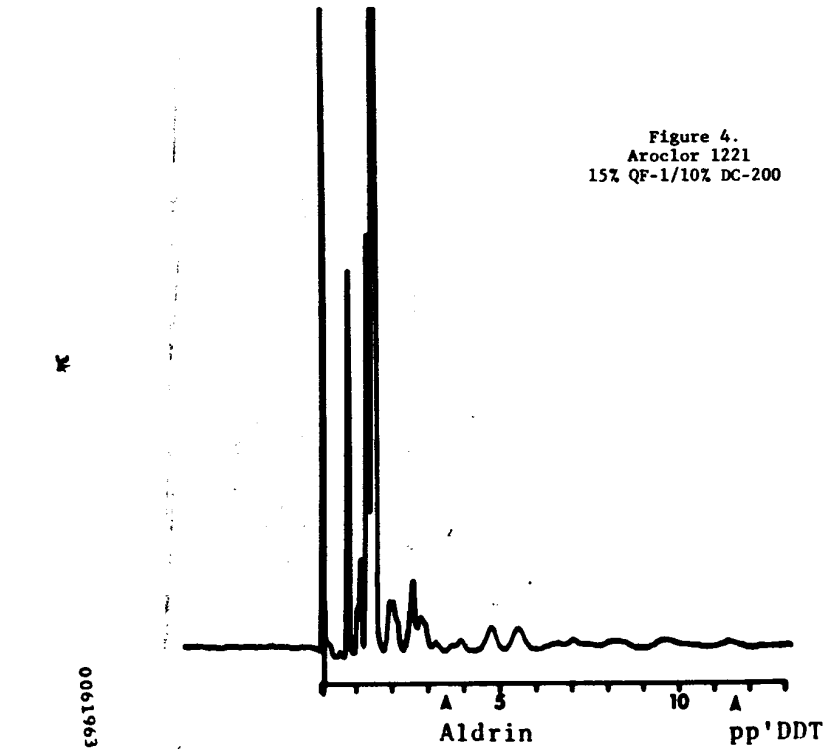

Figure 4.
Aroclor 1221
15% QF-1/10% DC-200

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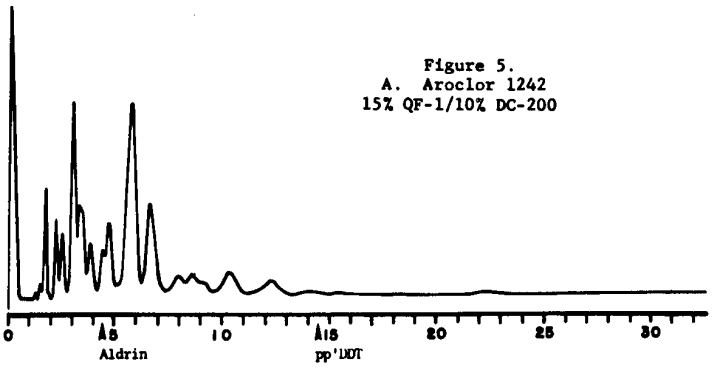
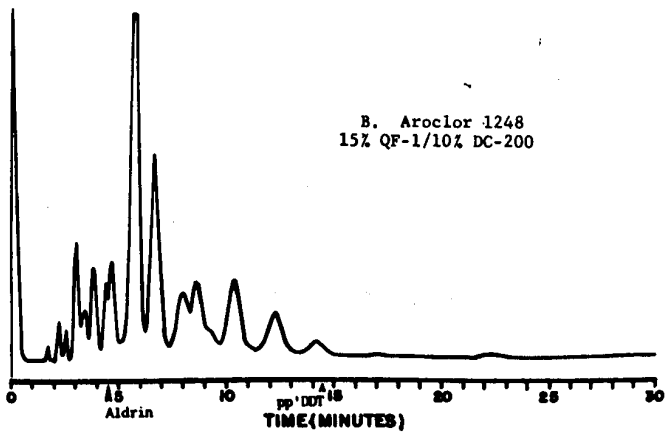


Figure 5.
A. Aroclor 1242
15% QF-1/10% DC-200



B. Aroclor 1248
15% QF-1/10% DC-200

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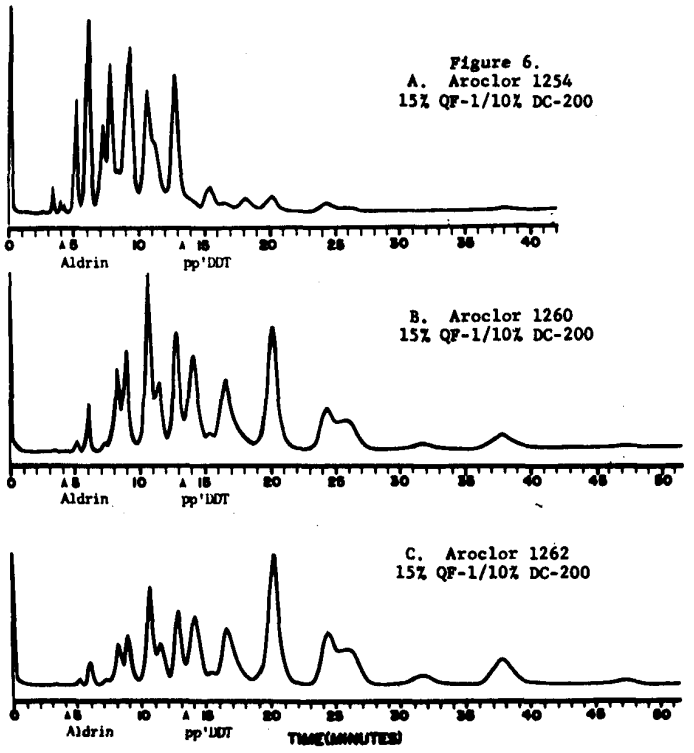

Figure 6.

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only approximate. At such time, when one is able to synthesize the individual PCB components commonly found in the environment and identify them in terms of individual peaks, then the estimation remains, as stated, a relative concentration.

However, for most biological assessment work, the correct order of magnitude and accurate relative amounts of PCB provide the requisite information for this purpose.

III. Contaminants, Impurities, or Other Chemical Moieties in PCBs

In the analysis of the polychlorinated biphenyls, one encounters a problem similar to that of the polychlorophenols and 2,4,5-T in that certain contaminants or impurities prevail. These impurities arise from the basic starting materials or compounds used in the synthesis and also from the processing conditions used in the chlorination procedures. In this respect, the series of compounds identified through GLC and mass spectrometry are somewhat similar in nature to the spectrum of dioxins found in working with chlorinated phenols, hexachlorophene, 2,4,5-T and related synthetic organic chemicals.

It has been noted in the bioassay of various Aroclors for toxicity, usually testing on chickens, that there was a variance in toxicity of certain PCB preparations. Since the occurrence of lesions resembled those of chick edema in birds fed PCB, this prompted a comparative study between three compounds, Phenoclor DP6, Clophen A60, and Aroclor 1260 by Vos et al in 1970 (24). In this study, the specific PCBs were fractionated, analyzed, and bioassayed using the chick embryo assay.

These studies revealed the presence of certain polar compounds which are present as impurities in the various PCBs and thus explain the variance in toxicity of certain commercial PCB preparations. In Table 2 some of the retentions and mass spectrometric data of some of the peaks identifying the presence of some dibenzofurans, chlorinated napthalenes, and associated chlorinated biphenyls are indicated, proving the presence of impurities.

Table 2
Relative Retentions, Mass Spectrometric Data on PCB Fractionated Sample

Peak No.	Relative Retention	Mass Nos. and No. of Cl atoms per mol	Identity of Compound
1	1.40	304 (4 Cl)	Tetrachlorodibenzofuran
2	1.58	332 (6 Cl)	Hexachloronapthalene
3	1.74	358 (6 Cl) 392 (7 Cl)	Hexachlorobiphenyl Heptachlorobiphenyl
4	2.42	338 (5 Cl) 392 (7 Cl)	Pentachlorodibenzofuran Heptachlorobiphenyl
5	3.48	366 (7 Cl)	Heptachloronapthalene

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Thus far, the dibenzofurans, the chlorinated napthalenes and chlorinated dipehnyls appear to be the major contaminants or impurities detected in various PCB samples. It is not unlikely that other chemical moieties will be characterized as further identification work proceeds. These contaminants arise, as previously indicated, by method of manufacture and the procedure for distillation of PCB, in which NaOH can be used. Through this NaOH treatment at elevated temperatures, sequentially polychlorohydroxybiphenyls are produced and then, through loss of hydrochloric acid, the dibenzofuran derivatives result (25).

The chlorinated napthalenes are far less toxic than the chlorinated dibenzofurans (26). Thus, one might expect, with the varying ratios of impurities with variant toxicities in different PCB samples assayed for biological effects and toxicity, that the end result will depend on such factors in examining various commercial PCB preparations.

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APPENDIX B

Use and Replaceability of PCBs

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Use and Replaceability of PCBs

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APPENDIX B

Use and Replaceability of PCBs

I. DIELECTRIC FLUIDS

Dielectric (electrically insulating) liquids are important to the electrical industry for filling agents or impregnants in transformers, capacitors, and other devices. Besides their electrical functions, the liquids may also be used for cooling and arc quenching functions. Detailed discussions of dielectric fluid applications are available (1-4).

A. CAPACITORS

Generally, industrially important capacitors use liquid impregnated cellulose paper as a dielectric. The required properties of the liquid are:

1. Non-flammability (important for preventing fires, particularly in indoor use).
2. Dielectric constant matching that of paper. A good match reduces electric field inhomogeneities, increases dielectric strength and lifetime, and allows decrease in capacitor size.
3. Low dissipation factor (reduces energy loss and destructive heating in a capacitor).
4. High dielectric strength (prevents breakdown and allows decrease in capacitor size).
5. High chemical stability (increases capacitor lifetime and stabilizes its performance).
6. Low vapor pressure (increases physical stability).
7. Inert decomposition products in an electric arc (prevents explosion or corrosion following breakdown).
8. Low toxicity of the material and its decomposition products.
9. Low cost.

1. Advantages and Disadvantages of PCB in Capacitors

The PCB capacitor liquids, commonly called askarels, are mixtures of chlorinated biphenyls and chlorinated benzenes. Several standard mixtures are specified by ASTM (5). The askarel capacitor liquids and their decomposition products are non-flammable. Thus their use in capacitors greatly reduces fire and explosion hazards. This characteristic permits economies where safety codes require fireproof enclosures for capacitors containing flammable liquids.

The dielectric constant of the askarels is high compared to other common dielectric liquids. Doubling the dielectric constant of the dielectric allows a reduction by half in the area of the capacitor electrodes, and a significant saving in the cost of construction and installation. The dielectric constant of askarels closely matches that of the capacitor paper.

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The askarels are adequate with regard to dissipation factor and dielectric strength and have good chemical stability and low vapor pressure. The breakdown products, in particular HCl, have the advantage of being non-flammable, but are highly corrosive. This dictates the use of special corrosion resistant materials inside the capacitors.

The major disadvantage of the askarels is their suspected toxicity. In ordinary capacitor usage the askarels are used in closed systems to prevent contamination from moisture. This practice also prevents the askarels from reaching the environment. However, when electrical failure occurs, sealed capacitors can leak and are ordinarily discarded.

2. Replaceability of PCB in Capacitors

Capacitors can be made dry or with gas dielectrics. Dry capacitors have inferior electrical strength to liquid-filled capacitors and, for comparable performance, must be made larger.

The major disadvantage of alternative capacitor liquids is their flammability. When comparing flash point data, as in Table 1, askarel flash points comparable to those of other capacitor liquids are sometimes listed. ASTM states that these are not true flash points but are pseudo-flash points which differ noticeably from the flash obtained on combustible materials, and such a flash point is not indicative of a fire hazard (5). The requirement of non-flammability for most capacitor uses is critical, and capacitors with flammable liquids are forbidden in many cases by the National Electrical Code (6). In other cases replacement of askarel capacitors with flammable capacitors requires use of fireproof installations (6).

The fluorocarbons are one group of non-flammable liquids which are used for some dielectric applications (1). The ones listed in Table 2 have low dielectric constants and could not directly replace askarels without increasing capacitor size. The fluorocarbons have low toxicity but the decomposition products may be toxic (1). They are generally more volatile than askarels and are considerably more expensive. The fluorocarbons are a possible replacement for PCB liquids for capacitor use, but no fluorocarbon liquid is known to be available and acceptable for this purpose.

Besides the problem of flammability, possible PCB replacement liquids generally lack either a sufficiently high dielectric constant to keep capacitor size down or a sufficiently high dielectric strength. For example (see Table 1) the silicones have the disadvantage of a low dielectric constant whereas the organic esters often have poor dielectric strength. Some silicones deteriorate rapidly under electric arcing (7).

The acceptance of a PCB substitute is a complex process involving not only users but also various regulatory groups. In most of the electrical industry, regulation is non-governmental, and the primary regulatory influence comes from Underwriters Laboratories, who test products and decide on their suitability (8). In addition, control over electrical materials is

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TABLE 1

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TYPICAL PROPERTIES OF LIQUIDS

	Mineral Oil				Polybutenes			Askarels				
	Uninhibited transf. oil	Capacitor oil	Pipe cable oil	Heavy cable oil	Pipe cable liquid	Paper impregnant	Capacitor liquid					
[illegible]					1,200[a]	8,000[a]	300,000[a]					
[illegible]	58.2[a]	103[a]	763[a]	2365[a]				40-42	44-51	82-92	185-240	1800-2500
[illegible]		38[a]	60[a]	101[a]	63[a]	176[a]	2,200[a]	30-31	31-32	34-35	36-37	44-48
[illegible]												
[illegible]	9.79	21						4.6	6.9	17.2	45.3	46.4
[illegible]		3.5	10	21				<1.8	<1.8	2.5	3.2	6.14
[illegible]	146[b]	154.4[b]	196.1[b]	243.3[b]	154[b]	160[b]	252[b]	146.1[f]	153.3[f]	182.2[f]	192.8[f]	none
[illegible]	0[c]	0[c]	0[c]	0[c]	0.01[i]	0.01[i]	0.01[i]	0.010 max	0.010 max	0.010 max	0.010 max	0.010 max
[illegible]	−45.6[c]	−45.6[c]	−26.1[c]	−17.8[c]	−34[c]	−23[c]	1.7[c]	1.1	−35.5	−19.0	−7.0	10.0
[illegible]	0.898[d,e]	0.907[d]	0.928[d]	0.926[d]	0.862[e]	0.870[e]	0.905[e]					
[illegible]								1.18	1.26	1.38	1.45	1.54
[illegible]	0.00063				0.00078	0.00076		0.00071	0.00073	0.00068	0.00070	0.00066
[illegible] (°C/cm)	0.00031[m]	0.00031[m]	0.00030[m]	0.00030[m]								
[illegible]	0.076[n]	0.076[n]	0.072[n]	0.072[n]		0.062[i]	0.062[i]	0.067	0.063	0.056	0.057	0.054
[illegible]								275.0	290.0	325.0	340.0	365.0
[illegible]	32.5[o]	>30[o]	>30[o]	>30[o]	>35[o,r]	>35[o,r]	>35[o,r]	>35	>35	>35	>35	35
[illegible]								4.5	5.7	5.8	5.6	5.0
[illegible]					2.14[p,q]	2.16[p,q]	2.22[p,q]					
[illegible]	0.001[f,s]	0.001[f,s]	0.001[f,s]	0.001[f,s]	0.0005[f,s]	0.0005[f,s]	0.0005[f,s]	0.001	0.001	0.001	0.001	0.001
[illegible]					>1x10[illegible]	>1x10[illegible]	>1x10[illegible]	>5x10[illegible]	>5x10[illegible]	>5x10[illegible]	>5x10[illegible]	>5x10[illegible]

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TYPICAL PROPERTIES OF LIQUIDS—continued

	Fluorocarbons			Silicones				Organic Esters							
	$(C_3F_6)_nN$	*n = 1/n = 5	$C_4F_{10}O$					Dielectric Grade Castor Oil	Glyceryl Tri-acetoxy-Stearate	Butyl Stearate	Butyl Sebacate	Butyl Naphth-enate	Tetra Hydro-furfuryl Oleate	Ethylene Glycol	Silicate Ester Base Fluid
Viscosity, SUS, 25°C															
37.8°C		—/35			360	720	180			48	46		61		
99-100°C					137.9										
Viscosity, cs, 25°C	2.59		0.82	10.5	100	200	500								
37.8°C	1.80	—/2.7	0.64		80	160	400			7	6		10		12.2
99-100°C	0.48	—/0.88	0.30		29	58	148	98							3.95
Flashpoint, open cup, °C					>30	>315	>325	291		167.8	175.0		203.9	116	187.8
Acidity, mgm KOH/gm															0.15 max
Pour point, °C	—50.0	—154/—84	—100.0	—90	—55	—52.7	—50.0	—23	4.4	21.7	—10.0		—17.8		<—89.5
Specific gravity, 15.6°C	1.89	1.57/1.82	1.79												
25°C	1.88	1.54/1.79	1.77	0.940	0.970	0.971	0.973	0.959	0.995					1.1154	0.887
Coef. of expan., cc/cc/°C	.0012	.00084/.00036	.0016	0.00095	.00097	0.00097	0.00097	0.00066	0.00066					0.00062	.000863
Thermal conduc., (gm-cal/sec)(cm²)(°C/cm)				0.00034		0.00037								.00063	.000327
(BTU/hr)(ft²)(°F/ft)	0.049	0.070/0.049	0.081		0.086	0.087	0.088	0.103	0.103						0.080
Boiling point at 760 mm, °C	177.8	40.8/224.2	102.2											196-206	>371.1
Volatility, weight loss				31%[k]	1%[k]	<2%[k]									
Dielec. strength, kv/.1" (0.254cm)	35 min	28/50	35 min	35[e]	35	35	35								27[e]
Dielectric constant, 60 Hz	1.90	3.02/2.45	1.86	2.7[mn]				3.74[ot]	3.57[ot]	3.3	4.4	3.1	4.3		
10⁶ Hz	1.89	3.02/2.45	1.84		2.75	2.75	2.75								2.65
10⁷ Hz[e]	1.90		1.87	2.7[mn]	2.75	2.75	2.75							41	2.65
Dissipation factor, 60 Hz	<0.0005[e]	<0.00006	<0.0005[e]	.00015[mn]				0.06[e]	0.0097[e]	0.01	0.01	0.016	0.019		
10⁶ Hz	<0.0005	<0.00006	<0.0005		<0.0001	<0.0001	<0.0001								0.0336
10⁷ Hz	<0.0005		<0.0005	.00001[mn]		0.00005				0.00013	0.00015	0.0013	0.00039		0.0042
Volume resistivity, ohm-cm	3×10^{11}	—/>4×10^{14}	6×10^{11}	1×10^{14}[j]	>1×10^{14}[j]	>1×10^{14}	>1×10^{14}	5×10^{14}[m]	6.3×10^{14}[m]	5×10^{14}		1×10^{14}	<1×10^{11}		9×10^{11}

[illegible]CFCF$_2$O$_2$CHFCF$_3$

[illegible]CF$_3$

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a ASTM D446
b ASTM D92
c ASTM D97
d ASTM D1250
e ASTM D877. Suitability of D877 for high viscosity oils such as cable oils and polybutenes listed in table has not been determined.
f ASTM D924
g ASTM D974
h Weight loss on a 15 gm sample in a 50 cm desiccator at 150° for 24 hrs.
i ASTM D664
j ASTM 1169
k Weight loss after 48 hrs. at 200°C
l 100°F
m 0°C
n —8°C
o ASTM D287
p ASTM D257
q 23°C
r 80°C
s 100°C
t Hz unknown
u 100 Hz
v ASTM D149
w 10⁶Hz
x ASTM D150
y Askarels have true flash point.

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Table 2

Physical and Other Properties of Lubricating Oils, Engine Oils, and Hydraulic Fluids

Fluid	Chemical Class or Compound	Viscosity, cs 100°F	Viscosity, cs 210°F	Specific Gravity (Water=1)	Flash Point °F	Fire Point °F	Autoignition/ Temperature °F	Decomposition Temperature °F
			Mineral Oils					
MIL-2190	Mineral Oil	32.2	5.8	0.86	~450	--	665(5)	--
Harmony 44 (Gulf)	" "	87.6	9.8	0.88	~460	--	680(5)	--
MLO-5731	" " - Naphthenic	--	--	--	--	--	--	640(28)
MLO-7277	" " "	--	--	--	--	--	464(50)	725(30)
MLO-60-294	" " - Paraffinic, deep dewaxed	14	3.15	0.88	385	430	700(27)	~620(30)
Mobil DTE-103	Mineral Oil	124	8.74 to 10.2	0.92	390	--	702, 675(50)	--
MIL-H-6083B	" "	--	--	--	255	--	470(50)	--
MIL-H-5606A	" "	--	--	--	--	--	437(4)	--
MIL-O-5606(Esso Univis J-43)	" "	--	--	--	195	225	437(2)	--
			Glycols and Water Glycols					
Ethylene Glycol	Glycol	8.7	--	--	240	--	856(38)	--
Propylene Glycol	"	19.6	--	--	230	235	835(38)	--
Ethylene Glycol + 50% Water	Water-Glycol	2.2	--	--	--	--	903(38)	--
Houghto-Safe 271	" "	~43	~16(150°F)	1.045	--	--	767(5)	--
" " 520	" "	43.2	25.1(130°F)	1.075	--	--	--	--
" " 620	" "	43.2	29.8(150°F)	1.055	--	--	--	--
Hyvac 20(Mobil)	Water-Glycol and additives	41	--	1.07	--	--	750(51)	--
Irus 902 (Shell)	Water-Oil Emulsion	97.4	51	0.93	--	--	709(51)	--
Ucon 50HB-260	Polyalkylene Glycol	56	--	--	455	500	743(38)	--
Ucon 50HB-280-X	" "	--	--	--	500	600	743(20)	--
Ucon LB-60	" "	10.7	--	--	310	325	653(38)	--
Ucon LB-400-X	" "	--	--	--	--	--	752(20)	--
			Phosphate Esters					
Houghto-Safe 1010	Triaryl Phosphate Ester	18.2	3.9	1.20	505	670	>1200(51)	--
" " 1055	" " "	13.0	8.0	1.145	505	680	1020,830(50)	--
" " 1115	" " "	32.2	4.1	1.165	--	680	>1200(51)	--
" " 1120	" " "	49.8	5.0	1.15	485	690	1020(5)	--
" " 1130	" " "	62.8	6.0	1.145	490	680	>1200(51)	--

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Table 2 (Cont)

Fluid	Chemical Class or Compound	Viscosity, cs 100°F	Viscosity, cs 210°F	Specific Gravity (Water=1)	Flash Point °F	Fire Point °F	Autoignition[2] Temperature °F	Decomposition Temperature °F
			Phosphate Esters (Cont)					
MIL-H-19457(Type 1)	Triaryl Phosphate Ester	--	--	--	--	--	1040(19)	--
Tricresyl Phosphate	" " "	38.3	4.48	1.17	470	--	1110(12)	680(48)
Trioctyl Phosphate	Trialkyl Phosphate Ester	--	--	0.926	405	--	545(13)	~380(28)
Trihexyl Phosphate	" " "	--	--	--	--	--	549(12)	--
Pydraul AC	Phosphate Ester-Chlorinated Hydrocarbon	88.8	5.0	1.36	450	745	1148(5)	--
Pydraul F-9	" "	50.9	5.9	1.285	430	675	1100(51)	--
Cellulube 220 (Shell S.F.R.)	Phosphate Ester	43.4	4.9	1.145	455	665	1038(5)	--
Pydraul 150 (Monsanto)	" "	30.>	7.9	1.125	380	470	975(51)	--
Skydrol	" "	--	--	--	360	470	>1300(51)	--
			Mono- and Dibasic Acid Esters					
Plexol 201	Di-2-hexyl Sebacate	12.7	3.31	0.912	420	450	--	--
Plexol 244	Di-isooctyl Adipate	9.64	2.77	0.926	400	445	712(12)	--
Plexol 273	Di-isodecyl Adipate	14.5	3.56	0.920	425	460	--	--
Plexol 79	Polyester	1,250	108	1.023	540	620	--	--
MIL-L-7808 (O-60-18, Esso 4040)	Sebacate-adipate Diester	12.1	3.1	--	437	460	728, 486(7)	490(26)
MIL-L-7808 (H-1026)	Di-2-ethylhexyl Sebacate	12.58	3.3	--	--	--	755(50)	575(15)
MIL-L-97368 (O-60-7, TP-6538)	Trimethylol Propane Ester	15.1	3.4	--	430	475	738, 491(7)	~650(24)
MIL-L-9236 (O-60-27)	" " "	14.8	3.45	--	435	485	--	--
MIL-L-9236 (O-60-23)	" " "	15.99	3.62	--	470	525	--	--
MIL-L-9236 (O-61-17)	" " "	15.78	3.59	--	490	535	--	--
MIL-L-97368	" " "	16	3.2	0.97	425	510	>800(51)	~650(24)
MLO-54-581 (Texaco, TL-2456)	Diester	--	--	--	435	475	734(2)	--
TP 6538 (Hey.Newport)	Trimethylol Propane Ester	--	--	~0.97	--	--	705, 507(50)	--
P/O (Esso 4275)	Polyester	--	8.94	0.951	510	--	711, 500(50)	--
MLO-60-50	Trimethylol Propane Ester	--	--	--	--	--	--	748(30)
Trimethylpropane Tri-pelargonate	" " "	--	--	--	--	--	--	606(26)
			Silanes					
MLO-54-408C	Tetra Dodecyl Silane	34.58	6.37	--	555	625	775(17)	658(15)
MLO-56-280	Diphenyl di-n-dodecyl Silane	37.2	6.2	--	530	580	690(17)	>680(15)
MLO-56-578	Octadecyl trioctyl Silane	27.5	5.76	--	520	590	790(17)	--

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estimated that one-third of the PCBs released to the air and one-half of those released to water have now been degraded. The PCBs in dumps probably have undergone less degradation.

Given the diversity of uses of PCBs and their chemical stability (greater stability in the higher chlorine species), it is not surprising that residues are now widespread. While satisfying quantitative estimates of the contribution of various pathways into the environment are not possible with existing data, there are enough data to be certain that they do reach the environment at least from the following sources:

-- Open burning or incomplete incineration (at usual temperatures) of solid wastes, municipal and industrial. Incineration at 2000° F or above for two seconds will destroy PCBs, but poorly operated incinerators or open burning may result in PCBs being released to the atmosphere unchanged.

-- Vaporization from paints, coatings, plastics, etc. (Nisbet and Sarofim, 3) estimate that as much as 20 percent may be vaporized.

-- Municipal and some industrial sewers (present in treated as well as untreated wastes). Tables 2 and 3.

-- Accidental spills or improper waste disposal practices.

-- Formerly, direct application to the environment as ingredients of pesticides or as carriers for pesticides (such uses are now prohibited).

-- Dumping of sewage sludge, municipal and industrial solid waste, and dredge spoil at sea.

-- Sewage sludges disposed of on land.

-- Migration from surface coatings (paints, etc.) and packaging materials into foods and feeds.

Probably the largest amounts of PCBs circulating in the environment reach it through industrial and municipal discharges to inland and coastal waters. Tables 2, 3, and 4 present data on such discharges. Based on Table 4, we can estimate 6,000 tons per year may reach these environments.

In addition, PCB residues occur in sludge from municipal sewage systems. Table 5 presents results of analyses from several such sludges. Sewage sludge is disposed of by incineration, landfill, spreading on the land, and dumping at sea. Four million tons per year reach the Atlantic Ocean and Gulf of Mexico, which would include only 10 or so tons of PCBs (4). Analysis of the waste water from the effluent scrubbers of three sludge incinerators showed no detectable residues of PCBs (level of sensitivity 0.1 part per billion), suggesting that most PCBs had been destroyed by incineration. The total amount of PCBs contained in the sludges would not be more than ~70 tons per year.

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TABLE 2 - CONCENTRATION OF PCBS IN MUNICIPAL SEWAGE TREATMENT PLANT OUTFALLS

Collection Site	Collection Date	Aroclor Compound Detected	µg/l (ppb)	Flow per Day Mgd 1/	Est. PCB Discharged per day (lbs.)	Source of Data
Ohio - Miami River						
Dayton	1-19-71 0800 to 1-20-71 0800	1254	17	48	6.2	EPA Unpb. Data
Hamilton	1-19-71 0000 to 1-19-71 2400	1248	10	8	0.6	"
Middleton	1-19-71 0800 to 1-20-71 0800	ND*		-	-	"
Wisconsin - Milwaukee River						
West Bend	3-26-70	1254	0.25	1.4	0.002	Veith & Lee
Fredonia	"	1254	0.12	0.1	0.002	"
Saukville	"	1260	0.13	0.1	0.002	"
Grafton	"	1254	0.04	0.8	0.002	"
California						
East Bay (San Francisco)	12-70	1254	3.1-3.8	155	4.2	Schmidt, et al, '71
San Francisco	"	1254	3.8-5.8	31.5	1.2	"
Terminal Island	"	1254	5.8-12.8	9.3	0.7	"
Orange County	"	1254	0.21-0.64	130	0.4	"
Hyperion	"	1254	0.16-0.37	340	0.7	"
White Point	"	1254	76	350	213	"
Richmond		ND*		-	-	
San Diego		ND*		-	-	
Oxnard		ND*		-	-	

*ND - Not Detected

1/ mgd = million gallons per day = (8,000,000 lbs./day)

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TABLE 3. PCB CONCENTRATIONS IN INDUSTRIAL EFFLUENTS

Location	Kind of Industry	Date	Aroclor Compound Detected	Concentration in Effluent (ppb)	Source of Data
Saukville, Wisc.	Chemical Plant	3/70	1242	2.50	Vieth and Lee, 1970
Ohio- Great	Paper Coating Co.	1/71	1242 & 1248	27	EPA data- Analytical Quality Control Lab.
Miami River	Paper Treatment	1/71	1242	430, 470*	"
	Appliance	1/71	1254	5	"
	"	1/71	1254	18	"
Florida- Escambia River	Chemical Plant	4/69-10/69	1254	2.5-275	Duke, et al., 1970

*Samples from treatment lagoon

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TABLE 4

TOTAL ESTIMATED CONTRIBUTION OF PCBS TO THE AQUATIC ENVIRONMENT

Municipal Outfalls

Assume: 150,000,000 sewered population x
130 gallons sewage per person per day x
8 pounds per gallon of sewage =
Approximately 160 billion lbs./day.

With concentration of 0.1 ppb in sewage, 16 lbs./day;
With concentration of 10 ppb in sewage, 1600 lbs./day.
[Note that only the White Point outfall and Dayton (largely serving industrial communities) exceed the 10 ppb concentration]

Annual contribution would not exceed 300 tons from this source, even if the average concentration in municipal sources was 10 ppb.

Industrial and Other Sources

Basis for Estimate:

Concentrations in the Great Miami River of 5.8 ppb and flow rate of 728 cfs yield estimated daily carriage to the Ohio River of ~95 lbs. per day. Contribution from the sewage treatment plants at Dayton (6.2 lbs./day) and Hamilton (0.64 lbs./day) account for 6.8 lbs. (~7% of the amount present in the river).

In the Milwaukee River, the estimated discharge of PCBs to Lake Michigan, based on a concentration in the water of 0.15 ppb (Veith and Lee, 1970) and flow rate of 500 cubic feet per second, is ~ 0.5 lb. per day. Contributions from the sewage treatment plants at West Bend, Fredonia, Saukville, and Grafton, total 0.002 lbs./day, (about 0.4% of the amount present).

Extrapolation:

The Great Miami River drains a heavily industrialized area and represents something near the upper limit of PCBs to be expected in municipal discharges. The Milwaukee River is less industrialized. If one assumes that municipal sources contribute on the average not more than 5% of the total, the annual contribution from other sources would be on the order of 300 x 20 = 6,000 tons per year.

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TABLE 5. CONCENTRATION OF PCBS IN SEWAGE SLUDGES

Collection Site	Date	Aroclor Detected	Concentration (ppb)	Sludge per day tons/day	Est. PCB Content Day (lbs.)	Source
California						
Hyperion (Los Angeles)	12/70	1254	85 (78.5-92.1)	20,000[1/]	3.2	Schmidt, et. al., (13)
Barstow	7/21/71	1254	1400	1.4	.004	EPA Unpublished data
Ohio						
Dayton (Miami River)		1254	105,000	47.9	10.1	EPA Unpublished data
Little Miami (Cincinnati)		1254	32,000	20.2	1.3	" " "
Mill Creek (Cincinnati)		1254	12,700	83.3	2.2	" " "
Lebanon (Turtle Creek)		1254	2,500	1.0	.005	" " "
Shayler Run		1254	3,200	-	-	" " "
Virginia						
Lorton		1254	1,200	-	-	" " "
Indiana						
Indianapolis		1254	3,500	126.1	1.03	" " "

1/ This number is based on outfall disqharge and represents a relatively dilute sludge. The estimated PCB content in lbs./day is the important figure here.

Assumptions: Each million gallons of sewage contains about 1 ton of sludge. The daily output of sludge, then is 150,000,000 sewered population x 130 gal. sewage per day = 19,500,000,000 gallons per day and 19,500 tons of sludge per day.

At 10 ppm of PCBs (highest level found), the daily output would be 19,500 tons x 2000 lbs. = 39 million lbs. x 10 ppm = 390 lbs. per day, at 1 ppm, 39 lbs./day. These would be respectively 70 and 7 tons/year.

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By far the largest quantities of PCBs must end up in dumps and landfills. As noted above, open burning will doubtless release some PCBs to the atmosphere. Little is known about concentrations in landfills, degradation, leaching or vaporization. Much of the material here will be in sealed containers, and will be sequestered or only slowly released to the environment. Analyses of stagnant water close to a Swedish landfill were below the level of detection of 4 parts per billion, suggesting little leaching from such sources (5).

PCBs are known to have reached the environment or man's food supply in a variety of other ways, no one of which is important as a source of environmental contamination, but each of which may have serious consequences locally. Spent transformer oil containing PCBs, used as an herbicide carrier, contaminated dairy cattle grazing area and resulted in residues in milk. Silo paints containing several percent of PCBs are thought to have migrated into silage, and in turn resulted in residues in milk (6). A leak in a heating element used in pasteurizing fish meal resulted in contamination of poultry feed which, in turn, resulted in reduced hatchability of eggs in some chicken flocks, and residues in excess of the FDA interim action level in some eggs and meat (6). In Japan, rice oil contaminated from leakages during heat treatment resulted in poisoning of hundreds of people and the only known disease attributable to PCBs. Residues in packaging materials have contaminated the foods they contained; presumably, most of the residues came from recycled paper which had included "carbonless" carbon paper with relatively high PCB levels. Virgin paper products have also been shown to contain PCBs, and their source of contamination is not well defined at this time. There are other cases of food contamination in which the source of the contaminant has not been identified.

From the sediment measurements, we can crudely estimate the burden in sediments underlying inland waters in the continental United States. There are about 58,000 square miles of such sediments. Assuming sediment has a specific gravity of 2.5; residues are in the top 3 inches; the weight of 3 inches of sediment is $\frac{2.5}{4}$ x 62.4 (wt. of a cubic foot of water) x 43,560 sq. feet/acre ≈ 1,600,000 lbs./acre, or ≈ 1 billion lbs. per square mile (1.6 million x 640). At 10 parts per billion, the PCB burden would be 58,000 sq. miles x 10 lb./sq. mile = 580,000 lbs. At 100 parts per billion, the PCB burden would be 5,800,000 lbs. -- 290 to 2900 tons. The higher estimate is less than 1/10 the amount Nisbet and Sarofim (3) estimate may be present in freshwater and coastal sediments.

I. OCCURRENCE IN THE ENVIRONMENT

Thousands of samples from various parts of the environment have been analyzed for PCBs. Table 6 presents selected data on occurrence in air, water, sediments, and various living organisms.

In air and water away from immediate sources of waste discharge, levels are low--a few nanograms per cubic meter (parts per trillion, ppt) in air and marine waters, and less than a part per billion (ppb) in fresh water; soil or bottom sediments contain a few parts per billion, up to several hundred parts per million (ppm) near some industrial outfalls; from tenths of a parts per million to tens of parts per million in fish and up to hundreds of parts per

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TABLE 6. A SAMPLING OF MEASURED OCCURRENCES OF PCBS IN THE ENVIRONMENT

	Location	No. Samples	Date	Aroclor Compound Detected	Concentration (ppb)	Source of Data	Remarks
Air							
Precipitation	United Kingdom		1968	N.S.	Detected but not quantified	Tarrant and Tatton, 1968	
	Florida		1971	N.S.	Below level of quantification	USGS- Unpbl.	
	Sweden		1970	N.S.	Present in snow	Smithsonian Inst. CFSLP- 1970	
Suspended Particulate	4 U. S. cities		1968-1970	N.S.	~27-230 ppm on suspended solids	EPA- Unpbl.	Quantification questionable
Water							
	Great Miami River, Ohio	19	11/70	1242	ND to 15.8 $\bar{x}$ = 5.7	EPA- Unpbl.	3 samples above Dayton below level of detection. Mean of 16 samples 5.7
	Ohio River	2	11/70		ND	"	
	Big Suamico River, Wisc.		-		<0.01	Veith, 1972	
	Pestigo River, Wisc.		11/70	1254	0.31	"	
			4/71	"	0.38	"	
			Summer '71	"	<0.01	"	
	Oconto River Wisc.		11/70	1254	0.45	"	
			4/71	"	0.16	"	
			Summer '71	"	<0.01	"	
	Milwaukee River	10	1970	1242 1260	0.03-2.07, $\bar{x}$ 0.29 0.02-0.13, $\bar{x}$ 0.08	Veith and Lee, 1970	

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TABLE 6 (Continued)

Location	No. Samples	Date	Aroclor Compound Detected	Concentration (ppb)	Source of Data	Remarks
Water (continued)						
Green Bay, Wisc.		-	1254	0.04-0.07	Veith, 1972	
Lake Michigan		Summer 1970		0.013	Veith, Personal Communication	$\bar{x}$ of 12 samples ½ mi. off 12 river mouths.
South Florida		6-7/71	N.S.	<0.01-0.02	USGS, Unpbl.	
Escambia River & Bay, Fla.		Fall '69	1254	ND to <0.1	Duke, et. al. 1970	
Irish Sea		10/69	N.S.	<0.01	Holdgate, 1970	
Sediments						
11 lakes in Pennsylvania		1971	N.S.	<10-50	USGS, Unpbl.	Data considered good to within ± 50%
7 locations in S.E. Florida		1971	N.S.	10-3200	"	
4 locations in West Central Fla.		1971	N.S.	<10-20	"	
6 locations in S. Florida			N.S.	10-1500	"	
Escambia Bay, Florida		8/69	1254	max. of 486,000	Duke, et al., 1970	Near industrial outfall
		8/69	1254	1700	"	
		8/69	1254	<300	"	6 mi. from outfall

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TABLE 6 (Continued)

	Location	No. Samples	Date	Aroclor Compound Detected	Concentration (ppb)	Source of Data	Remarks
Biota							
Marine Plankton	North Atlantic		1971	N.S.	$\bar{x}$ 200	Harvey in Nesbit & Sarofim, 1972	
Zooplankton	Irish Sea	7	10/69	N.S.	10-30	Holdgate in Nesbit & Sarofim, 1972	
Invertebrates							
Mussels	Irish Sea	(about 200)	10/69	N.S.	50-500	"	
"	Baltic Sea	40	1965-1968	N.S.	4300 (1900-8600)	Jenson, et al., '69	
"	Stockholm Archipelago	15	"		5200 (3400-7000)	"	
Oysters	Escambia Bay, Fla.	18	1971	1254	650 (100-1400)	EPA- Gulf Breeze, Unpbl.	
"	"	2	1970	"	840 (710-970)	"	
"	"	2	1969	"	1050 (1000-1100)	"	
	Florida	8	1964-1970	"	1400-2700	"	
	Georgia	12	1967-1970	"	2000	"	
	S. C.	3	1965-1969		Present but not quantified	"	
Blue Crab	Florida	10	2-3/70		"	"	
Crabs	S. C.		1969	N.S.	<100	Duke, et al., '70	
"	Escambia Bay, Fla.		1969	1254	1000-7000	"	
Shrimp	"		1969	1254	1500-2500	"	
Norway Lobster	Irish Sea	33			10-100	Holdgate 1970 in Nesbit & Sarofim, 1972	

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TABLE 6 (Continued)

Location	No. Samples	Date	Aroclor Compound Detected	Concentration (ppb)	Source of Data	Remarks
Biota (continued)						
Fish						
Marine (species not stated)	North Atlantic		1971	N.S.	100	Harvey in Nisbet & Sarofim, 1972
Herring	Baltic Sea	18	1965-1968	"	6800 (500-2300)	Jensen, et al., '69
"	Stockholm Archipelago	4	"	"	5100 (3300-8500)	"
"	Irish Sea	154	1969	"	10-2000	"
10 species	Bay of Fundy Gulf of Me.		1971	1254-1260	70-1540	Zitko, et al., 1972
Estuarine						
Menhaden	Florida		8/70	1254	6700	EPA- Gulf Breeze Lab., Unpubl.
"	"		10/69	1254	7300	"
Freshwater						

Nationwide fish-pesticide monitoring

1969. At least some fish at 47 of 50 sampling stations contained more than 100 ppb of PCBs (the lower limit of detection). Levels ranged from <100 to 14,800 ppb. Henderson, et al., 1971.

1970. Only 1 fish of 40 sampled contained less than 1000 ppb. The highest 20 fish averaged 36,000 ppb, ranging from 9600 to 213,000 ppb. Fish from the Ohio River and the Hudson River contained the highest residues-- 4 fish from the Ohio averaged 80,000 ppb; one fish from the Hudson River contained 213,000 ppb. Other waters in the top half included the Yazoo River, Allegheny River, Delaware River, Cape Fear River, Mississippi River, Missouri River, and Lake Ontario. (Stalling and Mayer, 1972)

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TABLE 6 (Continued)

	Location	No. Samples	Date	Aroclor Compound Detected	Concentration (ppb)	Source of Data	Remarks
Biota (continued)							
Birds - Land							
Starlings	Continental U. S.	124	11-12/70	N.S.	$\bar{x}$ 660 (50-24,300)	Bureau of Sport Fisheries & Wildl., Unpubl.	
Woodcock	N.S. - Northern U. S.		Fall 1971	N.S.	4000-9000	"	
Bald Eagle	25 states	69	1966-68	N.S.	Not quantified	"	
Birds - Water							
Guillemot Eggs	Baltic Sea	9	5/68		250,000	Jensen, et al., 1970 (22)	
White-tailed Eagle	Stockholm Archipelago	4	3/65-6/66		14,000,000, 8,400,000-17,000,000	"	
Heron	"	4	4/67		9,400,000	Jensen, et al., 1969 (19)	
Double-crested Comorant (eggs)	Bay of Fundy		1971?	1254	17,200	Zicko, et al., 1972 (20)	
Abdominal fat			"	1254	52,000	"	
Herring Gull Fat	Bay of Fundy		"	1254	75,000	"	

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TABLE 6 (Continued)

	Location	No. Samples	Date	Aroclor Compound Detected	Concentration (ppb)	Source of Data	Remarks
Biota (continued)							
Mammals - Sea							
Gray Seal	Baltic Sea	2	9-11/68	N.S.	30,000	Jensen, et al., 1969 (19)	
Gray Seal	Stockholm Archipelago	3	5/68	N.S.	30,000	"	
Man							
Plasma	South Carolina	723	1968	1254-1260	43% of samples up to 29	Finklea, et al., 1971 (23)	
Milk	Sweden	22			$\bar{x}$ 40	" Westoo, et al., 1970 (24)	
	California	-			$\bar{x}$ 60	Risebrough & Brodine, 1969 (25)	
	Germany	-			$\bar{x}$ 100	Acker and Schulte, 1971 (26)	
Fat	Germany	-			5700	"	
	United States	235			N.D.	Yobs, 1972 (27)	
		229			<1000		
		188			1000-2000		
		36			>2000		

— Unknown
N.S. Not specified
N.D. Not detected

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million in some fish and birds near the top of the food chain (1/8 of an inch is about one trillionth of the distance to the moon; and a part per million is about 5 steps on a walk from Washington to San Francisco). Man, who is also at the top of a food chain, carries residues ranging up to 2 parts per million or occasionally more.

II. BEHAVIOR IN THE ENVIRONMENT

A. Air - The relative importance of the atmosphere as a transport mechanism is not known. While PCBs have been identified in air, the residence time, transformations, and movement from air to land or water surfaces through fallout or rainout, or return to the atmosphere are virtually unknown. There are at least two observations that suggest substantial aerial transport; data on residues in fish in Lake Minto in a remote part of northern Quebec (7), and residues in woodcock which feed almost exclusively on earthworms, which in turn pick up residues from the soil. Only by invoking aerial transport can we account for residues in arctic lakes or in more or less wilderness areas of the North where woodcock summer. Nisbet and Sarofim (3) suggest airborne PCBs will have been adsorbed on particles and have a relatively short residence time--thus most will have been redeposited on the U. S. continent, but some will have reached the oceans.

B. Water and Sediment - The water environment is probably the principal sink and transport mechanism for PCBs. Calculations based on measured occurrences in municipal and industrial outfalls, in the receiving waters, and the downstream reaches of the waterways demonstrate transport through the aquatic system. Measured residues in fishes from various environments suggest accumulations at the downstream ends of the drainageways.

There are few data on removal, disappearance, and sequestering of the substances in soils or bottom sediments of rivers, lakes, estuaries, or the ocean. Table 6 includes some data that indicate presence in bottom sediments, and suggest that sediments may be a major reservoir of PCB residues. Work by Nimmo and his colleagues at Gulf Breeze (8) has shown that at least pink shrimp and fiddler crabs are able to take up PCB residues from this source. Fiddler crabs and pink shrimp exposed to clean flowing sea water in aquaria containing sandy silt with initial residues of 61 parts per million (dry weight basis) of Aroclor 1254 accumulated an average of 80 $\pm$ 25 parts per million in the whole crab and 240 parts per million (one pooled sample) in the hepatopancreas of the shrimp. Accumulation was much less, 17 $\pm$ 9 parts per million and 6.1 parts per million, respectively, with silt initially containing 30 parts per million. Some accumulation took place 3.2 $\pm$ 0.9 parts per million (in crabs) and 1.1 parts per million (shrimp hepatopancreas) from silt initially containing 2.5 parts per million. The same investigators have shown transfer of residues from sediments to overlying water. Effluent water from the aquarium with 61 parts per million Aroclor in sandy silt contained 3.5 parts per billion; from the aquarium with 30.0 parts per million in silt, effluent water contained 0.5 parts per billion.

III. EXPOSURE AND BIOLOGICAL ACCUMULATION

Experimental work on biological accumulation by individual species of vertebrates and invertebrates has been conducted by a number of laboratories in the United States and elsewhere. Table 7 presents selected data that demonstrate accumulation factors of up to 75,000 in whole organisms, and in the

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TABLE 7. ACCUMULATION OF PCBS BY VARIOUS AQUATIC ORGANISMS

Species	Aroclor Compound	Exp. Time	Environmental Concentration (ppb)	Residue (ppb)	Concentration Factor	Source
Catfish	1248	60 da.	13.3	958,000	72,000	Stalling and Mayer, '72(7)
	1254	60	4.1	312,000	76,000	"
Bluegill	1248	60	4.9	312,000	63,700	"
	1254	60	6.8	87,000	12,800	"
Fiddler Crab	1254	30	3.5	80,000	22,900	"
			0.5	17,000	34,000	"
Pink Shrimp	1254	30	3.5	240,000	69,000	"
			0.5	6,100	12,000	"

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hepatopancreas of pink shrimp. The data reported in the preceding section demonstrate that pink shrimp are able to accumulate PCB residues from environmental levels as low as 0.5 parts per billion; and Table 7 shows accumulation by fish from levels as low as 1 part per billion.

Evidence from environmental samples (Table 6) suggests uptake of PCB residues from exceedingly low environmental concentrations. Thus the plankton samples from the North Atlantic contain something like 200 parts per billion, yet PCB levels in marine waters are believed to be exceedingly low--certainly less than 0.01 parts per billion.

PCBs, like many of the organic insecticides, are fat soluble, and are stored in the lipids of animals. Like the insecticides, they resist metabolic changes, and tend to be concentrated (at least to some extent) at succeedingly higher levels as they pass through various steps in the food chain.

The data presented in Table 6 suggest that there are two components of movement through the biota. One is the familiar pattern of food chain accumulations. The other involves direct uptake from the environment by various trophic levels; e.g., soil to earthworms; water to phytoplankton, zooplankton, larger invertebrates, and fish. The fish and plankton data from the North Atlantic reported by Nisbet and Sarofim (3) are consistent with the hypothesis, in that plankton residue levels are higher than fish levels, suggesting direct accumulation rather than food chain transfers. So, too, is the evidence from feeding studies reported by Stalling and Mayer (9) that suggest accumulations of no more than a factor of 2 over dietary intake levels in fish. It seems reasonable that food chain transfers are the principal route of accumulation in warm blooded vertebrates, and possibly in the highest levels of carnivorous fish. Conversely, direct environmental uptake is probably the most important for aquatic invertebrates and fish.

That humans are exposed to PCBs is evident from the data in Table 6. There are a number of possible routes: air, water, food. Fish and shellfish through uptake from water would be expected to provide the principal continuing source in the diet. Sampling of fish to keep those containing more than the FDA interim action level from the market is carried out by FDA and the various States where fish are known to have substantial PCB contamination. Other foods have been found to contain PCB residues, but these residues are for the most part traceable to accidents; e.g., residues in eggs and poultry whose diet included fishmeal contaminated by leakage of PCBs from processing equipment (Monsanto reports they no longer sell PCBs for this purpose); residues in milk traceable to silage stored in silos painted with PCB-containing paints; residues in dry food packaged in PCB-contaminated packaging materials. Aside from the occurrences traceable to incidents such as the above, residues have been found in only 479 of 15,000 food samples by FDA during November 1969-June 1971 (200 of the 479 were followups on samples found to contain PCBs). In the FDA diet studies during FY 70 and 71, only 22 of 720 composite samples contained PCB residues. It is clear that only a small fraction of the U. S. food supply contains detectable levels of PCBs.

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Other routes by which man may be exposed, or may have been exposed, include breathing of PCB-containing house dust and dermal uptake from carbonless copy paper or PCB-containing inks. One sample of house dust from Michigan was reported to contain 200 parts per million of PCBs (10). Carbonless "carbon" paper was reported to contain 2-6 percent of PCB Kanechlor 300 (11); experiments with this paper demonstrated that PCBs rubbed off when handled, and that even after washing the hands, approximately two-thirds of the PCBs still remained on the skin. Dermal uptake through intact skin has been demonstrated in the rabbit, guinea pig, and rat, and presumably would occur in man.

IV. DISCUSSION

PCBs occur widely in the environment. It seems reasonable that by far the largest amount is present in dumps and landfills where it is thought to be more or less sequestered from the rest of the environment.

It is clear from environmental samples that smaller but significant amounts are present in the terrestrial and aquatic environments. Much smaller total amounts are present in the biota, but levels in some organisms in some places are sufficiently high to cause undesirable biological effects. The residues present in soils and bottom sediments are potentially available for transfer to the biota, directly or through movement to water and uptake by aquatic organisms.

A relatively small amount of PCBs is required to contaminate a large part of the biota (for example, 10 tons would be sufficient to contaminate the entire U. S. population to a level of 1 part per million). Organisms can accumulate residues from remarkably low environmental concentrations. PCBs have been shown to accumulate in fish and aquatic invertebrates to levels of 75,000 times that present in the water (up to 200,000 times in selected tissues), and to be accumulated from concentrations as low as 0.06 parts per billion. Thus, to prevent levels in fish from reaching the 5 parts per million established by FDA as the interim action level for fish as human food, concentrations in water would have to be less than 0.07 parts per billion, or, to allow some safety factor, 0.01 parts per billion. This level should be sufficiently low that fish and shell fish would not be adversely affected.

Monsanto has taken a number of voluntary steps to reduce the amounts of PCBs reaching the environment--both through restricting sales for such uses and by providing a disposal service for its customers. (During the first year after this service was announced, more than 500,000 pounds accumulated awaiting disposal in the 10,000,000 pounds-per-year incinerator being constructed (3). Despite these steps, substantial amounts of PCBs are still reaching the environment. They are present in detectable levels in virtually all municipal sewage effluents. Effluents from many industrial plants such as paper mills, appliance manufacturers, electrical equipment manufacturers, and chemical industries, included higher levels than municipal sewage. As the restrictions continue, a decrease in residues should occur through gradual exhaustion of existing stocks and uses.

When the restrictions become fully effective, the principal source to the aquatic environment can be expected to be that part of the electric equipment manufacturing industry and the electric utility industry that require these valuable materials because of their fire resistance and dielectric properties.

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The effluents from such installations should be regulated and closely monitored to assure that no more than 0.01 parts per billion results in the receiving water. (So, too, should the manufacturing and disposal facilities of Monsanto.) Good housekeeping should permit this level to be achieved. An educational campaign aimed at users to assure proper disposal will also be necessary--so long as the materials are referred to as transformer oils, cutting oils, hydraulic oils or fluids, etc., care in disposal will be hard to assure. After all, oils in moderate quantities aren't regarded as troublesome substances.

V. RESEARCH NEEDS AND OPPORTUNITIES

1. Data provided by Kuratsune and Masuda (11) suggest that PCB-containing carbonless copy paper may have been an important source of PCB residues in man. An epidemiological study, involving a group of regular users of such copy paper (airline ticket salesmen; clerical workers; etc.), would shed much light on this question.

2. The detection of high levels of PCBs in house dust by Price (10) suggests that inhalation may be an important source of PCB levels in man. Both the question of occurrence in dust and of respiratory uptake from such dust should be explored.

3. Residues of PCBs have not been determined in soils, though by inference they must be present. A set of samples from the program of pesticides monitoring in soils should be analyzed for PCBs; the set should be drawn with care to illuminate distribution patterns in, near, and remote from industrial areas.

4. Data on pesticides in air are unsatisfactory. A few samples, including both vapor and particulates, should be collected from industrial areas and analyzed. If the methodology proves satisfactory, a small-scale survey should be undertaken to determine the importance of the atmosphere as a transport mechanism.

5. The presumption that PCBs do not move to ground water should be tested. The volume of water in this reservoir, coupled with its relatively long residence time, suggests that even very low levels of contamination may be significant.

6. Dumps and landfills are thought to be the principal reservoir of PCBs, but there are virtually no data on behavior of PCBs in these locations. Small-scale sampling should be undertaken to determine the concentrations brought to dumps, the fate of PCBs from open burning, and into leachate and gases from sanitary landfills. Degradation in place should also be investigated.

7. The presumption that submerged sediments contain a large amount of PCBs should be examined. Such questions as vertical distribution, degradation, movement, transfer of water, should be explored as well as the current distribution of residues beneath inland and inshore marine waters.

8. The reported finding of PCB residues on the order of 0.2 parts per million in marine plankton of the mid-North Atlantic requires elaboration.

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PCB content of plankton from equal volumes of water in inshore and mid-ocean waters, as well as sampling of the surface film in both areas, would be informative.

9. Carbon filter samples going back to the mid-1960's are available from some fresh water locations. If a satisfactory analytical method can be worked out, samples from a few locations with continuous records should be analysed to provide some data on trends that might be useful for correlation with manufacturing data. Fish samples should be considered for the same purpose.

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APPENDIX E

Occurrence And Sources Of PCBs In Food

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APPENDIX E

Occurrence and Sources of PCBs in Food

Residues of polychlorinated biphenyls (PCBs) as potential food contaminants were first identified by(Jensen, 1) in fish from various Swedish waters. Other early interests in the occurrence of PCB residues in foods were directed to PCB interferences in the determination of organochlorine pesticide residues, particularly DDT and its analogs. (Widmarck, 2).

In 1967 the Food and Drug Administration initiated a methods development project to determine the analytical behavior of PCBs and to devise a means of separating PCBs from chlorinated pesticides in the regulatory analysis of food. Experiments demonstrated that PCBs are recovered and detected by the FDA methodology routinely employed for multiple residues of organochlorine pesticides at a sensitivity of detection approximately one-tenth that for p,p'DDT. For example: using FDA's standardized procedures the limit of detectability for p,p'DDT in butterfat is about 0.15 parts per million, while for PCBs it is about 1.5 parts per million. Further investigation led to a procedure,(Armour and Burke, 3) that separates PCBs from organochlorine pesticides after initial sample extraction and cleanup, and permitting separate determinations of the groups of chemicals. This procedure is designed for use with the FDA multiple pesticide residue procedure as the basic analytical method. The analytical method, along with the PCB-separation step, has application to a wide variety of food and animal feed commodities and many types of environmental substrates.

In July 1969, FDA field laboratories were provided with the analytical instructions for separating and determining PCB residues in foods. Since all objective food samples collected under FDA's pesticide surveillance programs are examined for chlorinated pesticide residues, the advances in analytical methodology facilitated analysis for PCBs in food. The food surveillance programs formally incorporated examination of all samples for PCBs and reporting of results in November 1969. The United States Department of Agriculture incorporated similar procedures in its sampling programs for meat and poultry in January 1971.

The objective sampling programs represent the basic Federal activity for monitoring the Nation's food and feed supply for pesticide residues (Duggan and Cook, 4). The main components of these programs are as follows:

I. FDA PESTICIDE SURVEILLANCE PROGRAM

This program is designed to gather information on the extent of pesticide (including PCBs) contamination of foods and feeds on a geographical basis through the use of a statistical sampling plan. Samples are collected at shipping points to facilitate compliance action when illegal residues or potential residue problems are encountered. FDA field offices select commodities grown or processed within their areas. Sampling is based on possible residue problems, volume of production, and other related factors. Commodities may include fresh fruits and vegetables, dairy products, shell eggs, grains, fish, animal feeds, and processed foods. The limits of detectability as applied to objective samples is about 0.3 - 0.5 parts per million PCBs in non-fatty foods and about 1.5 parts per million PCBs in the fat for fatty foods.

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II. FDA TOTAL DIET STUDIES

The total diet program is designed to determine the levels of pesticides, PCBs, and trace heavy metals in the dietary intake on a geographical and seasonal basis. Market basket samples, representing the two-week diet of a 15-20 year old male -- which is approximately twice that of the normal diet -- are collected at retail stores, bimonthly, in five regions of the United States. Food items are cooked or prepared for table-ready use by dieticians and are divided into 12 food class composites, such as dairy products; meat, fish and poultry; and leafy vegetables. Each composite is analyzed for a variety of chemical contaminants, including PCBs. The limit of detectability as applied to total diet composites is approximately 0.05 parts per million PCBs. When abnormally high residues are detected in any composite, follow-up analysis is made of the individual food commodities of the composite to determine which food is contributing the excessive residues and to determine whether compliance action is warranted.

III. USDA SAMPLING PROGRAMS

The Consumer and Marketing Service, USDA, has primary responsibility for sampling and analyzing meats, poultry, and broken egg products for pesticide, environmental (PCB), and other chemical or biological contaminants. The USDA program involves all federally inspected slaughtering plants (about 1,200) and egg breaking establishments (about 140). The instructions for sampling request that the agricultural producer of the animal, bird, or eggs, be named so that the State of origin will be known. This facilitates follow-up if a violative sample is found and identifies those samples which originate from the same farm. Inspectors are instructed to collect objective samples from different agricultural producers. The time of sampling and the plant location are determined on a random basis, by computer, for this program.

IV. OTHER REGULATORY PROGRAMS

In addition to these routine sampling activities, FDA and USDA learn of PCB contamination of foods in other ways. As part of their enforcement responsibilities, they conduct in-plant establishment inspections; conduct special investigations and surveys to determine the cause and extent of specific PCB problems; and maintain close contact with State officials and industry who also monitor the occurrence of chemical contaminants in food.

When required, there is also a selective phase to these sampling programs and investigations. Selective sampling is used to determine the extent of violations when a violative sample is encountered or a report of possible harmful residues is received by the responsible agency. An increased number of samples is taken in the suspected area to determine

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the extent of the problem, and the degree of regulatory control required to safeguard the wholesomeness of the food supply and to correct the problem. The selective program has been used to control specific PCB incidents.

V. SOURCES OF CONTAMINATION

Because of their widespread industrial applications, their chemical stability and persistence, and their ubiquitous presence in the ecosystem, it is not unexpected that PCB residues have been detected in a variety of food commodities. Several sources of contamination have been identified. These can be generally divided into three categories:

1. Environmental contamination - background levels of PCBs in fish from contaminated lakes and streams.
2. Industrial accidents - isolated incidents involving direct leakage and spillage or contact of PCB fluids and other PCB containing materials on animal feeds, feed ingredients or food.
3. Food packaging materials - PCB migration to foods packaged in PCB contaminated paper products. Identification of these categories is not intended to imply that all positive PCB findings in food and other materials have been successfully traced to any one of the three sources. Samples have been reported where the cause of the PCB residue was not clearly defined, and one could only speculate as to the source of the residue.

Since examination for PCB residues was incorporated into the routine sampling programs of FDA and USDA, thousands of objective samples have been analyzed. Additionally, numerous investigative surveys and selective samplings have been conducted to respond to specific "accidents" and other problems associated with PCBs in food.

VI. RESULTS OF SURVEILLANCE SAMPLING PROGRAMS

Since November 1969, FDA has analyzed for PCB residues all raw agricultural commodities and other food classes sampled under its pesticide surveillance program. More than 15,000 sample examinations had been completed as of June 1971. A total of 279 of the objective samples was reported to contain PCBs (Table 1). An additional 200 food samples, collected as follow-up samples of suspected lots because previous analyses indicated a potential problem, were also found to contain PCB residues (Table 2).

PCBs were encountered most frequently in fish, with 317 of the total positive samples (479). The levels in fish reported in Tables 1 and 2 indicate that most residues are between 1 and 10 parts per million. The Department of the Interior and others have gathered extensive amounts of data on PCB residues in fish, and it appears that the occurrence of PCB residues in fresh water fish is widespread geographically. Residue levels are related to location (highest in waters near industrial and metropolitan centers) and the species of fish (high or low fat, and feeding habits). In order to control the interstate shipment of fish containing excessive residues of PCBs, FDA (February 1970) established

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TABLE 1

POSITIVE ANALYSES OF VARIOUS FOOD SAMPLES
November 1969 through June 1971
(15,000 Samples Analyzed --all prior to
East Coast Terminal Fish Meal Incident)
Number Positive Samples (Range-ppm) Average ppm

Examining District	Fish	Fish By Product	Cheese[a]	Milk[a]	Shell Eggs	Potato By Product	Oysters	Misc.	Total Positive All Products
Los Angeles	23(0.1-2.60).62	5(0.2-5.0)2.10		6(0.15-0.30).19	1(0.03).03				35
Minneapolis	2(.12-35.29)17.70								2
New Orleans	23(T-1.70)0.4[d]							Milled Rice 1(0.20)	24
New York	3(0.21-4.78)2.49								3
San Francisco					1(0.50).50				1
Seattle	1(0.40).40					8(0.10-4.20)1.16		Straw 1(0.30) Wheat 1(0.32)	11
Kansas City	19(0.60-13.30)3.61								19
Atlanta	1(0.53).53[e]		1(T)	2(0.94-0.97).96				Silage 1(0.82) Silo Coating 1(11.3)	6
Baltimore				22(T-12.58)2.69	5(T-T).00		12(T-T).00	Crabmeat 1(0.24)	40
Boston	1(6.08)6.08								1
Buffalo	19(T-13.00)3.28[d]	1(T).00							20
Chicago				1(0.23)0.23					1
Cincinnati	55(0.04-4.70)1.77			4(0.22-0.24).24					59
Denver						2(0.15-1.12).64			2
Detroit	49(0.25-15.5)2.96			4(0.49-1.86)1.27				Cat Food 1(6.49) Duck 1(0.70)	55
Dallas									0
Total Positive	196	6	1	39	7	10	12	8	279

[a]ppm fat basis [d]T=Trace; not incl. in ave. [e]marine species

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TABLE 2 POSITIVE FOLLOW-UP INVESTIGATIONAL SAMPLES collected because of suspected PCB Residues November 1969 through June 1971

Number Positive Samples (Range-ppm) Average-ppm

Examining District	Fish	Fish By Product	Cheese[a]	Milk[a]	Shell Eggs	Potato By Product	Oysters	Misc.	Total Positive All Products
Los Angeles	31(0.1-6.0)1.54		43 (.03-1.0).30[c]						74
Minneapolis	58(T-9.37)1.97[d]								58
New Orleans	4(0.26-2.0)1.03							Boiled Oyster 1(0.23)	5
New York								Frog Legs 1(0.09)	1
San Francisco									0
Seattle	1(1.98)1.98					2(1.39-1.73)1.56			3
Kansas City	1(8.39)8.39								1
Atlanta	2(0.43-1.23).83[e]			2(6.60-22.80)14.7					4
Baltimore	5(T-T).00			13(0.57-9.96)4.03	10(T-.01).01			Apple Pomace 1(T)	29
Boston									0
Buffalo	3(1.51-2.29)1.95								3
Chicago				6(0.14-0.24).19					6
Cincinnati	3(1.00-1.50)1.21								3
Denver	3(0.32-0.77).54[e]								3
Detroit	10(0.73-4.70)2.47								10
Dallas									0
Total Positive	121	0	43	21	10	2	0	3	200

[a]ppm fat basis [c]Import [d]Greece;not incl. in ave. [e]Marine species

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an interim action level of 5 parts per million in the edible portion of the fish.

The surveillance data for milk and manufactured dairy products also show this class of food to have a relatively significant incidence of positive findings. For the most part, the PCB sources were traceable to localized "industrial accidents" which are described elsewhere in this report and were not always present as environmental contaminants. The occurrence of PCBs in milk does not appear to be widespread nationally.

Although no PCBs were found in fresh fruits or vegetables, residues were reported in potato by-products used for animal feed. The source of contamination in this case has not been established.

The FDA total diet studies for Fiscal Years 1970, 1971, and 1972 (first-half only) included PCB analysis for 75 market baskets representing 900 composite samples. These analyses showed 54 composite samples to contain PCB residues ranging from a trace to 0.36 parts per million (Table 3). Twenty-six of the positive samples were present in the meat, fish, and poultry composite, and nineteen in the grain and cereal composite. Findings in other food class composites were at less than 0.05 parts per million, with no consistent pattern of PCB occurrence. These studies indicate the dietary intake of PCBs is of a low order. Expressed as mg/kg body weight/day, the PCB level for FY 70 was less than 0.0001 and 0.0001 in FY 71 and FY 72.

The objective phase of the USDA sampling program for meat and poultry during 1971 detected PCBs in 164 of the 4,175 samples analysed. There were 25 samples positive in the 2,404 samples collected from cattle, swine, calves, and sheep with a range in the positive results from .1 parts per million to 3.0 parts per million. The poultry samples reflected the contaminated feed supply in the Southeastern United States and in the 1,804 samples analysed, there were 140 positive with an analytical range of 1 parts per million to over 15 parts per million. The three poultry classes, young chickens, fowl, and turkeys, all had between 7 - 9 percent samples positive for PCBs with a range of 1 parts per million. Only two samples were over guidelines established by FDA.

VII. INDUSTRIAL ACCIDENTS

The following isolated incidents of avoidable PCB-contamination of food led to the actions described to remove the contaminated food from the market.

A. POULTRY

1. New York State Incident. The Campbell Soup Company, Camden, New Jersey, noted in December 1970 surveillance data excessive PCB residues in chickens grown in New York State. The State of New York placed a quarantine on the three counties involved and pretesting of all fowl was mandatory before slaughter.

Analysis indicated PCBs in poultry fat varying from a non-detectable level to 26.8 parts per million. FDA advised the State of New York and USDA on February 1, 1971, that FDA would not object to the

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distribution of poultry containing less than 5 parts per million. This level was applicable to the edible tissue on a whole tissue basis or to the separate fat removed during slaughter or processing and intended for use as a food or feed ingredient.

The regulatory control action extended from December 1970 until August 1971, when all samples submitted for testing prior to slaughter were found to be below the 5 parts per million guideline. To support this control activity and determine disposition of the fowl scheduled for slaughter the following samples were analyzed for PCBs: 1,566 dozen eggs, 198 feed, and 5,790 chicken samples. Most of the analytical work was done in the New York State chemical laboratory. On the results of these samples, 140,450 chickens were killed on the farm and buried, 75,740 chickens were passed for restricted slaughter, and 409,000 chickens were released for normal slaughter.

The alleged source of the PCBs in this incident is believed by State officials to be plastic bakery wrappers. Bakery goods were used as a feed ingredient for the poultry and the plastic wrappers which may have contained high PCB levels were ground with the bakery goods.

2. East Coast Terminal Incident (FDA Actions). The Monsanto Chemical Company informed FDA in July 1971 that large amounts of fish meal might have been contaminated with Aroclor 1242 leaking from a heating system during pasteurization of fish meal at East Coast Terminal, Wilmington, North Carolina. Aroclor 1242 was used as the heat exchange fluid. FDA inspection revealed PCB contamination of processed fish meal on hand at the firm. Investigation indicated the leak began in April 1971 and continued through July.

The fish meal on the premises was embargoed and the firm initiated a voluntary recall of fish meal processed since April 1971. An estimated 12,000 tons were distributed. Over 2,000 tons were recalled. Individual fish meal samples examined contained from 14 to 30 parts per million PCB.

FDA also initiated follow-up sampling of fish feeds, catfish from fish farms, and eggs when the contaminated fish meal was implicated. USDA was informed when investigation indicated eggs were being distributed to commercial egg breakers. As of September 1971, 224 samples of eggs had been analyzed with 71 containing residues in excess of 0.5 parts per million.

FDA seized 3 lots of eggs. The samples representing these lots contained from 0.7 to 1.9 parts per million PCB.

FDA seized 5 shipments of fish feeds that were manufactured from contaminated fish meal. These seizures were in the States of Louisiana, Georgia, and Mississippi, and were on feeds that contained from 0.6 to 4.5 parts per million PCB. In addition, a shipment of the contaminated fish meal from East Coast Terminal that had not been recalled was seized. The analysis of this seized product showed levels in excess of 350 parts per million PCB. Catfish sampled from commercial fish farms contained less than 3.0 parts per million PCB.

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3. East Coast Terminal Incident (USDA Actions). The Meat and Poultry Inspection Program, USDA, was notified by Holly Farms, Wilkesboro, North Carolina (July 1971) that poor hatchability had alerted them to a problem in their poultry operation. The cause was PCBs in the poultry ration. The contaminated feed ingredient was the fish meal from one supplier in North Carolina. The USDA objective surveillance program had recent reports of PCBs in poultry fat from the southeastern United States

The Food and Drug Administration informed USDA of the confirmed PCB contamination problem on July 19, 1971. The 5 parts per million guideline for poultry was reaffirmed.

The list of primary consignees, received from FDA, showed the contaminated fish meal had been distributed throughout 10 southeastern States.

USDA required all poultry coming to market from this area to be either pretested or tested after slaughter before marketing. This program is continuing with owner's certification now being accepted since the source and feed distribution channels have been identified.

The Poultry Division examined 900 lots of broken eggs, and 123,750 lbs. of product were removed from the market. The Meat and Poultry Inspection Program has examined samples from over 5,174 flocks of broilers, fowl, and turkeys during this time. One producer had to destroy over 88,000 broilers.

The present trend in PCB levels is downward, and the contamination is subsiding. Therefore, an effort is being made by USDA to test all remaining flocks that were exposed to the contaminated feed so that the pretesting requirement can be discontinued. Any chicken or turkey flocks identified as having violative levels of PCBs by this final testing program will remain under surveillance until disposed of by slaughter.

4. Minnesota Incident. USDA notified FDA in August 1971 that USDA and Swift and Company had found excessive PCB levels in turkeys.

Investigation indicated that the PCB residues found in the turkeys were caused by the feed. In addition, it appeared that the fat used as an ingredient of the turkey feed was the source of the PCB. The suspect fat used by the feed mill was received from a processor in Minnesota. It was found that the fat being manufactured at that time contained negligible levels of PCB. The source of the PCB levels found in the turkeys was not established.

USDA surveyed turkey flocks for PCBs in the immediate geographic area, which included parts of Minnesota, North Dakota, and South Dakota. There were 140 flocks tested, and no residues of PCBs were found above 2 parts per million (fat basis) except in the original grower's flocks, where PCBs were present at levels up to 20 parts per million (fat basis).

This one grower has been required to pretest all flocks prior to slaughter. Today the PCB level (fat basis) is approximately 1 part per

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million. Approximately 1 million turkeys approaching market weight were withheld from market until residue levels were reduced to less than 5 parts per million.

5. Oklahoma Incident. On August 20, 1971, USDA informed FDA of excessive PCB findings in chickens in Mississippi during routine sampling. Investigation revealed that the birds came from a grower in Oklahoma and the feed from a mill also in Oklahoma. FDA analysis of eggs and feeds from these firms showed no PCBs.

6. California Incident. USDA examined turkeys after slaughter in warehouse storage in California. PCBs were found in the amount of 1.41 to 28.0 parts per million in the fat tissue. There were 100,000 pounds of turkeys detained until testing was completed. The turkeys had originated from flocks raised in four counties in California. The source of the PCBs could not be determined.

B. MEAT BY-PRODUCTS

National By-Products, Inc., Mason City, Illinois Incident - On July 28, 1971, FDA inspection of this firm revealed that PCBs were used in heat treatment equipment. Sampling showed the pasteurized meat meal to contain PCBs. The firm initiated recall of the contaminated product.

C. MILK

1. West Virginia Incident. In July 1969, FDA's Baltimore District found PCBs in milk samples collected in the routine food surveillance program. Baltimore District investigated possible routes of contamination, and by February 1970, the investigation pointed to spent transformer fluid used as a vehicle for herbicide sprayed along power right-of-ways in the Martinsburg, West Virginia area. Through this route, PCBs contaminated dairy cattle grazing areas. The dairy farms involved were taken off production by State officials.

2. Ohio Incident. In April 1970, the State of Ohio notified FDA's Cincinnati District of unidentifiable residues in milk. FDA identified the residues as PCBs and advised the State of a guideline of 0.2 parts per million (whole milk). The State of Ohio and FDA investigated the problem and determined that the dairy farms were using a PCB-containing sealant in silos that migrated to the silage. The State of Ohio banned milk from some producers and destroyed an undetermined amount of milk.

3. Florida-Georgia Incidents. The States of Florida and Georgia reported findings of PCBs in milk to FDA's Atlanta District in August 1970. A PCB-containing sealant in silos was found to be the source of contamination in this incident. FDA found approximately 11 percent PCB in the silo coating.

VIII. PAPER FOOD PACKAGING

FDA first learned of the PCB food packaging problem in July 1971. The total diet market basket samples showed low level PCB residue in a grain and cereal composite (Table 3). The PCB was traced to the Shredded Wheat

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TABLE 3 - SUMMARY OF PCB FINDINGS IN FDA TOTAL DIET SAMPLES

		FY 1970		FY 1971		1/2 FY 1972	
		30 Market Baskets 360 Composites		30 Market Baskets 360 Composites		15 Market Baskets 180 Composites	
	Food Class Composites	Positive	Range (PPM)	Positive	Range (PPM)	Positive	Range (PPM)
I	Dairy Products	1	T [a]	-	-	1	T
II	Meat, Fish, Poultry	3	0.02-0.03	14	T-0.15	9	T-0.08
III	Grain and Cereal Products	- [b]	-	4	T-0.36	15	T-0.10
IV	Potatoes	-	-	-	-	1	T
V	Leafy Vegetables	-	-	-	-	-	-
VI	Legume Vegetables	-	-	-	-	-	-
VII	Root Vegetables	-	-	-	-	-	-
VIII	Garden Fruits	-	-	-	-	1	T
IX	Fruits	-	-	-	-	-	-
X	Oils, Fats & Shortening	-	-	-	-	2	T
XI	Sugar & Adjuncts	1	0.08	-	-	2	T
XII	Beverages	-	-	-	-	-	-
TOTALS		5		18		31	

a. Trace (generally less than 0.05 ppm PCB)
b. None detected

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packaging material was identified as the source of PCB in the food. The manufacturer of the packaging material used about 95 percent recycled paper to manufacture paperboard containers. FDA analysis of different types of the firm's paperboard showed PCB levels ranging from about 2 to 433 parts per million. Various types of packaged food products, some of which used this firm's paperboard, were also analyzed as part of FDA's investigation. Nine samples of the 28 packaged foods examined contained PCBs in the food portion.

As a result of this limited investigation, FDA initiated a nationwide survey in September 1971, to determine the extent of the PCB food packaging problem. The survey included analysis for PCBs in all paper packaging components and the packaged food portions of 15 different representative food categories. This survey was completed in late December 1971. A detailed statistical analysis of the results of the survey is currently being compiled. Sixty-seven percent of the packaging portion of the samples contained PCB residues as high as 338 parts per million; 19 percent of the food portions of the samples contained PCB residues, with an average PCB concentration of 0.1 parts per million. The maximum PCB level found in food was 5 parts per million.

The FDA survey, as well as other studies by the paper and food industries, show a significant correlation between the presence of PCB residues in the food component and the packaging component. The mechanism of PCB migration from the packaging to the food probably occurs through both the vapor phase and abrasion or physical contact. The level of PCB contamination is dependent upon many factors -- levels of PCBs in the packaging materials, type of food, length and conditions of storage, and others. The extent of migration of PCB from paperboard packaging to the food contents is being investigated (Trout, 5). There is also significant correlation between the presence of PCB residues in packaging and the presence of recycled paper components in the packaging. The occurrence of PCBs in recycled paper materials is attributed primarily to the recycling of the so-called "carbonless" carbon paper (contains 3-5 percent PCB -- use of PCBs for this purpose has been discontinued) and to a lesser degree, the use of certain printing inks. PCB residues also were found in some packaging components that appeared to be composed entirely of virgin paper material. This source of PCBs probably occurs in the packaging manufacturing processes.

Industry has taken steps to reduce the levels of PCBs in food packaging materials by avoiding the recycling of carbonless carbon paper. Although it is not know if this practice has been instituted industry wide, data provided by the Grocery Manufacturers of America, Inc., does reflect a change. For example: recycled board manufactured during the period June 1970 - January 1972 shows that only 18 percent of the samples contained less than 5 parts per million PCB; the same type of recycled board manufactured from November 1971 through January 1972 shows that 95 percent of the samples to be below 5 parts per million PCB.

IX. SPECIAL SURVEYS

FDA is currently conducting a national survey to determine the extent and levels to which complete animal feeds are contaminated with PCBs.

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The survey covers feeds for cattle, sheep, swine, chickens, turkeys, and other miscellaneous animals. The survey results available to date show that less than 5 percent of the complete animal feeds sampled contain PCBs. Levels range from non-detectable (less than 0.1 parts per million) to a maximum level of 0.6 parts per million PCB.

Another investigation survey was recently initiated by FDA to survey the milk supply on a State by State basis to determine the extent of PCB contamination of milk intended for bottling and manufacturing use. Results from this survey are not available at this time.

The results of tissue sample studies of several domestic animals and poultry are shown in Table 4.

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TABLE 4 - OBJECTIVE SAMPLES - CY 1971
for PCB's

SUMMARY
PPM - fat Basis

ANIMAL AND POULTRY TISSUE CY 1971

Class	N.D.	.01-.1	.11-.50	.51-1.5	1.51-3.0	3.01-5.0	5.01-7.01	7.01-15	Over 15
Animals	2379	1	9	11	4				
Poultry	1664	4	25	53	23	4	5	15	11

PCB RESIDUES IN ANIMAL AND POULTRY
TISSUES COLLECTED IN OBJECTIVE PHASE
DURING CY 1971

Animal or Poultry	Number of Samples Analyzed	Number of Samples with A Residue	Percent of Samples with A Residue	Number of Samples Exceeding Guidelines	Percent of Samples Exceeding Guidelines
Cattle	722	9	1.2	0	0.0
Calves	66	4	6.1	0	0.0
Swine	1436	7	0.5	0	0.0
Sheep	180	5	2.8	0	0.0
Young Chickens	1637	127	7.8	2	0.1
Mature Chickens	69	5	7.2	0	0.0
Turkeys	88	8	9.0	0	0.0
Ducks	10	0	0.0	0	0.0
	4208	165	3.9	2	0.04

0062011

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FOOTNOTES

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2. Widmark, G. (1967). J. AOAC 50, 1069.

3. Armour, J. A. and Burke, J. A. (1970). J. AOAC 53, 762-768.

4. Duggan, R. E. and Cook, H. R. (1971). Pesticide Monitoring Journal 5, 37-43.

5. Trout, P. E. (1971). Grocery Manufacturers of America "PCB Workshop", November 17, 1971.

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APPENDIX F

Human Directed Aspects of PCBs

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APPENDIX F

Human Directed Aspects of PCBs

I. INTRODUCTION

The salient aspects dealing with the chemistry and toxicology of the polychlorinated biphenyls have been summarized previously in a number of Status Reports of the FDA (Kolbye, 1; Burke and Fitzhugh, 2; Cook, 3). Additional literature reviews have stressed the chemical and biological aspects of the PCBs (Peakall and Lincer, 4; National Swedish Environment Protection Board, 5; Reynolds, 6; Zitko and Choi, 7; and Fishbein, 8).

The major objectives of this report are to review the current status of the toxicologic (acute and chronic), carcinogenic, teratogenic, mutagenic, metabolic, biological, and epidemiological aspects of the PCBs that are of greatest relevance to man and, based on the above, to cite areas of suggested future research that will more definitively relate to the human aspects of the PCBs.

II. TOXICOLOGICAL ASPECTS

A. HUMAN EPISODES

Compared to the chlorinated hydrocarbon pesticides, definitive aspects of acute, sub-acute and chronic toxicity still remain rather poorly known. Chloracnegen effects were reported as early as 1936, following industrial exposure to the PCBs,(Schwartz, 9, 10, 11; Jones and Alden, 12; Meigs, et al., 13). Occupational chloracne, however, has not been a problem with recent usage of the PCBs. Approximately 10 cases of fatal intoxication involving persons who handled or were exposed to chlorinated biphenyls or naphthalenes in their occupations have been described (Flinn and Jarvik, 14; Greenburg, et al., 15; and Drinker, et al., 16). In all cases histological examination revealed liver fatty degeneration, necroses and cirrhosis. It is important to note that chlorinated naphthalene (as well as chlorinated dibenzofurans) have been recently identified in two commercial polychlorinated biphenyl samples (Phenoclor DP 6 and Clophen A60),(Vos, et al., 17).

Human intoxication with the heat exchanger Kanachlor 400, a Japanese manufactured PCB with 48 percent chlorine and containing as its main components, 2,4,3',4'-, 2,5,3',4'-, 2,3,4,4'- and 3,4,3',4'-tetrachlorobiphenyl, and 2,3,4,3',4'-pentachlorobiphenyl,(Saeki, et al., 18), was reported in Western Japan in 1968.

More than 600 people were eventually affected following the consumption of contaminated rice oil containing levels estimated to range from 2,000 to 3,000 parts per million PCBs (average of about 2,000 parts per million). These levels were derived from the known organic chlorine content of the rice oil related to the known organic chlorine content of Kanachlor 400. Exposure levels to the oil were calculated to approximate 15,000 milligrams per day. The lowest reported figures allow an estimate of a minimal positive effect level at 3 milligrams PCB per day over several months. However, the average doses associated with significant disease in the "Yusho" incident were much higher and were in the range of 30 milligrams/day. (Kolbye, 1).

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The clinical aspects associated with "Yusho" included: chloracne, blindness, systemic gastrointestinal symptoms with jaundice, edema and abdominal pain. Chloracne is very persistent with some patients showing evidence of it after three years. Table 1 lists the subjective symptoms of 89 male and 100 female Yusho patients.

New-born infants born from poisoned mothers had skin discoloration due to the presence of PCB via placental passage. (The dark skin discoloration regressed after a period of 2-5 months). Gingival hyperplasia with pigmentation was seen in several cases. Decreased birth weights were also noted but no evidence could be obtained in regard to the possible retardation in physical and mental activities of the babies (Yamaguchi, et al., 19). The skin of stillborn infants showed hyperkeratosis and atrophy of the epidermis and cystic dilation of the hair follicles. Residues of PCB have been found in fetal tissue (Kojima et al., 20 ; Inagami et al., 21).

The components of Kaneclor 400 with longer retention times have been detected in sputa and fatty tissues of patients (Kojima, 22). Serum triglyceride levels were higher than 300 mg/ml in 60 out of 396 subjects investigated before the end of 1969 (Uzawa et al., 23, 24). Among the incidence of hyperglyceridemia (triglyceride>300 mg/ml) of the six decade groups (age 0-9, 10-19, 20-29, 30-39, 40-49, 50-), that of the first decade group was the highest while that of the third decade was the lowest. The mechanism of the hyperglyceridemia was suggested to be possibly due to an observed decrease in post-heparin lipolytic activity and impaired plasma triglyceride removal (Uzawa et al., 24). Cholesterol and phospholipid concentrations were increased while lecithincholesterol acyl transferase activity was decreased. In female patients plasma lipoprotein lipase activity was decreased (Uzawa et al., 24; Nagai et al., 25). Nearly 40 percent of the examined patients exhibited an elevated excretion of steroids with the 17-ketosteroids (androsterone, etiocholanolone and dehydroepiandrosterone) found to increase in males and to decrease in females (Nagai et al., 25).

Examination of autopsy tissues of two Yusho fatalities revealed the presence of chlorobiphenyls in all of examined organs, especially mesenterial fatty tissues, skin and bone marrow (Kikuchi, et al., 26). Kojima, et al.,(20) found PCBs with longer retention times (probably pentachloro- and higher chlorinated biphenyls) in autopsy tissues and it was assumed that their presence might have been responsible for the observed long duration of the intoxication symptoms.

B. ANIMAL TOXICITY

Early studies of acute oral, dermal and vapor exposure of the polychlorinated biphenyls have involved in many cases mixtures or compounds of undefined specifications and hence have been difficult to interpret unambiguously. Tables 2-4 describe the oral, dermal and vapor exposure toxicity involving a number of chlorinated biphenyls and the results would suggest that the toxicity of these compounds is proportional to their degree of chlorination. These investigations have primarily focused on liver damage. The toxicity of 11 Aroclors in terms of oral LD_{50} (rats) and skin MLD (rabbits) is summarized in Table 5.

a. Sublethal and acute effects: Analogously with the chlorinated hydrocarbon pesticides, the most important effects are long-range sublethal effects. The pathologic changes in various organs are summarized in Table 6 illustrating some interesting differences between mammals and birds. For example, the most

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TABLE 1.

SUBJECTIVE SYMPTOMS COMPLAINED BY YUSHO PATIENTS[a,b].

Symptom	Male %	Female %
Dark brown pigmentation of nails	83.1	75.0
Distinction of hair follicles	64.0	56.0
Increased sweating at palms	50.6	55.0
Acnelike skin eruptions	87.6	82.0
Red plaques on limbs	20.2	16.0
Itching	42.7	52.0
Pigmentation of skin	75.3	72.0
Swelling of limbs	20.2	41.0
Stiffened sole and palm	24.7	29.0
Pigmented mucous membrane	56.2	47.0
Increased eye discharge	88.8	83.0
Hyperaemia of conjunctiva	70.8	71.0
Transient visual disturbance	56.2	55.0
Jaundice	11.2	11.0
Swelling of upper eyelids	71.9	74.0
Feeling of weakness	58.4	52.0
Numbness in limbs	32.6	39.0
Fever	16.9	19.0
Hearing difficulties	18.0	19.0
Spasm of limbs	7.9	8.0
Headache	30.3	39.0
Vomiting	23.6	28.0
Diarrhea	19.1	17.0

[a]Eighty-nine male and 100 female patients diagnosed before October 31, 1968 were examined.

[b]From a report of "Yusho, A Poisoning Caused by Rice Oil Contaminated with Chlorobiphenyls", Kuratsune, M., Yoshimura, T., Matsuzaka, J. and Yamaguchi, A, Fukuoka Acta Med., 60[6] (1969) 513.

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TABLE 2.

ORAL TOXICITY OF CHLORINATED BIPHENYLS.

Chlorine %	Chlorine atoms	Animal	Dose	Effect
42	3	Guinea Pig	69 mg/animal 2 doses 1 week apart	Death in 11-29 days. Liver damage. (Miller, 1944) (53)
42	3	Rat	139 mg/animal 25 daily doses	All animals survived. Liver damages. (Miller, 1944) (53)
65	7	Rat	50 mg/animal every second day	50% dead within 35 days. Severe liver damages. (Bennett, et al., 1938).(54)

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TABLE 3.

DERMAL TOXICITY OF CHLORINATED BIPHENYLS.

Chlorine		Animal	Dose	Effect
%	atoms			
42	3	Guinea pig	34.5 mg/daily over 11 days	All animals died within 21 days. Liver damage. Miller, 1944) (53)
42	3	Rat	34.5 mg/daily over 25 days	All survived. Minor liver changes. Skin affected. (Miller, 1944) (53)
42	3	Rabbit	86 mg/day with 2-day intervals for 7 applications and 172 mg/day with 2-day intervals for 8 applications	All died between 17 and 98 days. Liver damages more pronounced than in rat or guinea pig. (Miller, 1944) (53)

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TABLE 4

VAPOR EXPOSURE TOXICITY OF CHLORINATED BIPHENYLS.

Chlorine %	Chlorine atoms	Concentration mg/m3	Concentration ppm	Daily exposure time (hours)	Exposure number	Exposure period (Days)	Symptoms
42	3	8.6	830	7	17	24	No effect on cats, rabbits, rats and mice. Poor growth in guinea pigs. (Treon, 1956) (55)
42	3	6.83	660	7	84	122	No effects on animals as above, Treon, 1956) (55)
54.3	5	5.4	410	7	83	121	Liver cell injury. Increased liver weight in the rat. (Treon, 1956) (55)
54.3	5	1.5	115	7	150	213	Histological changes in the liver in the rat. (Treon, 1956) (55)
65	7	0.57 0.93		16 8		37-134 42-143	Advancing liver damage. (Bennett, et al., 1938). (54)

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striking findings in mammals are alterations to the liver, whereas fluid in the pericardial sac, kidney damage and reduced spleen are found in birds.

The FDA has conducted a subacute feeding study (lasting up to 90 days) in rats with dosage levels of 25, 75, 150, 300 and 500 parts per million of Aroclors 1254 and 1260 and including sacrifices at 5, 15, 30, 60 and 90 days (Burke and Fitzhugh, 2). Liver weight to body weight ratios increased at all levels. Dose-related increases were also found in liver aniline hydroxylase and nitroreductase activity at all levels with the magnitude of the increases approximately the same at all time levels for both Aroclors. Aroclors 1254 and 1260, in contrast to many chlorinated hydrocarbon pesticides, did not stimulate liver aliesterase activity.

(Nishizumi, 27) studied the effects on mouse and monkey liver of chlorinated biphenyls (48 percent chlorine, equivalent to three to four atoms of chlorine per molecule, with a trace (0.01 percent) naphthalenes. Groups of 30 female mice were given a dosage level of 0.2 ml rice bran oil containing 1600 parts per million or 0.5 percent PCB in olive oil by stomach tube each day for 4 to 26 weeks resulting in marked liver enlargement. (Light microscopy revealed only slight liver changes, but electron microscopy disclosed marked alterations in the liver cells.) A similar study with eight monkeys (5 cynomolgus and 3 squirrel) given chlorinated biphenyls in dosage levels of 1.4 to 16 mg/day in their diet for 40 to 48 days showed both liver cell enlargement and fatty degeneration. The major abnormality reported for the administration of chlorinated biphenyls to mice and monkeys was an increase in the smooth endoplasmic reticulum in the liver cells.

The metabolism and distribution of Aroclor 1254 in normal and carbon tetrachloride-treated Wistar strain rats have been studied by (Grant, et al., 28). Residues of Aroclor 1254 (following oral administration of a 500 mg per ml corn oil solution of Aroclor at 500 mg/kg) were found in all tissues analyzed with fat and blood having the greatest and least concentration, respectively (Table 7).

Aroclor 1254 was found to potentiate the toxicity of carbon tetrachloride in a manner similar to that reported for DDT (McLean and McLean, 1966; Cawthorne, et al., 1970). The studies (Grant, et al., 28) suggest that the liver is the main site of Aroclor 1254 metabolism since rats with carbon tetrachloride damaged livers were unable to metabolize this mixture of chlorinated biphenyls as rapidly as rats with normal livers. Aroclor 1254 significantly increased the size of the liver and also the percent lipid in the liver. The same study revealed that the components of Aroclor 1254 with the shorter GLC retention times, presumably with the lowest chlorine content (Bagley, et al., 29), were metabolized to a greater degree than those with the longer retention times. This effect is in agreement with the studies of Phenochlor DP6 fed to Japanese quail (Koeman, et al., 30).

Yoshimura and co-workers, (31) studied the effects of a single 2.0 mg/mouse dose of polychlorinated biphenyls of the KC-400 type. The concentration of PCB in the skin one day after ingestion was twice as high as that in the liver. Although tetrachlorobiphenyls were almost completely eliminated from the tissues in 3-4 weeks, small amounts of penta- and hexachlorobiphenyls were still detectable after 9-10 weeks.

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TABLE 5.
TOXICITY OF AROCLORS*.

	Aroclors										
	1221	1232	1242	1248	1260	1262	1268	4465	5442	5460	2565
Oral LD_{50} mg/Kg (rats)	3980[a]	4470[a]	8650[a]	11,000[a]	10,000[b]	11,300[b]	10,900[b]	16,000[b]	10,600[b]	19,200[c]	6,310[c]
Skin MLD mg/Kg (rabbits)	>2000[a]	>1260[a]	>794[a]	>794[a]	>1260[b]	>1260[b]		>2000[b]	>1260[b]	>7940[c]	>2000[c]
	<3169[a]	<2000[a]	<1269[a]	>1269[a]	<2000[b]	<3160[b]	>2500[c]	<3160[b]	<2000[b]		<3160

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[a]Undiluted.

[b]Administered as 50% solution in corn oil.

[c]Administered as 33.3% solution in corn oil.

*FDA Status Report on the Chemistry and Toxicology of Polychlorinated Biphenyls (PCB) or Aroclors as of June 1, 1970.

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TABLE 6.

PATHOLOGIC CHANGES INDUCED BY PCBs.

Treatment	Animal	Liver	Kidney	Pericardium & Peritoneum	Other Observable Changes	References
Single oral dose of 69 mg (42% Cl)	Guinea Pig Rat Rabbit	Small droplets through lobules, slight to moderate central atrophy, focal necrosis noted in a few animals	Essentially normal	No noteworthy changes	Adrenals, spleen, & pancreas showed no noteworthy changes.	Miller, 1944 (53)
300 mg daily for 6 days (65% Cl)	Rat	Cells swollen, hyaline granules present, most died within few days.				Bennett, et al., 1938 (54)
50 mg daily for up to 6 months (65% Cl)	Rat	Enlarged (33% weight increase), large number of hyaline globules in cytoplasm. Several died during experiment.				Bennett, et al., 1948 (54)
25,50 & 100 ppm in diet for 15 days (21-68% Cl Aroclors)	Rat	Increase in weight, effect increasing with increasing chlorine content. Aroclor 1232-10%, 1242-12%, 1254-14%, 1268-24% at 50 ppm.				Street, et al 1969 (56)
100 ppm in diet 200 ppm in diet 400 ppm in diet 800 ppm in diet (Aroclor 1242)	Chicken	No effect No effect Enlarged & Mottled Damaged	 Damaged	 Slight Hydropericardium Hydropericardium Hydropericardium, hydroperitoneum, Enlarged		McCune, et. al., 1962 (57)

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TABLE 6. (Cont'd.)

PATHOLOGIC CHANGES INDUCED BY PCBs.

Treatment	Animal	Liver	Kidney	Pericardium & Peritoneum	Other Observable Changes	References
200 & 400 ppm in diet for 3 weeks (42%, Aroclor	Chicken	No changes noted	Paleness at 200 ppm, extensive hemorrhage, and enlargment at 400 ppm	Increased fluid in pericardial sac at the higher concentration.	Paleness of pancreas, enlargment of adrenal and small apleen at low concentrations. At higher concentrations pale cream-colored pancreas, adrenals hemorrhagic.	Flick, et al., 1965 (58)
Various doses (54% Cl, Aroclor)	Bengalese Finch	No weight changes	Weight was 32.4% of brain weight for controls and 53.5% for those dying from PCB poisoning.	Slight weight increase, a few showed liquid in pericardial sac.		Presst, et. al., 1970 (59)
400 ppm in diet for 60 days (60% Cl[a])	Chicken	Centrolobular necrosis (compd. 1 & 2). Liver weight increased from 2.76 g/100g to 4.31g/100g (compd. 3). Fatty degeneration.	Tubular dilatation, compd. 1 & 2). Rare with compd. 3.	Hydropericardium common with compds. 1 & 2. Rare with compd. 3.	Increased porphyria, spleen small with reduction of red pulp and atrophy of white pulp (compd. 1 & 2). Spleen decreased from 0.14g/100g to 0.136g/100g. (compd. 3).	Vos and Koeman, 1970 (42)

[a]Phenoclor DP 6 (compd. 1). Clophen A60 (compd. 2) and Aroclor 1260 (compd. 3) were used. Differential effects noted under compd. numbers. All chickens died on compd. 1 and 2 within 60 days; only 15% mortality on compd. 3.

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TABLE 7.

RESIDUES IN TISSUES OF RATS ORALLY DOSED WITH AROCLOR 1254 (500 mg/kg)*.

	Residue found (ppm, wet tissue)			
Group	1	2	3	4
Blood	1.96[a] ± 0.23[b]	0.42 ± 0.07	3.85 ± 0.46	0.25[c]
Testes	19.22 ± 0.59	4.30 ± 0.44	33.18 ± 1.35	5.62
Heart	24.16 ± 2.84	5.83 ± 0.53	62.40 ± 4.37	6.17
Spleen	29.17 ± 3.44	5.82 ± 1.17	36.60 ± 4.39	—
Kidney	31.14 ± 2.09	11.20 ± 1.76	57.38 ± 3.91	11.08
Brain	39.98 ± 5.91	4.01 ± 0.31	41.91 ± 3.30	5.96
Liver	115.66 ± 10.55	18.85 ± 1.65	796.47 ± 64.96	18.79
Liver[d]	1868.14 ± 166.63	—	6137.64 ± 556.06	—
Fat	996.16 ± 98.58	672.66 ± 155.12	900.46 ± 106.16	1149.05

[a]Mean of five values.
[b]Standard error of the mean.
[c]Single value.
[d]Ppm on a fat basis.

*Grant, D. L., Phillips, W. E. J. and Villeneuve, D. C., Bull. Env. Contam. Toxicol., 6 (1971) 102. (34)

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Street and co-workers (32) studied the effects of diets of 50 parts per million to 100 parts per million of 10 Aroclors ranging in chlorine content and 21 percent to 68 percent fed to rats for 15 days. Their effect on sleeping time induced by hexobarbital, in vitro rates of aniline hydroxylation and demethylation of p-nitroanisole, and the rate of excretion were all found to be increased with increasing chlorine content. Aroclor 1221 (50 parts per million) reduced hexobarbital sleeping time by 11 percent, whereas for Aroclor 1248 and 1268 the figures were 35 percent and 48 percent, respectively. Liver weights were also found to increase with increasing chlorine content of the Aroclors. The storage of dieldrin was decreased in relationship to the chlorine content. For example, with Aroclors containing 60 percent chlorine, or more, the storage in adipose tissue was reduced to the levels found in untreated control animals. The induction of PCBs of hepatic microsomal hydroxylating enzymes has been demonstrated in the American kestrel (Peakall and Lincer, 4), and pigeons (Risebrough, et al., 33).

Fujita, et al. (66) studied the enhancement of the liver drug-metabolizing enzyme system in the rat by several individual chlorinated biphenyls. The most potent inducer was 2,3', 4,4', 5-pentachlorobiphenyl and the effect induced with a single dose of 3,3',4,4'-tetrachlorobiphenyl was found to persist for 6 weeks.

Villeneuve, et al. (34) studied the effects of PCB administration on microsomal enzyme activity in pregnant rabbits. The no-effect level of Aroclor 1254 for enzyme induction in the pregnant rabbit is between 1.0 and 10 mg/kg body weight when administered for 28 days during gestation. Aroclor 1221 did not induce any enzyme activity in the dose, fetus or placenta, so its no-effect level must be considered higher than that for Aroclor 1254. Placental transfer was shown to occur for both Aroclor 1254 and 1221 but does not cause any changes in the biochemical or physiological parameters measured, e.g., total amount of Vitamin A stored per liver, protein levels, aniline hydroxylase enzyme activity, serum cholesterol, no effect in reproductive processes. The drug-metabolizing enzymes aniline hydroxylase and aminopyrine-n-demethylase were both induced by 10 mg/kg Aroclor 1254.

Ito and co-workers (35) found that the administration of PCBs to rabbits increases the total lipid, triglyceride, and cholesterol content of liver and decreases the total liver phospholipid content. (The concentration of serum triglycerides was abnormally increased).

Kimbrough (36) described some aspects of the toxicity of Aroclor 1254 and 1260 in Sherman strain rats. The acute oral LD_{50} for Aroclors 1254 and 1260 in adult rats was greater than 4 and 10 grams/kg, respectively. The salient features of the subacute feeding and reproduction study in both male and female rats can be summarized as follows:

1. Feeding 500 parts per million of Aroclor 1254 and 1260, respectively, to both male and female rats over a period of about 240 days resulted in less weight gain as well as lowered hematocrit.

2. Some male rats fed Aroclor 1254 at a dietary level of 100 parts per million (approximately 5 mg/kg per day) developed porphyria. Porphyria was also found in female rats fed Aroclor 1254 at the 20 parts per million level and in both male and female rats fed 100 parts per million Aroclor 1260.

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3. Liver changes found with both Aroclor 1254 and 1260 include accumulation of fat, inclusions within the liver cells and so-called margination (also found with chlorinated hydrocarbons).

4. Male rats fed Aroclor 1260 at a dietary level of 100 parts per million on up had larger and heavier livers. In the females the liver weights were not significantly increased at any of the dietary levels that were tested.

5. Adenofibrosis was found in: (a) 1/10 males and 6/10 females at the dosage level of 100 parts per million Aroclor 1254 in the diet for about 8 months; (b) in 1/10 female rats at 100 parts per million Aroclor 1260 in the diet for about 8 months; and (c) in several male rats at 1000 parts per million Aroclor 1260 in the diet for 8 months (none at 500 parts per million). The adenofibrosis consisted of fibrosis in the livers concomitant with a proliferation of cells which is considered by some to represent proliferated bile ducts.

6. Two bladder cancers in rats fed 100 parts per million of Aroclor 1260 were also found and the significance of this finding has not been fully assessed at this time.

7. A definite effect on reproduction was produced by feeding 100 parts per million of Aroclor 1254. The first breeding was performed after 76 days on the treated diets and resulted in less offspring. The offspring at weaning were smaller and the survival was decreased compared to control animals. An increase in the liver weight in the F_{2a} generation of the weanlings at a dietary level of 20 parts per million Aroclor 1254 was also found.

8. A dosage of Aroclor 1260 equivalent to 100 mg/Kg/day during the 7th and 15th days of pregnancy reduced the survival and number of young.

Curley and co-workers (37, 38) studied the distribution and storage of PCBs in body fluids and tissues of Sherman rats following: (a) a single oral dose; (b) repeated dietary intake: and (c) after discontinuance. The salient findings of this study could be summarized as follows:

(1) Following acute or chronic administration of Aroclor 1254 or 1260, residue amounts can be detected in all body tissues, fluids and excrement.

(2) Pronounced changes in the gas chromatograms or mass spectroscopic total ion current traces between the Aroclor standards and components observed in the fat and urine suggest possible metabolism or differential absorption.

(3) Rats given single oral doses of Aroclor 1254 at 1600 mg/Kg or of Aroclor 1260 at 3200 mg/Kg had essentially the same levels of residue in their tissues after 24 hours (although individual variation was high).

(4) At the same dosage level rats store more PCBs than DDT.

(5) No significant differences were apparent in the storage of PCBs by male or female rats when fed the same dietary levels with PCBs stored primarily in adipose tissues.

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(6) Toxicity of the Aroclors apparently increases with decrease in chlorine content suggesting the enhanced reactivity and/or less stability of some isomeric chlorobiphenyl in the mixture.

(7) Rats fed Aroclor 1254 at 100 parts per million for 58 days and untreated food for 71 days showed gradually increasing residues during dosage while the excretion trend was quite erratic.

(8) Aroclor 1254 administered at a dietary dosage of 1000 parts per million for 98 days resulted in deaths of 9 of 10 male rats and 8 of 10 females with deaths commencing on the 35th day. Although the survivors appeared normal, there was no weight gain after 77-84 days.

Table 8 illustrates the storage of Aroclors 1254 and 1260 in plasma and tissues after oral ingestion by stomach tube at levels of 1600 mg/Kg and 3200 mg/Kg, respectively. Table 9 depicts the distribution of PCB-derived material following 98-day exposure to a dietary level of 1000 parts per million of Aroclor 1254.

Figure 1 shows the storage (mean conc. vs. time) of PCB-derived material in tissues and plasma. Figure 2 shows the excretion of PCB and PCB (mean conc. vs. time) derived material in feces and urine.

The distribution of PCB in rats dosed with PCBs has been studied with the aid of X-ray fluorescence analysis. PCB residues were found mainly in the skin, and to a lesser degree in muscles, intestines, livers, pancreas and lungs, and the GLC patterns of PCB varied from tissue-to-tissue (Sekita et al., 39).

The intestinal absorption in the rat of a number of isomeric chlorobiphenyls has been studied by Albro and Fishbein (40). PCBs having from one to six chlorine atoms per molecule were very well absorbed and/or metabolized when the compounds were fed in a single dose between 5 and 100 mg/Kg body weight. No significant difference in excretion rates (e.g., retentions) were observed over the molecular weight range 116.5 to 289, suggesting that diffusion may not be rate-limiting for the uptake of these compounds. (When similar tests were performed using aliphatic hydrocarbons, a clear relationship between percentage excreted in the feces and molecular weight was observed). Less than 10 percent of the amount fed were excreted in the feces (over a 4-day period) indicating a high degree of absorption and/or metabolism of these compounds.

b. Long-term studies: The chronic PCB studies of Monsanto with Aroclors 1242, 1254, and 1260, e.g., two-year albino rat and beagle dog, three-year rat reproduction as well as the twenty-five week chicken, have been completed and a copy of the results sent to the FDA. As of this writing, the full report has not been evaluated. However, Dr. Elmer Wheeler of Monsanto has verbally touched on the highlights of these studies supplementing information previously reported in the FDA Status Reports of the PCBs of June, 1970, and December, 1970, as well as reported by Keplinger and co-workers (41) and discussed with the Interdepartmental PCB-Task Force at a meeting in Washington on September 15, 1971. These results can be briefly summarized as follows:

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TABLE 8.

STORAGE OF AROCLORS (IN PPM) 24-HOURS AFTER ORAL INGESTION BY STOMACH TUBE.

	Aroclor 1254 (dosage level - 1600 mg/kg)	Aroclor 1260 (dosage level - 3200 mg/kg)
Plasma		
Mean	24.03	15.79
SE	± 8.08	± 0.99
Brain		
Mean	138.00	145.17
SE	± 36.41	± 23.50
Fat		
Mean	1146.9	930.0
SE	± 574.5	± 426.6
Liver		
Mean	141.20	236.1
SE	± 47.80	± 116.3
Kidney		
Mean	274.03	328.5
SE	± 30.47	± 114.9
Lung		
Mean	65.91	105.17
SE	± 29.48	± 21.13
Muscle		
Mean	80.29	37.00
SE	± 67.58	± 22.37

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TABLE 9.

DISTRIBUTION OF PCB-DERIVED MATERIAL FOLLOWING 98-DAY EXPOSURE TO A DIETARY LEVEL OF 1000 PPM AROCLOR 1254.

	Males (ppm)	Females (ppm)	Males vs. Females
Plasma			
Mean	17	18	p >0.50
SE ±	6	5	
Fat			
Mean	11278	8431	p >0.50
SE ±	5742	579	
Muscle			
Mean	155	753	p >0.20
SE ±	117	721	
Lung			
Mean	78	52	p >0.50
SE ±	23	20	
Brain			
Mean	111	94	p >0.50
SE ±	14	21	
Kidney			
Mean	56	54	p >0.50
SE ±	9	24	
Liver			
Mean	155	210	p >0.20
SE ±	30	13	

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Figure 1. Storage of PCB-Derived Material in Tissues and Plasma.

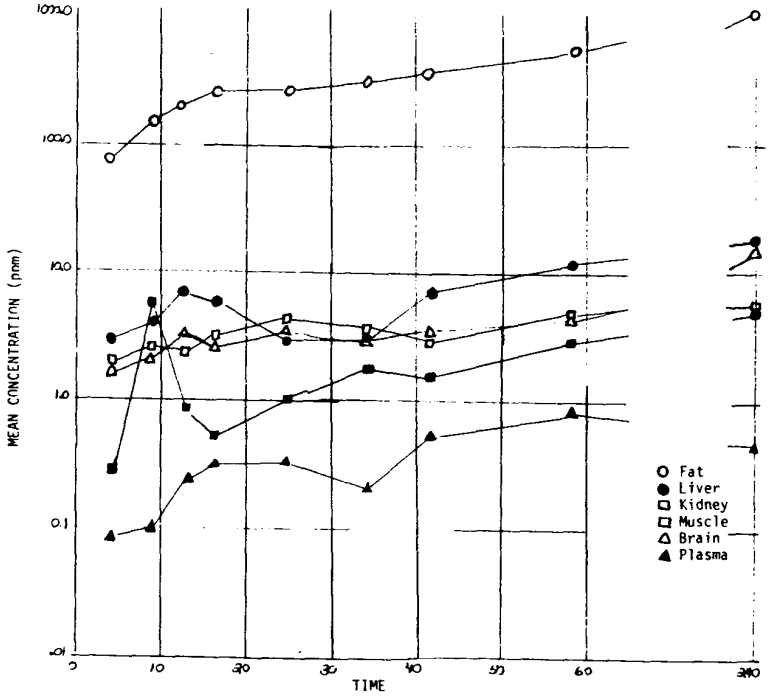

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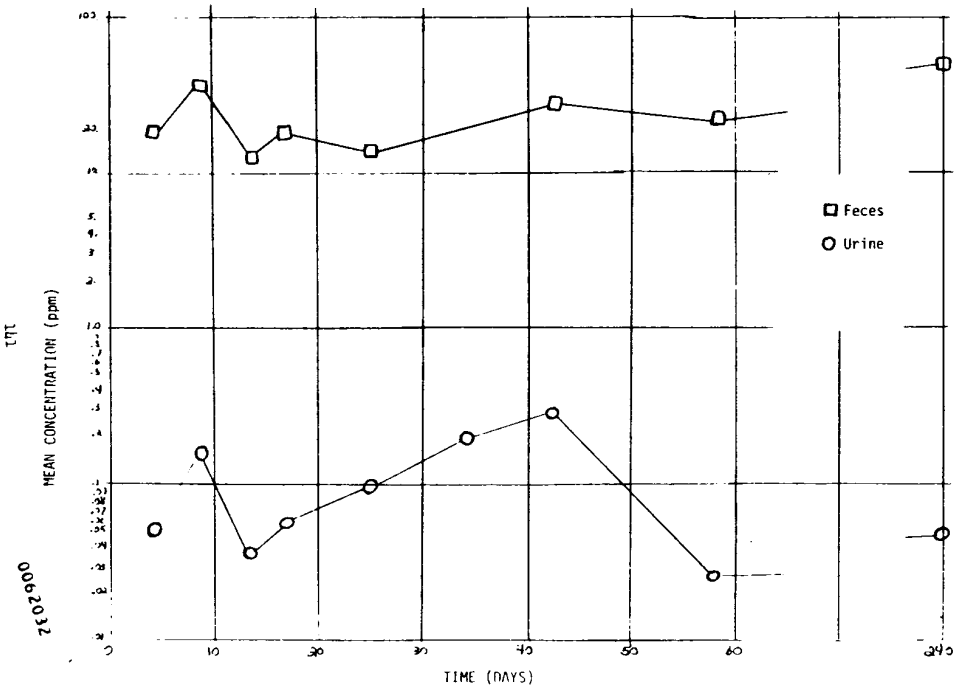
Figure 2. Excretion of PCB and PCB-Derived Material in Feces and Urine.



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(1) Aroclor 1242 is the least toxic of the Aroclors tested in two-year rat and dog studies. Aroclor 1242 was negative at 1, 10 and 100 parts per million levels.

(2) Rats fed compounds 1254 or 1260 had increased liver weights at 100 parts per million, but not at 1 or 10 parts per million with no other effects having been observed.

(3) Dogs fed 100 parts per million of 1254 or 1260 did not gain weight as well as the controls. Animals were sacrificed at 3,6 and 12 months and revealed no abnormal tissue histology.

(4) In the rat reproduction study, there was decreased survival of pups at 100 parts per million of 1242 or 1254, and decreased mating indices with 1242 at 100 parts per million were also observed. No adverse effects were found at any of the three levels of 1260 or at 1 or 10 parts per million levels of Aroclors 1242 and 1254.

In regard to the 25-week chicken study, there were no effects found following feeding with Aroclor 1260 at 1, 10 and 100 parts per million levels. There was anorexia, loss of body weight, decreased thickness of egg shells, and poor hatchability of eggs from chickens fed 10 or 100 parts per million of 1242 or 100 parts per million of 1254. There were no effects observed for Aroclor 1254 at the 1 and 10 parts per million levels. Oral feeding studies with Aroclor 1242 were repeated at the 2, 4 and 8 parts per million level and decreased hatchability was observed at 8 parts per million.

A rat teratology study of Monsanto following FDA protocol in which 35-65 mg/Kg of PCB is administered has produced no effect.

In regard to the mutagenesis study of Monsanto (Dominant Lethal Test), two dose levels of Aroclors 1242, 1254 and 1260 are being used, e.g., 500 and 1000 mg/Kg. As of the fourth week since mating, no adverse effects have been found.

It is of interest for comparison to recite the reproduction studies of the Atlanta Laboratories of the FDA (FDA Status Report, December, 1970). Weanling male and female rats were fed diets containing Aroclor 1254 at dosage levels of 0, 100 or 500 parts per million for 67 days and then pair-mated. The rats fed 100 parts per million were comparable with the controls in numbers of litters and pups per litter. However, 76.8 percent of the pups in the 100 parts per million group survived to weaning compared with 95.5 percent of the controls. The mean body weight of the 100 parts per million group at weaning was 31.4 gm and that of the controls was 39.2 gm. Only two of ten females fed 500 parts per million of Aroclor 1254 had litters (1 and 7 pups, respectively), and these pups died within 3 days after birth. In a similar study with Aroclor 1260 no difference in reproduction was found between the controls and the 100 parts per million group.

C. CONTAMINANTS IN POLYCHLORINATED BIPHENYLS

(Vos and Koeman, 42) reported a significant difference in toxicity between three commercial PCB preparations (Clophen A60, Phenoclor DP6 and Aroclor 1260), despite the marked resemblance of the gas chromatograms and the mass spectra

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(Koeman et al., 30). One hundred precent mortality, centrolobular liver necrosis and abdominal edema were found only in chicks fed the samples of Clophen A60 and Phenoclor DP6. Hydropericardium was recorded in nearly all chicks fed these PCBs and only rarely seen in chicks fed with Aroclor 1260. Porphoryia was found as a general PCB effect in chicks as well as in Japanese quail and rats (Vos and Koeman, 42).

A subsequent study by Vos and co-workers (17) revealed the presence of tetra- and pentachlorodibenzofuran as well as hexachloronaphthalene in Phenoclor DP6 and Clophen A60, but not in the Aroclor 1260 sample. Since hydropericardium occurred occasionally in chicks fed Aroclor (Flick, et al.,43). Vos and co-workers (17) felt that this could possibly be indicative of small quantities of a toxic factor in this preparation.

Dermal toxicity studies in rabbits of technical PCB samples which contain an average of 60 percent chlorine (Phenoclor DP6, Clophen A60 and Aroclor 1260) as well as fractions containing tetra- and pentachlorodibenzofuran have been recently described by Vos and Beems (44). PCB-induced skin lesions were hyperplasia and hyperkeratosis of the epidermal and follicular epithelium following application of 118 mg of the three PCBs (5 times per week for 38 days) in the back skin of adult female New Zealand rabbits. Histopathology of the liver included centrolobular degeneration, centrolobular liver cell atrophy, focal necorsis and cytoplasmic hyalin degeneration. PCB-induced kidney lesions were hydropic degeneration of the convoluted tubules and tubular dilatation with the presence of casts. Definitive hyperplasia and hyperkeratosis of the follicular epithelium of the ear skin were seen after the topical application of fractions of Phenoclor and Clophen (eluted from chromatographic columns with 25 percent diethyl ether in hexane) while the fraction from Aroclor caused a minimal hyperplasia and hyperkeratosis of the follicular epithelium. Other effects elicited by the dermal application of the PCBs included thymus atrophy and lymphopenia as well as elevated excretion of fecal coproporphyria and protoporphyria.

From the response of the back skin and the liver of the rabbit to the three PCB mixtures, and from the response of the ear to the 25 percent diethyl ether-hexane fractions, it was concluded that there were definite quantitative differences in toxicity, at least between the samples that were used in the above study (Vos and Beems, 44). and the prior studies (Vos et al., 17). The extent to which these samples are representative of the normal commercial output has not been established and emphasizes the difficulty in the evaluation of toxicity data of PCBs in which the samples may differ in the amount and nature of toxic impurities.

Vos and Beems (44) also raised the possibility that since PCB is a porphyrogenic chemical, it is possible that the skin lesions in man due to PCB may be due to a combination of chloracne and acquired porphyria cutanea tarda.

It is important to additionally stress the toxic nature of the polychlorodibenzofurans. For example, tri- and tetrachlorodibenzofuran in a single oral dose of 0.5 - 1.0 mg/Kg caused severe and often lethal liver necrosis in rabbits (Bauer et al., 45). The related compound 2,3,7,8-tetrachlorodibenzo-p-dioxin

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(which has been identified as a contaminant in 2,4,5-trichlorophenol and 2,4,5-T) caused a lethal liver necrosis in the rabbit after a single oral dose of 0.05 - 0.1 mg/Kg, and when applied to the ear again in a dose 10 times lower than that found to be effective in the case of chlorinated dibenzofuran, resulted in chloracne. Vos and co-workers (44) calculated a maximum dose/egg of 0.2 ug pentachlorodibenzofuran (obtained from Clophen A60) that caused 100 percent embryonic mortality when injected into the air cell of chicken eggs. (The analogous effect was obtained with 0.05 ug hexachlorodibenzo-p-dioxin.) (Higginbotham et al., 60). The relationship between the toxic nature of PCB and the chick edema factor 1,2,3, 7,8,9-hexachlorodibenzo-p-dioxin has been described by Flick and co-workers (58).

III. EPIDEMIOLOGICAL ASPECTS

Finklea and co-workers (46) compared both polychlorinated biphenyl and chlorinated hydrocarbon pesticide residues in 723 plasma samples collected from healthy volunteers (not occupationally exposed to pesticides) who resided in Charleston County, South Carolina. PCB residues were found in 43 percent of the samples ranging up to 29 parts per billion. DDD and dieldrin residues were found in 84 and 63 percent of the people, respectively, and p,p'-DDT and DDE residues were almost universal. PCB residues were intermediate in concentration (e.g., less than half those of p,p'-DDT and DDE but six or seven times those of o,p'-DDT, DDD and dieldrin). Maximum levels of PCB, p,p'-DDT and DDE were similar, exceeding such values for the other tested residues. The prevalence of PCB residues varied remarkably over race-residence groupings, being greatest in urban residents and rural whites. PCB residues were rare (4.1 percent) in rural blacks. Rural blacks had an arithmetic mean of 0.3 parts per billion, urban blacks, 1.9 parts per billion, urban whites 2.3 parts per billion and rural whites 3.1 parts per billion, respectively. Urban exposure to the PCBs was suggested to be via polluted air and contaminated water. (PCBs in suspended particulates have already been found).

Aspects of the determination of PCBs in the adipose of the general population of the U.S.A. to date have been summarized by Dr. Yobs in a preliminary report in the Human Monitoring Survey (originally established by the Pesticides Program of the DHEW, now Division of Pesticide Community Studies, EPA).

Table 10 depicts the preliminary summary of distribution of PCB levels in adipose tissue of general population (as analyzed by three laboratories) in the analysis of Human Monitoring Survey samples since April 15, 1971. A total of 688 samples were analyzed of which 235 (34.2 percent) were negative for PCBs and 453 (65.8 percent) were positive for measurable amount of PCBs by TLC; of these 229 (33.3 percent) contained trace to <1.0 parts per million and 188 (27.3 percent) contained ≥ 2.0 parts per million PCBs. The Aroclor formulation most frequently encountered is 1254 with Aroclor 1260 also well represented in human tissues (Enos, 47). (Aliquots of all samples in which measurable amounts of PCBs were found will be analyzed by mass spectroscopy at the Perrine Primate Laboratory for future confirmation). The correlation of PCB information in this study with age, sex, geographic distribution or diagnoses has not yet been completed.

These samples were collected in late 1968 and 1969 and the sties of the positive samples were located in the following states: Michigan, New York, Minnesota, California, Massachusetts, Georgia, Kentucky, Illinois, North Carolina, South Dakota, Ohio, Louisiana, Delaware and Arkansas.

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TABLE 10.

DISTRIBUTION OF PCB LEVELS IN ADIPOSE OF GENERAL POPULATION AS SHOWN IN ANALYSIS OF HUMAN MONITORING SURVEY SAMPLES SINCE APRIL 15, 1971.

(parts per million, wet tissue basis)

	Michigan		Florida		Colorado		Total	
	No.	%	No.	%	No.	%	No.	%
Samples Analyzed	144	100	274	100	270	100	688	100
PCB Levels Negative	12	8.3	181	66.0*	42	15.6	235	34.2
Trace—<1.0 ppm	64	44.4	44	16.0*	121	44.8	229	33.3
1.0 - 2.0 ppm	55	38.2	40	14.6	93	34.4	188	27.3
>2.0 ppm	13	9.0	9	3.3	14	5.2	36	5.2

*Actual decimal reading .05.

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Dr. G. J. Love (61) of EPA has reported that 72 sets of specimens (blood, urine, and hair) collected from 36 workers occupationally exposed to burning automobiles or refuse dumps and from an equal number of controls are to be collected and analyzed for PCB levels.

It is of interest to note the analysis of PCB in human adipose tissue. Hirao and co-workers (48) examined two human adipose tissue samples by combined GLC-mass spectrometry and found substantial quantities of PCBs ranging from pentachlorobiphenyl to decachlorobiphenyl and including at least 14 isomers and chlorine homologs. The samples were estimated to contain 200 parts per million and 600 parts per million total PCB, respectively, as determined by electron-capture gas chromatography.

IV. STUDIES IN PROGRESS

Curley and co-workers (37) have described results in a study with Aroclor 1254 to determine placental transfer, rates of excretion in milk and consequent tissue distribution and storage levels in fetuses and weanling rats following oral dosage to the mother daily on the 7th through the 15th day of gestation at 10 mg/Kg, respectively, by stomach tube. Table 11 lists the experimental protocol for the above studies. This is summarized as follows:

Fetus Analysis: Samples were taken by Caesarian section on the 20th day of pregnancy. The mean concentrations of Aroclor are given for the respective dosages as parts per million.

	Controls	10 mg/Kg	50 mg/Kg
Mother 1	0	1.42	2.42
Mother 2	0	1.24	2.90
Mother 3	0	1.14	2.93

10 mg/Kg

	Wt. at onset (gms)	Age (days)
Mother 1	264	90
Mother 2	265	90
Mother 3	258	90

50 mg/Kg

Mother 1	242	90
Mother 2	244	90
Mother 3	261	90

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Tissue Analysis: Samples were taken from 21-day-old male and female weanling rats. The concentrations in the tissues for three animals are given in ppm.

	Controls		10 mg/Kg		50 mg/Kg	
	Males	Females	Males	Females	Males	Females
Liver	<0.05	<0.05	4.06	2.85	17.36	21.63
			3.28	8.28	25.56	12.02
			2.70	4.11	11.80	15.99
		Mean $\bar{X}$	3.35	5.08	18.24	16.55
		S.E. ±	0.39	1.64	4.00	2.79

	Controls		10 mg/Kg		50 mg/Kg	
	Males	Females	Males	Females	Males	Females
Brain	<0.08	<0.08	1.96	1.94	10.16	10.48
			2.28	2.72	5.44	3.89
			2.24	1.24	7.47	6.75
		Mean $\bar{X}$	2.16	1.97	7.69	7.04
		S.E. ±	0.10	0.43	1.37	1.91
Kidney	<0.04	<0.04	1.52	1.54	7.53	2.41
			2.42	2.19	6.73	2.25
			1.14	2.32	6.70	4.10
		Mean $\bar{X}$	1.69	2.02	6.99	2.92
		S.E. ±	0.38	0.24	0.27	0.59

Milk Analysis: Milk samples were taken from suckling rats 11 days after the last dose to the mother. Concentrations are given in ppm.

	Controls	10 mg/Kg	50 mg/Kg
Range	0	16.48 - 24.90	45.80 - 100.29
Mean	0	20.60	66.35
S.E.		±1.59	±8.36

Ar 7-70-G4

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The results of a study with Ar 1260 show consequent tissue distribution and storage levels in weanling rats following oral dosage to the mother daily on the 7th through 15th day of gestation at 50 mg/Kg by stomach tube and is summarized as follows:

Note: One female and one male taken from each of 3 litters for analysis.

Tissue	Sex	Mean (3 animals)	±S.E.
Kidney	M	7.38	2.56
	F	5.44	1.41
Brain	M	7.53	1.04
	F	7.76	2.54
Liver	M	12.80	1.72
	F	16.13	6.75

Weights of Mothers 284, 264, and 288 gm.

Mean weight of Mothers 278 gm.

Additional residue studies completed include the following:

Experiment No.	Tissue	Mean ppm (4 animals)	±S.E.
Ar-7-70-B12	Fat	778	72.4
	Liver	9.9	1.16
	Kidney	5.1	.95
	Muscle	1.22	.104
	Brain	6.96	.68
	Plasma	1.6	.311

When compared to males at the same dosage level for an equal amount of time, Student's t-test shows significant difference in brain and liver storage. When these data are compared to data from male rats on the same dosage for an equal amount of time, the Student's t-test shows significant differences for brain and liver storage.

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Experiment No.	Tissue	Mean ppm (3 animals)	±S.E.
Ar-7-70-B12	Fat	1022	127
	Liver	14.6	0.69
	Kidney	2.8	0.21
	Muscle	1.2	0.11
	Brain	9.3	0.57
	Plasma	2.1	0.43

When these data are compared with 8 mo. study test for significant difference shows $p < 0.05$ for liver only.

Experiment No.	Tissue	Mean ppm (3 animals)	±S.E.
Ar-7-70-B12	Fat	820	216
	Liver	22.5	2.51
	Kidney	4.7	.24
	Muscle	2.9	.96
	Brain	11.2	1.71
	Plasma	3.0	0.78

When these data are compared with 10 mo. study t-test for significances show $p < 0.05$ for liver and kidney.

Experiment No.	Tissue	Mean ppm (4 animals)	±S.E.
Ar-7-70-B10	Fat	274	48
	Liver	2.2	.10
	Muscle	.32	.06
	Kidney	.60	.05
	Brain	2.6	.50
	Plasma	.42	.06

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TABLE 11.

EXPERIMENTS TO DATE NOT INCLUDED IN THE MANUSCRIPT, "POLYCHLORINATED BIPHENYLS: DISTRIBUTION AND STORAGE IN BODY FLUIDS AND TISSUES OF SHERMAN RATS" - A. CURLEY, V. W. BURSE, M. E. GRIM, R. W. JENNINGS AND R. E. LINDER. (37)

Experiment No.	No. of Rats	Sex	Age at Onset of Experiment (days)	Wt. (Gms)	Dosage/Route
Ar 7-70-B12	4 (analyzed)	F	30	224*	100 ppm in Diet for 247 days *8.1 mg/kg/day
Ar-7-70-B12	3 (analyzed)	F	30	224*	100 ppm in Diet for 300 days *8.1 mg/kg/day
Ar-7-70-B12	3 (analyzed)	F	30	224*	100 ppm in Diet for 366 days *8.1 mg/kg/day
Ar-7-70-B10	4 (analyzed)	F	30	240*	20 ppm in Diet for 280 days *1.6 mg/kg/day
Ar-8-71-B	A study to determine placental transfer, excretion in milk and consequent tissue storage in weanling rats of Aroclor 1254 when the compound is given to the mother rat during organogenesis.				
Ar 7-70-G4	The effect of Aroclor 1260 upon reproduction in female rats when the compound is given during organogenesis.				

*Average weight of group and average rate of food consumption for group.

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Mink reproductive studies are being carried out by Ringer, Johnson and Hoopingarner (49) of Michigan State University involving the initial feeding salmon containing 15 parts per million PCB or 10 parts per million each of Aroclor 1242, 1254 and 1260 was fatal to mink which appear to be extremely sensitive to PCB. Reproductive failure in ranch mink fed Coho salmon and other Great Lakes Fish (possibly containing PCB) have been reported by Aulerich, et al. (62).

Courtney and Chernoff (65) of the Perrine Primate Laboratory are studying the teratogenicity of PCBs. No effects have been observed in rats (CD-1 strain) given a variety of Aroclors at a level of 200 mg/Kg. Mixtures of PCBs and DDT have been found negative as well in the above strain of rats. Studies involving individual isomeric chlorobiphenyls as well as commercial PCB mixtures in in-bred strains of the rat will also be undertaken.

Wilson and Sharp (63) of the National Institute of Environmental Health Sciences are studying aspects of the interaction of beef brain (Na^++K^+) ATPase with the polychlorinated biphenyls. Enzyme activity was inhibited by 50 per cent in the presence of Aroclor 1221 or Aroclor 1254 at PCB concentrations in the range of 8-12 parts per million at 37. Under similar conditions 2.5×10^{-5}N (9 parts per million) DDT also inhibits the beef brain enzyme by about 50 percent. The PCB- and the DDT-sensitivity of the (Na^++K^+) ATPase are similar whether the enzyme is obtained from beef brain or from fish (blue gill) brain. On a parts per million basis a commercial preparation of 3-chlorobiphenyl is about as inhibitory as Aroclor 1221, whereas biphenyl, 4-chlorobiphenyl and 2, 4-dichlorobiphenyl were less inhibitory. The technique for exposure of the (Na^++K^+)ATPase to the Aroclor preparations was found to influence the degree of the enzyme inhibitory response. Beef brain (Na^++K^+)ATPase responsiveness to commercial PCBs has been demonstrated to simulate several aspects of enzyme responsiveness to DDT.

Young (50) of the Virginia Polytechic Institute is studying PCB levels (in feces and urine samples) of pre-adolescent girls fed diets typical of low income families. PCB levels in paper bags, marking tags, turkey fat, heavy fowls, eggs and poultry feeds are also being assessed and have been found in all but two samples with concentrations ranging from 0.063 parts per million to 4.56 parts per million.

The possibility of the PCBs exhibiting immunosuppressive effects is being further studied by Vos (51). The leucopenia, the reduced number of germinal centers in the spleens and lymph nodes, the atrophy of the cortex of the thymus (Vos and Beems, 44), and the atrophy of the white pulp and lymphoid foci found in the spleens of chicks fed PCB (Vos and Koeman, 42) are believed by Vos to be strong indications for an immunosuppressive effect. It was further suggested that the extent to which these observations are due to stress (release of gluco corticoids) would have to be elaborated.

Flamm and Clive NIEHS (52) are studying the mutagencity of a spectrum of Aroclors as well as individual isomers of polychlorinated biphenyls in L5178-Y mouse lymphoma cells heterozygous at the thymidine kinase locus ($TK^+/-$)3.

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Spalding NIEHS,(64) is elaborating the effect of Aroclors 1221, 1242, 1254, 1260 and 1268 as well as a number of isomeric polychlorinated biphenyls on mouse lymphoma and DDT-resistant mouse lymphoma cells.

V. SUMMARY AND CONCLUSIONS

Despite the nearly four decades of PCB use in a broad spectrum of applications, the increasing awareness of its environmental aspects as well as an increasing number of recent mammalian toxicological investigations, aspects of definitive acute, sub-acute and chronic toxicity still remain poorly known as regards man. The chemical and physical properties, e.g., the stability, complexity and heterogeneity of the material per se, the difficulty of separation and analysis as well as the non- or ill-defined nature of the material actually used or reported in many studies, have all certainly contributed in making the evaluation of toxicity and biological data difficult. Table 12 summarizes some biological and toxicological effects of the PCBs.

The above review of the current status of the toxicologic and biologic aspects of the PCBs suggests a number of future studies that hopefully should yield additional information to enable a more definitive evaluation of the risks involved in exposure to chlorinated biphenyls. A number of possible future studies are outlined in Table 13.

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Spalding NIEHS,(64) is elaborating the effect of Aroclors 1221, 1242, 1254, 1260 and 1268 as well as a number of isomeric polychlorinated biphenyls on mouse lymphoma and DDT-resistant mouse lymphoma cells.

V. SUMMARY AND CONCLUSIONS

Despite the nearly four decades of PCB use in a broad spectrum of applications, the increasing awareness of its environmental aspects as well as an increasing number of recent mammalian toxicological investigations, aspects of definitive acute, sub-acute and chronic toxicity still remain poorly known as regards man. The chemical and physical properties, e.g., the stability, complexity and heterogeneity of the material per se, the difficulty of separation and analysis as well as the non- or ill-defined nature of the material actually used or reported in many studies, have all certainly contributed in making the evaluation of toxicity and biological data difficult. Table 12 summarizes some biological and toxicological effects of the PCBs.

The above review of the current status of the toxicologic and biologic aspects of the PCBs suggests a number of future studies that hopefully should yield additional information to enable a more definitive evaluation of the risks involved in exposure to chlorinated biphenyls. A number of possible future studies are outlined in Table 13.

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TABLE 12.

SOME BIOLOGICAL AND TOXICOLOGICAL EFFECTS IN THE PCB'S.

1. Acute oral LD_{50} in mammals varies from approximately 2-10 gm/kg. (Apparent increase in mammalian toxicity with decrease in chlorine content).
2. Generally, enzyme induction increases with increase in chlorination of PCB's.
3. Induction of hepatic hydroxylating microsomal enzymes and increased estrogenic activity in the rat.
4. Enlargement of the liver and vacuolar or fatty degeneration of liver cells in rats, guinea pigs and monkeys.
5. Production of hydropericardial edema in chickens and Japanese quail.
6. Teratogenic effect in chick embryo.
7. Adverse reproductive effects in rats at levels of ca. 100 ppm in diet.
8. Possible adverse reproductive effects in mink.
9. Possible implications in aberrations in calcium metabolism and reproduction in ring doves.
10. Effects on hatchability in chickens, Japanese quail.
11. Skin, liver and kidney lesions in rabbits following dermal exposure.
12. Possible immunosuppressive effects in rabbits.
13. Chemical porphyrogenic effects in many species.
14. Chloracnegenic and hepatotoxic effects in man.
15. Hyperglyceridemic effects in man.
16. Human miscarriages, still births and transplacental transmission in abnormal pigmentation from "rice-oil disease" ("Yusho").
17. PCB residues in human adipose tissue, serum and milk.
18. Hepatoxic, chloracnegenic and porphyrogenic effects of chlorinated dibenzofuran contaminants in several species.
19. Chloracnegenic effects of chlorinated naphthalene contaminants in man.

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TABLE 13.

POSSIBLE FUTURE STUDIES INVOLVING PCB'S, THEIR INDIVIDUAL ISOMERS AND CONTAMINANTS.

1. Definitive studies on the mammalian distribution of the Aroclors, a number of key common individual isomers and their metabolites and/or degradation products in different tissues, the metabolic rate, retention times or turnover and excretion rates.

2. Elaboration of the relative toxicity of a number of the various purified isomers of PCB.

3. Elaboration of the effects of Aroclors and isomeric PCB's in mammalian reproduction studies are required because of the apparent anomalous relationship found in the pattern of chronic studies.

4. Elaboration of the teratogenicity of Aroclors and isomeric PCB's in several mammalian species at a range of dose levels.

5. Mutagenic studies involving the Aroclors and isomeric PCB's in test systems other than the Dominant Lethal (e.g., host mediated assay).

6. Elaboration of short-term toxicity effects on liver microsomal enzymes, study the influence of dilantin and hexobarbital and possible synergism and/or potentiation.

7. Study of the interaction of PCB's with other chemicals and drugs.

8. Study of the toxicity of the Aroclors and their individual isomers for different cell systems.

9. Elaboration of mammalian tissue distribution, metabolic rate and retention times of chlorinated dibenzofurans.

10. Liver function studies as well as cytogenic studies on workers in a) PCB production, b) application, e.g., packing of transformers, capacitors, c) waste disposal, and d) fishmeal processing plants.

11. Elaboration of aspects of hyperlipemia. Does it occur with low exposures and could it aggravate arteriosclerosis? Does it affect the pancreas, etc.?

12. Study of induced porphyria and clinical consequences.

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APPENDIX G

Biological Data On PCBs In Animals Other Than Man

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APPENDIX G

Biological Data On PCBs In Animals Other Than Man

Polychlorinated biphenyls have become ubiquitous in the world ecosystem in quantities similar to those of DDE. Their presence has caused concern and stimulated research to evaluate their role in the biosphere.

The significance of PCBs to wild animals depends upon both their lethal toxicity and their sublethal physiological effects. These are the subjects of the present paper. Coordinate knowledge of level of exposure, as shown by frequency and levels of occurrence of PCBs in the environment, is essential to complete the understanding. These are summarized elsewhere in this report.

I. TOXICITY

Outright mortality of wild animals can affect populations, particularly those of long-lived species. Measurements of direct toxicity are therefore important first steps in evaluation of a chemical. Other laboratory studies also are needed for proper interpretation of field observations. These include studies to diagnose cause of death by behavior of poisoned animals, tissue changes, and concentrations of chemical in critical tissues.

A. BIRDS

The toxicities of different PCBs to pheasants (Phasianus colchicus), mallards (Anas platyrhynchos), bobwhite quail (Colinus virginianus), and coturnix quail (Coturnix coturnix) were compared with the toxicities of DDT, dieldrin, and other insecticides (Heath, et al., 1). Tests of six PCB mixtures, containing 32 to 62 percent chlorine, showed that the toxicity increased with the percentage of chlorine. In general, toxicities were similar to those of DDE. There were some differences in sensitivity of the species. Bobwhite were most sensitive, followed in turn by pheasants, mallards, and coturnix quail. Bobwhite were 3-4 times as sensitive as coturnix. Special tests with coturnix quail showed that the toxic effects of DDE and Aroclor 1254 were additive but not synergistic.

In other studies, Aroclor 1254 was approximately as toxic as DDE to four species of blackbirds: grackles (Quiscalus quiscula), cowbirds (Molothrus ater), starlings (Sturnus vulgaris), and redwings (Agelaius phoeniceus) (Dustman, et al., 2). Redwings were somewhat more susceptible to DDE than to PCBs. Signs of poisoning were sluggishness with slight tremors of moderate amplitude, much as with chemicals of the DDT group. Internally, livers frequently had hemorrhagic streaks or spots, and the gastrointestinal tract commonly contained blackish fluid, but these signs were not sufficiently consistent for distinctive diagnosis.

Aroclor 1254 was approximately 1/13 as toxic as DDT to Bengalese finches (Lonchura striata) (Prestt, et al., 3). Tremoring and other signs were similar to those observed among blackbirds; the finches had enlarged kidneys and some had hydropericardium.

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Chicks kept in batteries recently painted with an epoxy-resin paint, (McCune et al., 4). died as a result of Aroclor 1242 in the paint. Others fed this compound developed hydropericardium and enlarged livers and kidneys.

Egg injection studies showed that Aroclor 1242 had a relatively high toxicity (McLaughlin, et al., 5).

Aroclor 1248 fed to 10-day-old chicks depressed growth rates at dietary levels of 50, 100, and 150 parts per million (Rehfeld et al., 6). Only 2-4 of 10 chicks survived each of the three higher dosages. Growth rates were reduced at 20, 30, and 40 parts per million in a second experiment; mortality was 16 of 30 birds at 50 parts per million dosage, 4 of 20 at 40 parts per million, and 1 of 30 at 30 parts per million to 5 weeks of age. Birds fed diets containing more than 20 parts per million developed general edema; those fed diets containing 40 and 50 parts per million developed noticeably smaller combs and wattles than normal. Liver weight increased as a percentage of body weight, primarily as a reflection of lower body weights.

Mortality was high among White Leghorn cockerels fed a dietary dosage of 500 parts per million of Aroclor 1254 from the day of hatching; the birds died between the third and tenth week of feeding (Platonow and Funnell, 7). At 250 parts per million birds did not die until the thirteenth week. At this dosage, weight gain was poor, livers increased in weight relative to body weight, combs and wattles were abnormally small, and testes were small. The effect on combs appeared before the effect on the testes.

Polychlorinated biphenyls supplied by three different manufacturers gave strikingly different results in toxicity tests with domestic chickens, although all formulations contained 60 percent chlorine (Vos and Koeman, 8). The PCBs were Phenoclor DP6, manufactured in France; Clophen A60, manufactured in Germany; and Aroclor 1260, manufactured in the United States. Twenty-four birds fed 400 parts per million of Phenoclor DP6 all died within 60 days; all had liver necrosis and 18 had hydropericardium. Twenty-two of 24 birds fed Clophen at the same dosage also died with liver necrosis and 20 with hydropericardium. However, none of the birds fed Aroclor died; none had liver necrosis, and only three had hydropericardium. Chickens fed all formulations developed atrophy of the spleen and excess quantities of porphyrins. These different effects of the three brand-named products were later largely explained by the identification of chlorinated dibenzofuran and chlorinated naphthalene as contaminants in Phenoclor and Clophen (Vos et al., 9).

In a further study with coturnix quail, porphyria again resulted from dosage with Aroclor 1260 that was carefully tested to insure the absence of measurable amounts of contaminants (Vos et al., 10). Males given a daily dose of 50 mg/kg of PCBs (the lowest dosage tested) developed porphyria, as did females given 100 mg/kg, although those given 10 mg/kg or less did not. The porphyria was closely associated with an increase of mitochondrial ALA synthetase.

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1. Residues in Birds Killed by PCBs. Chickens killed by PCB dosage in the studies of(Vos and Koeman, 8) generally contained from 120 to 420 parts per million in the brains, but the overall range was from 40 to 700 parts per million. Residues in livers were 120 to 2,900 parts per million.

Coturnix quail poisoned by PCBs (Phenoclor DP6) contained residues of 342-1710 (av. 1158) parts per million in the brain and 1079-8350 (av. 3280) parts per million in the liver (Koeman, 11).. Bangales finches killed by PCBs had residues of 70 to 697 parts per million in the livers; those sacrificed at the end of the experiment contained 3 to 634 parts per million (Prestt,et al., 8). Residues in brains were somewhat lower; the proportional amount in the brain in comparison with the amount in the liver averaged higher in the birds that died than in those that were sacrificed.

A bald eagle found sick in the field contained high residues of both DDE and PCBs in its brain, suggesting that PCBs may have contributed to its death. Residues of DDE in the brain were 385 parts per million, which is within the lethal range for DDE (Stickel et al., 12). However, the brain also contained 230 parts per million of PCBs, 6 parts per million of DDD, 2.2 parts per million of dieldrin, and 0.4 parts per million of heptachlor epoxide.

B. INSECTS

The toxicity of PCBs to insects also is related to the chlorine content, but in the reverse order to the result with birds. PCBs with lower amounts of chlorine were more toxic to flies than PCBs with higher chlorine content, and the toxicity of mixtures with more than 48 percent chlorine was very low (Lichtenstein et al., 11). Toxicity of dieldrin and DDT was enhanced beyond an additive effect by the addition of the lower chlorinated PCBs.

Topical applications of Aroclor 1254 to a grasshopper (Clorthippus brunneus) produced delayed mortality that occurred at the time of molt (Moriarty, 14).

C. FISH AND AQUATIC INVERTEBRATES

Shrimp (Panaeus duorarum) are sensitive to low concentrations of PCBs (Duke,et al., 15). All individuals died as a result of a 48-hour exposure to flowing seawater containing 100 parts per billion of Aroclor 1254; 80 percent died in 24 hours. These shrimp accumulated 3.9 parts per million in their tissues. Shrimp exposed to 10 parts per billion did not die, but accumulated 1.3 parts per million of PCBs in their tissues.

Seventy-two percent of the juvenile shrimp died from a 20-day exposure to 5 parts per billion of Aroclor 1254 and the tissues accumulated 16 parts per million. Crabs (Callinectes sapidus) were less sensitive, but accumulated an average of 23 parts per million in a 4-week exposure at 5 parts per billion and still contained 22 parts per million after a week in clean water and 11 parts per million after 4 weeks in clean water.

The small crustacean,(Gammarus oceanicus) had a lethal threshold in 30-day tests between 0.001 and 0.01 parts per billion in colloidal solution and between

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0.01 and 0.1 parts per billion in emulsions of Aroclor 1254 solubilized in Corexit 7664 (a commercial nonionic surfactant preparation) in seawater (Wildish, 16). Gammarus that died had severely necrosed branchiae, and some animals exposed to as little as 0.001 parts per billion developed a less extensive sublethal necrosis. Moulting or freshly moulted animals were particularly vulnerable.

Shell growth of oysters stopped completely in a 96-hour exposure to 100 parts per billion of PCBs. Shell growth was reduced by 40 percent in exposure to 10 parts per billion and the oysters accumulated 33 parts per million of PCBs in their tissues (Duke, et al., 15).

Pinfish (Lagodon rhomboides) survived exposure to 10 or 100 parts per billion of Aroclor 1254 for 48 hours, but those exposed to 100 parts per billion accumulated 17 parts per million (Duke, et al., 15).

In further studies at the same laboratory, pinfish and spot (Leiostomus xanthurus) died when exposed for 14-45 days to 5 parts per billion of Aroclor 1254, but spot appeared unaffected by exposure to 1 part per billion for periods up to 56 days (Hansen, et al., 17). Onset of death in both species exposed to 5 parts per billion was delayed. Pinfish developed fungus-like lesions on the body, with some hemorrhaging. Spot usually ceased feeding, became emaciated, and developed ragged fins and lesions. Some fish that survived exposure to 5 parts per billion Aroclor 1254 became diseased and died even though they were placed in flowing water free from PCBs.

Rainbow trout (Salmo gairdneri) are very sensitive to the terphenyls (Guthrie and Acres, 18). Fifty percent of the fish exposed to Santowax OM* + 30 percent high boilers at concentrations of 10 parts per billion or greater died in 48 hours or less under normal oxygen conditions. With reduced oxygen, 50 percent died in less than 2 hours even at a concentration of 2 parts per billion (the lowest tested). The fish became hyperactive in less than 1/2 hour in water containing 25 parts per billion or more. Within 1 to 5 hours, gills reddened, swimming slowed, and balance and avoidance reactions were altered. Less than 5 percent of the fish recovered after showing these signs. HB-40* was less toxic, and less than 25 percent of the fish died within 48 hours.

II. PHYSIOLOGY

The effects of PCBs on reproduction and other physiological processes of wild animals are apt ultimately to have the most serious impact on the populations. Yet these effects are the most difficult to evaluate. Pertinent data are summarized here, with the exception of data for laboratory mammals, which are included in other sections of this report.

A. METABOLISM AND KINETICS

In Swedish waters, lower organisms such as mussels and fish contained a greater preponderance of PCBs with lower chlorination than did birds, which suggested that the compounds with fewer chlorines were metabolized or excreted

* Monsanto Chemical Company trade name. Santowax is a mixture of ortho-, meta-, and para-terphenyls. HB-40 is a mixture of hydroterphenyls and terphenyls. High boilers are tar-like decomposition products produced as a result of the exposure of terphenyls to a high radiation field.

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faster than those with more chlorines, so that the latter were subject to greater increase in the food chain (Jensen, et al., 19).

In two simple food chains, fish to eagles (Haliaeetus albicilla) and fish and mussels (Mytilus edulis) to seals (Phoca vitulina and Pusa hispida), concentrations increased hundreds to thousands of times from prey to predator (Jensen, et al., 19). Fish contained hundredths to tenths of parts per million; fresh mussels contained hundredths of parts per million; seal blubber contained 5-21 parts per million; and the muscle of the white-tailed eagle contained 150-240 parts per million.

In Great Britain, birds that feed on birds or mammals contained the highest concentrations of PCBs, those that have a mixed diet contained the next highest, and those that feed on insects had the lowest (Prestt, et al., 3). The fish-eating herons from the southeast of England contained higher residues of PCBs than birds of any other area.

Many species of California birds contained hundredths to tenths of parts per million of PCBs; peregrines (Falco peregrinus) contained greater amounts, one as high as 98 parts per million in muscle (Risebrough, et al., 20), while fish in the same area contained only thousandths of parts per million.

Residues of PCBs in the industrially polluted Escambia Bay increased in the expected order. Water contained a maximum of 275 parts per billion, and sediment a maximum of 486 parts per million. Oysters (Crassostrea virginica) contained 2-3 parts per million, shrimp (Penaeus duorarum) 1.5-2.5 parts per million, blue crabs (Callinectes sapidus) 1-7 parts per million, and pinfish 6-12 parts per million (Duke, et al., 15).

Subsequent samplings from three stations in the Bay showed little change in concentrations of PCBs in sediments even after 9 months (Nimmo, et al., 21). Experiments were then undertaken that showed that shrimps (Penaeus duorarum) and fiddler crabs (Uca minax) could accumulate Aroclor 1254 from the sediments. Relationships between concentration of PCBs in sediment and concentration in crabs and shrimps were variable. The maximum accumulation was from sandy silt containing 61 parts per million (dry weight) of Aroclor 1254; fiddler crabs accumulated 80 parts per million (wet weight) in their bodies; shrimp accumulated 240 parts per million (wet weight) in the hepatopancreas. Some PCBs were present in the water and a portion of the accumulation could have been from that source. The ratio of individual PCB isomers maintained integrity in the sediments and tissues of test animals throughout the investigation, indicating no pronounced metabolic changes of the PCBs.

Spot (Leiostomus xanthurus) exposed to 1 part per billion of Aroclor 1254 for 56 days attained maximum concentrations in 14-28 days, although absolute amounts continued to increase as the fish grew (Hansen, et al., 17). Maximum concentration in whole spot was 37,000 times that in the test water. These results were very similar to those for DDT reported earlier from the same laboratory. The PCBs were lost slowly from the tissues of spot after they were placed in clean flowing water. After 84 days of flushing, the concentration had dropped 73 percent and the absolute amount had dropped 61 percent. Isomers of Aroclor 1254, with the exception of one peak, maintained their integrity in spot.

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Marine diatoms (Cylindrotheca closterium) exposed to Aroclor 1242 at 0.01 and 0.1 part per million in culture flasks absorbed the chemical to a concentration of 4.7 and 109.2 parts per million, 470 and 1,100 times the concentrations in the surrounding medium (Keil, et al., 22). The higher concentration (0.1 part per million) sharply inhibited growth as indicated by harvest weights and cell counts and reduced RNA synthesis and the chlorophyll index but had no effect on DNA levels. No such effects were produced by the 0.01 part per million concentration. Some early eluting PCB materials not common to the known mixture of Aroclor 1242 were isolated from the diatoms, suggesting possible metabolism of the Aroclor.

A species of salt-water diatom (Thalassiosira pseudonana) grew at a significantly reduced rate when exposed to Aroclor 1254 at concentrations of 25, 50, and 100 parts per billion in the culture medium (Mosser, et al., 23). Survival was reduced by exposure to 50 and 100 parts per billion. A second species (Skeletonema costatum) was more sensitive and grew somewhat more poorly when exposed to 10 parts per billion. Both species were more sensitive to PCBs than to an equivalent amount of DDT. By contrast, a marine green alga and two species of freshwater algae were not inhibited by these or higher concentrations of PCBs.

The small crustacean,(Gammarus oceanicus),appeared to absorb Aroclor 1254 across the general integument (Wildish and Zitko., 24). Uptake was not altered by removal of branchiae epi-flora and by removal of epi-fauna, or by the stage of intermoult. Rate of uptake was greater at higher concentrations and declined after a few hours. Dead Gammarus absorbed significantly smaller amounts of PCBs from the seawater than did the living animals.

PCBs inhibited ATPases in bluegills (Lepomis macrochirus). Aroclor 1254 was the most effective inhibitor, followed by Aroclor 1221 (Yap, et al., 25). Aroclor 1268 and 5460 showed less pronounced effects. Tissues (brain, kidney, liver, and muscle) also differed; muscle ATPáse showed the greatest sensitivity, with a response similar to that observed for DDT.

In the Netherlands, the PCBs of lower chlorination were more common in fish (Leuciscus rutilus) than in sea birds, and it was concluded that compounds with fewer chlorines probably were metabolized or excreted relatively rapidly (Koeman, et al., 26). This hypothesis was confirmed in an experiment with Japanese quail; the gas chromatographic pattern of PCBs in the quail tissues was considerably altered from that of the fed material, and many of the peaks representing lower chlorinated compounds disappeared. There was a similar difference in pattern of the PCB compounds present in the egg of a mallard duck and the pattern of the Aroclor 1254 that the duck had eaten (Heath, 1).

PCBs increased the breakdown of estradiol in domestic pigeons (Risebrough, et al., 20) and kestrels (Falco sparverious) (Lincer and Peakall., 27). demonstrating their capability to induce microsomal enzyme activity. The PCBs were given by injection (pigeons) or ingestion (kestrels) of relatively high dosages; the birds were sacrificed and in vitro laboratory studies were made of the homogenized livers.

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Mallard ducklings demonstrated the possibility of interacting effects between PCBs and disease organisms (Friend and Trainer, 28). Thirty-five to 44 percent of the 10-day-old ducklings exposed for 10 days to a dietary dosage of 25, 50, or 100 parts per million of Aroclor 1254 died upon subsequent exposure to duck hepatitis virus in contrast to only 14 percent of the birds exposed only to the virus.

Swedish robins (Erithacus rubecula) given 5 micrograms of Clophen A50, for 11-13 days showed a greater migratory restlessness than controls (Ulfstrand and Södergren, 29).

Pheasant hens absorbed 94 percent of the Aroclor 1254 given as a single capsule dose, a very efficient entry of this chemical into the system (Dahlgren, et al., 30). Residues in muscle declined 82 percent in 28 days after dosage. Residue were excreted in both eggs and feces.

Residues in the milk of cattle inadvertently exposed to PCBs declined at the rate of 1.3 percent per day when uncontaminated feed was restored (Fries, et al., 31). The rate of loss of DDE residues was identical. PCBs in milk fat decreased from 12.6 parts per million to 5.8 parts per million in about 3 weeks and to 2.1 parts per million in about 4 months.

A cow given 10 mg/kg of Aroclor 1254 in a single dose released an average of 3.9 parts per million into the whole milk during the next 4 days' milking. A dosage of 100 mg/kg produced 36 parts per million in the milk of another cow (Platonow, et al., 32). The gas chromatographic pattern of the Aroclors in the milk was very similar to that of the fed compound.

B. REPRODUCTION

Pheasants given a capsule dose of 50 mg of Aroclor 1254 weekly for 17 weeks produced fewer eggs than controls, and a higher percentage of chicks pipped the shell but did not hatch (Dahlgren and Linder, 33). Chicks that hatched weighed less and survived more poorly than controls. Eggshell thickness was not affected. In behavioral tests of the offspring on a visual cliff, more of the chicks from the dosed parents made the undesirable choice of jumping to the deep side, or made no choice, in the 5-minute test period. None of these effects occurred among the groups whose female parents were dosed with 12.5 mg.

Mallards fed a dietary dosage of 25 parts per million of PCBs from about 11 weeks before their first breeding season and through their second year laid eggs with shells of normal thickness (Heath, et al., 1). The number of eggs laid, hatchability, and survival of young did not differ significantly from the untreated controls. In another test, mallard ducks fed 10 or 500 parts per million of Aroclor 1254 in the diet for about 5 weeks laid eggs with normally thick shells. Bobwhite quail fed diets containing 50 parts per million of PCBs, or 30 parts per million of DDE, or a combination of 25 parts per million of PCBs plus 15 parts per million of DDE for about 11 weeks before their first breeding season reproduced as well as controls.

Ring doves (Streptopelia risoria) fed 10 parts per million of Aroclor 1254 for 6 months laid eggs no lighter than those laid by control birds. (Peakall, 34). Fourteen birds fed 10 parts per million of PCBs before and after dosage and nine birds injected intraperitoneally with 160 mg/kg 1-4 days before egg laying confirmed the lack of effect of PCBs on shell weight.

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Chickens fed Aroclor 1242 at 10 parts per million or 100 parts per million and Aroclor 1254 at 100 parts per million laid fewer eggs, hatched fewer chicks, and laid eggs with thinner shells than controls in tests made for the Monsanto Chemical Company by the Industrial Bio-Test Laboratories. Those fed Aroclor 1242 at 1 part per million, Aroclor 1254 at 1 part per million or 10 parts per million, or Aroclor 1260 at 1 part per million, 10 parts per million, or 100 parts per million did not differ significantly from controls.

The studies with chickens and with ducks differed in dosage levels and in the type of PCBs employed, so that the differences cannot necessarily be ascribed to the difference in species. Further tests with ducks and with other species are needed in order to understand the potential for PCBs to affect reproduction of wild birds.

In recent studies at the Patuxent Wildlife Research Center, a statistical evaluation of the role that different chemicals may play in thinning the shells of brown pelicans in the field has shown that DDE residues correlate much better with shell thinning than do residues of dieldrin or PCBs (Blus, et al., 35; Anderson, et al., 36) reported similar results from studies of shell thinning and residue content of the eggs of cormorants and white pelicans.

Reproductive failure occurred among ranch mink that were fed fish from the Great Lakes and the Miramichi (Gilbert, 37 ; Aulerich, et al., 38). Such effects could not be produced experimentally by dosages of DDT or its metabolites in levels far in excess of those present in the fish (Aulerich, et al., 38). This led to the belief that other contaminants must be responsible, and tests have been initiated with PCBs as a possible candidate.

III. SUMMARY

Polychlorinated biphenyls have become ubiquitous in the world ecosystem in quantities similar to those of DDE.

Experimental studies have shown that PCBs have a toxicity to mallards, pheasants, bobwhite quail, coturnix quail, red-winged blackbirds, starlings, cowbirds, and grackles that is of the same order as the toxicity of DDE to these species. Overt signs of poisoning also are similar to those caused by compounds of the DDT group. Toxic effects of DDE and Aroclor 1254 to coturnix chicks were additive, but not synergistic.

PCBs containing higher percentages of chlorine are more toxic to birds than those containing lower percentages. PCBs of foreign manufacture contained contaminants to an extent that greatly increased their toxicity.

Residues of PCBs in the brains of birds killed by these compounds measure in the hundreds of parts per million. PCBs may have contributed to mortality of some birds in the field.

Toxicity to insects of PCBs of different degrees of chlorination is the reverse of the pattern in birds: the lower chlorinations are more toxic to insects. PCBs enhanced the toxicity of dieldrin and DDT to insects.

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Shrimp are very sensitive to PCBs and most will die as a result of 20-day exposure to a concentration of 5 parts per billion. PCBs also inhibit shell growth of oysters. Crabs are less sensitive; all accumulate residues to many times the concentrations in the water, and a test with crabs showed that they lost the residues very slowly.

Growth of certain species of marine diatoms was experimentally inhibited by PCBs, but algae were not affected.

The small marine crustacean, Gammarus, is sensitive to PCBs in concentrations of thousandths to tenths of a part per billion.

Exposure to 5 parts per billion of Aroclor 1254 caused mortality of two species of fish in 14-45 days. Onset of death was delayed and was accompanied by fungus-like lesions.

Rainbow trout were quickly killed by polychlorinated terphenyls at 10 parts per billion under normal oxygen conditions and at 2 parts per billion with reduced oxygen.

Metabolic changes of PCBs have been suggested by environmental observations of different isomeric patterns in animals of different trophic levels. Quantitative differences also are pronounced, with magnifications of hundreds to thousands of times.

Laboratory studies have shown no metabolic changes of PCBs by crabs and shrimps, minimal changes by fish, and pronounced changes by birds.

PCBs induce microsomal enzyme activity in birds. Exposure to PCBs increased the susceptibility of mallard ducklings to duck hepatitis virus.

Offspring of pheasants whose parents received high dosages of PCBs made poor choices in visual cliff tests. Egg production and hatching after pipping also were affected. Migratory restlessness was increased in English robins exposed to PCBs.

Long-term studies of the reproductive effects of Aroclor 1254 on mallards and bobwhite quail and of Aroclor 1254 plus DDE on quail showed no significant differences from controls. In studies of chickens, however, egg production and hatchability were impaired by high doses of Aroclor 1254 and by low doses of Aroclor 1242.

Statistical evaluations of the role that different chemicals may play in thinning eggshells of brown pelicans showed that DDE residues correlate better with shell thinning than do residues of dieldrin or PCBs, confirming observations with cormorants and white pelicans.

IV. CONCLUSIONS

PCBs are man-made biologically active substances that are dispersed throughout the environment and stored in the tissues of animals. They are lethally toxic to fish and aquatic invertebrates in concentrations measured

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in parts per billion or less. They are metabolized and excreted very slowly by these organisms.

They are only moderately toxic to birds and mammals and the lethal levels are similar to those of DDE. In sublethal exposure, they are physiologically active and induce enzyme activity. Effects on reproduction have been shown for chickens but not for ducks, quail, or doves, and for rabbits only at high dosages.

Full evaluation of their actual or potential effects in the environment is hampered by the complex nature of the mixtures that compose them, and by the inclusion of contaminants in these mixtures. Experimental studies have been conducted with the unaltered products, as sold, and the results may not properly reflect the effects of the components as they exist in the environment.

The evidence available, however, indicates that PCBs must be viewed as potential problems. The difficulties of attaining a proper evaluation in any reasonable length of time suggest that the least costly course would be to take all measures possible to prevent their escape into the environment.

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APPENDIX H

Regulatory Action on PCBs

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APPENDIX H

Regulatory Actions on PCBs

I. EXISTING REGULATORY AUTHORITY

At least ten Federal laws are potentially relevant for the regulation of PCBs. However, not all of them have actually been utilized to deal with the PCB problem. Federal Food, Drug, and Cosmetic Act (21 U.S.C. 301 et.seq.)

Section 402 of the Federal Food, Drug, and Cosmetic Act (FDC Act) defines an "adulterated food" as a food which (among other criteria):

1. Contains any poisonous or deleterious substance which may render it injurious to health; or
2. Contains any food additive unless the use of the additive conforms to exemptions or regulations issued by the Food and Drug Administration (FDA). FDA has applied both of these criteria to PCBs, depending upon the circumstances under which the PCB entered the food.

The FDC Act prohibits the introduction of adulterated food in interstate commerce; prohibits the adulteration of food while moving in interstate commerce; and prohibits the receipt of an adulterated food in interstate commerce and subsequent delivery of it for sale or otherwise when the initial recipient is aware that the food is adulterated. The Act also authorizes FDA to seize any adulterated food products which have entered interstate commerce or which become adulterated while held for sale after receipt in interstate commerce.

The FDA enforces the FDC Act by various means including inspections of food establishments (other than meat, poultry, and egg breaking plants) to determine whether the provisions of the Act are being violated. These inspections include the collection and analysis of food samples. Some of the samples are of a routine surveillance nature to determine the presence of adulterants such as pesticides which exceed tolerance levels and other contaminants such as mercury, lead, cadmium, harmful bacteria, natural poisons, etc. However, the actual number of routine surveillance samples taken is small, and effective enforcement relies heavily on information on the known or suspected existence of specific instances of food adulteration.

A. FEDERAL INSECTICIDE, FUNGICIDE, AND RODENTICIDE ACT (7 U.S.C. 135-135k)

Under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) all pesticides (referred to in the act as "economic poisons")

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shipped in interstate commerce must be registered with the EPA. EPA can refuse to register a product if it will cause injury to man or the environment if used according to the label. The registration of a product can be suspended or cancelled if it is found to no longer meet the criteria for registration.

On October 29, 1970, the Pesticides Regulation Division (then in the Department of Agriculture; now in EPA) issued a notice (PR Notice 70-25) to all pesticide manufacturers, formulators, and distributors. The notice stated that, "Formulators and manufacturers of economic poisons containing polychlorinated biphenyls and polychlorinated terphenyls should change their formulations to eliminate such chemicals either as active or inactive ingredients. It is believed that a period of six months is a reasonable period of time within which to affect such formula changes." Assuming that the notice has been complied with, there should be no pesticides which currently contain PCBs.

B. FEDERAL WATER POLLUTION CONTROL ACT (33 U.S.C. 466 et.seq.)

The Federal Water Pollution Control Act (FWPC Act) authorizes the Administrator of EPA to enforce State water quality standards established by the States and approved by the Federal Government if the State is not adequately enforcing the standards. No States have established water quality standards relating specifically to PCBs. Thus the water quality standards part of the FWPC Act is not a useful tool for dealing with the PCB problem.

Section 12 of the FWPC Act applies primarily to accidential discharges of hazardous polluting substances. It requires the immediate reporting to EPA or the Coast Guard of any discharge of a hazardous substance from a vessel or an onshore or off-shore facility. The discharger is responsible for making the report and for clean-up, but EPA is authorized to remove or arrange for the removal of the hazardous substance if the discharger is unable or unwilling to do so. PCBs are being designated as hazardous substances under section 12, and the authority contained in the section could be used if an accidental spill of PCBs into water should occur.

C. THE REFUSE ACT OF 1899 (33 U.S.C. 407)

Another legal authority for controlling water pollution is section 13 of the 1899 Refuse Act which forbids discharge of any wastes (other than municipal wastes) into navigable waters without a permit, issued by the Army Corps of Engineers, which can limit the discharge of substances into water. EPA and the Justice Department are prepared to use the Refuse Act to prevent PCB discharges.

D. THE CLEAN AIR ACT (42 U.S.C. 1857 et. seq.)

The general authorities contained in the Clean Air Act are not, for the most part, applicable to PCBs because PCBs are not generally emitted into the air in the normal operations of a municipal or industrial facility. PCBs may become air contaminants through the burning of refuse, but such emissions usually could be controlled only by preventing the substance from initially getting into the refuse.

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Section 112 of the Clean Air Act authorizes the establishment of national emission standards for hazardous air pollutants. However, it is unlikely that section 112 would be invoked to deal with PCBs for the same reasons that the other Clean Air Act authorities are generally not applicable.

E. THE EGG, MEAT, AND POULTRY ACTS

The Consumer and Marketing Service of the U.S. Department of Agriculture (USDA) administers three acts relevant to the PCB problem: The Egg Products Inspection Act (P.L. 91-597); the Wholesome Poultry Products Act (P.L. 90-492); and the Wholesome Meat Act (P.L. 90-201).

These authorities apply to meat, egg, or poultry products from the time they reach the processing plant until they are purchased by the consumer. Once they leave the plant, they also fall under the legal authority of the FDC Act as do all foods. USDA routinely checks meat, poultry, and egg processing plants, but relies primarily on local jurisdictions for inspection at retail outlets.

The egg, meat, and poultry acts essentially define a product as adulterated if it contains any substance in an amount which is judged deleterious to health. USDA follows guidelines established by FDA under the FDC Act for determining what amounts of a particular substance are deleterious. USDA can seize adulterated products or prevent them from being distributed.

F. THE OCCUPATIONAL SAFETY & HEALTH ACT (29 U.S.C. 651-678)

Chemical hazards in the workplace are regulated under the Occupational Safety and Health Act. The Secretary of Labor, in cooperation with the Secretary of Health, Education, and Welfare, is authorized to set and enforce occupational safety and health standards applicable to businesses affecting interstate commerce.

No specific standard has been set for occupational exposure to PCBs. However, the Department of Labor has set exposure limits for comparable substances, and it could enforce limits on PCBs by applying limits set for compounds such as chlorodiphenyl. The standard for chlorodiphenyl is 1mg per cubic meter - 8 hour average exposure.

G. ACT TO REGULATE TRANSPORTATION OF EXPLOSIVES AND OTHER DANGEROUS ARTICLES (18 U.S.C. 831-835)

The Department of Transportation (DOT) regulates the transport of hazardous substances under the Act to Regulate Transportation of Explosives and Other Dangerous Articles. The Act authorizes DOT to promulgate regulations for the safe transportation of such materials.

DOT has issued tentative test protocols for classifying substances according to their acute toxicity, but these have not yet gone into effect. Responsibility for insuring that standards are complied with rests initially with the manufacturer or shipper.

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II. STANDARDS, TOLERANCES, OR GUIDELINES ESTABLISHED

The most important limits which have been established for PCBs have been those established by FDA to deal with particular instances of food or feed contamination.

Standards or, more accurately, tolerances for products such as for PCBs may be established under Section 406 or Section 409 of the Food, Drug, and Cosmetic Act, which require a finding that the substance is required in the production of the food or cannot be avoided by good manufacturing practices or a showing of safety and a need for use. No permanent tolerances for PCBs have been established, since all the safety data necessary and the need for use to support such tolerances are not available at this time. In the absence of tolerances, the Food and Drug Administration, in order to respond to accidental contamination, has set interim guidelines for levels of PCB in some foods at which it will take action under the Food, Drug, and Cosmetic Act. It is now proposing to establish temporary tolerances for permitting unavoidable PCBs in several categories of food.

FDA has taken action to remove from the market shell eggs containing more than 0.05 parts per million PCBs and feeds containing more than 0.5 parts per million PCB where the PCBs present appears to be from accidental contamination of feed. The U.S. Department of Agriculture has also taken action to remove poultry from the market containing 5.0 parts per million PCBs where such contamination was the result of accidental contamination of feed fed to poultry. The action level of 5 parts per million (whole tissue basis) was established in 1971 by FDA. This level was reduced to 3 parts per million on a fat basis in 1972.

The Food and Drug Administration has taken no action against any other products for PCB contamination but has informed the States and others that it would consider action on milk if PCBs exceed 0.2 parts per million (whole basis) and on fish if PCBs exceeds 5 parts per million. Some States have taken action to remove milk from the market that contained more than 0.2 parts per million PCBs.

The temporary tolerances recently proposed by FDA are listed in Table 1.

The guidelines and the temporary tolerances taken into account available toxicological data, the estimated amount and type of intake of PCB from food, and also the sensitivity of the methods used for detecting PCB. Thus more PCB is allowed in poultry than in eggs because children are fed egg yolk, and children are not considered to have as efficient detoxication mechanism for handling PCBs as adults. It should be noted that these limits were established to deal with particular incidents, and thus may be changed as new knowledge develops or as circumstances change.

Some food has been discovered to be contaminated because of migration from food packaging materials containing PCBs. Thus FDA also has proposed that a temporary tolerance of 5 parts per million in paper packaging

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Table 1

FDA Proposed Temporary Tolerances for PCB Residues

Food	Tolerance
Milk	2.5 ppm (fat basis)
Dairy Products	2.5 ppm (fat basis)
Poultry	5 ppm (fat basis)
Eggs	0.5 ppm
Complete animal feeds	0.5 ppm
Animal feed components (including fish meal)	5 ppm
Fish	5 ppm (edible portion)
Infant and junior food	0.1 ppm
Food-packaging materials	5 ppm

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materials be established permitting unavoidable PCB residues in such materials for a period of one year. This will provide an opportunity for the orderly elimination of PCB-containing raw materials used in the manufacture of food packaging materials.

EPA recently has proposed regulations which would prohibit all intentional discharges of PCB into the water, and would limit PCB levels in the water to .01 parts per billion. These regulations probably would be enforced through use of the Refuse Act.

III. APPLICATION OF REGULATORY AUTHORITIES

Within the past two years there have been several incidents involving PCB contamination. These include the following:

In September 1969 FDA detected PCBs in West Virginia milk. The source of PCBs was suspected to be the use of spent transformer fluid as a solvent for herbicide spray. Grade A milk shippers involved were taken off production by the State.

In April 1970 FDA identified the presence of PCB residues in milk sampled by the Ohio State Department of Agriculture. The Department removed a large quantity of milk from the market. The chemical was traced to a material used as a sealant in the silos where dairy feed was stored. Similar occurrences of PCB contamination of dairy herds have been reported in several Southeastern States.

Since June 1971, the Meat and Poultry Inspection Program (MPIP) and the Poultry Division, Consumer and Marketing Service, USDA, have been surveying specifically for PCBs. Prior to that time, the MPIP's ongoing survey of chlorinated hydrocarbons should have detected any PCBs present in animal or poultry fat at a level of 1 parts per million or above. None was detected in the fat of poultry, swine, cattle, or sheep prior to December 1970. Since that time, there have been five separate incidents of contamination of poultry with PCBs. Each appears to be a single-source, one-time occurrence. The Boultry Division's egg sampling program in the Southeastern U.S. and in Minnesota has not detected levels of PCBs which are significant--most values falling below the sensitivity of the method.

Contamination of hens slaughtered on December 2, 1970, led to placing all laying hens in Orange, Sullivan, and Ulster Counties, New York, under quarantine through August 30, 1971, requiring pretesting for PCBs prior to slaughter. Levels up to 26.8 parts per million PCBs were found in the fat. Approximately 137,100 hens were condemned and buried on the farms. Flocks containing less than 5 parts per million PCBs were sold in commercial channels, frequently at drastically reduced prices. Eggs from contaminated flocks were destroyed. The source of contamination was feed containing returned bakery product wrappings.

Frozen turkeys produced on farms from Modesto, Fresno, Stockton, and Santa Rosa, California, were found to contain up to 28 parts per million of of PCBs in the fat. The source of contamination is unknown. All turkeys

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from these sources (approximately 100,000 lbs.) were detained until testing was completed (Feb.-May 1971). Muscle meat was salvaged and used in turkey rolls. Fat and skin containing over 5 parts per million PCBs went to nonfood uses.

Contamination of anchovy fish meal with PCBs from a leaky heat exchanger was discovered by the Monsanto Company in July 1971 in Wilmington, N.C. One immediate and noticeable result was poor hatchability of eggs from the poultry fed the PCB contaminated diet. Accordingly, USDA required that all poultry coming to market from these States be tested before marketing. During the first 2 weeks of testing, 541 samples were analyzed, representing over 2.5 million birds. Of these, 2.4 percent were retained until disposition could be made. The 88,000 chickens which had been fed the fish meal were destroyed by the owner of the chickens. FDA seized 75,000 eggs, 5 shipments of commercial fish feed, and 1 shipment of fish meal which had been contaminated, and USDA barred from market more than 30,000 pounds of processed frozen eggs. In addition, the processing firm recalled over 16,000 tons of the PCB-contaminated fish meal.

In September 1971, turkeys from one grower on eight farms in Detroit Lakes, Minnesota, were found to contain up to 20 parts per million PCBs (fat basis). One hundred forty flocks in the immediate area, including parts of Minnesota, North Dakota, and South Dakota, were tested , but no levels above 2 parts per million were found. Approximately 1 million turkeys were withheld from market by the single grower until residue levels were acceptable. The source of the PCBs has not been determined, but feedgrade fat contaminated with PCBs from a heat exchanger is suspected.

Also in September, the State of Michigan stopped its annual program of distribution of Cohoe Slamon from Lake Michigan, because the fish contained high residues of PCB and DDT.

In February 1972 chickens in Maine were discovered to contain PCB contamination of up to 172 parts per million in their fat. These contaminated chickens had been given feed from a plant in Thorndike, Maine, but officials have been unable to locate the exact source of the PCBs. More than a million chickens have been destroyed.

Many of these incidents have evoked criticism of the Federal Government for failure to detect the problem sooner or for failure to act more stringently. It must be recognized that insofar as detection of problems relies on inspection of food products being shipped to market, it is impossible to inspect even a small fraction of the foods being shipped. Factory inspection can detect some problems, but even this approach is severely limited by available resources. The prime reliance must be on controlling the use of the offending substance.

IV. FUTURE ACTIONS AND NEEDS

Enough data are available to indicate that PCBs are pervasive in the environment. The basic regulatory challenge is thus twofold: to minimize human exposure to PCBs already present in the environment and to prevent more PCBs from entering the environment.

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Based on available monitoring data and reports, one route of human exposure to PCBs currently in the environment is through food. The PCB contribution made by other routes of exposure has not been sufficiently measured.

The FDA has issued a proposal which will prohibit the use of PCBs in and around food processing plants and will establish limitations on the use of salvaged paper containing industrial chemicals for food packaging. These proposed actions should prevent occurrences such as the Wilmington incident and the problem of PCBs in food packaging materials. However, there will still be problems, as evidenced by the Coho Salmon contamination, the mysterious Minnesota turkeys, and the discovery of high levels of PCBs in cardboard food containers. The solution to these problems seems to lie in a better understanding of the path which PCBs follow through the environment and on full use of existing regulatory authorities. Even with better understanding we can probably expect future isolated incidents of PCB contamination.

Existing regulatory authority is generally inadequate to prevent more PCBs from entering the environment. The Monsanto Company has reported voluntarily limiting the distribution of PCBs to "closed systems" but this limit has no force of law behind it. The government has no power to restrict imports of PCBs by foreign manufacturers, and if it disagreed with Monsanto's judgment on allowable uses it could not impose more stringent limitations on Monsanto or on any other potential manufacturer.

This regulatory gap would be filled by the Administration's proposed Toxic Substances Control Act. The proposed Act, sent to the Congress in February 1971, would authorize the Administrator of EPA to restrict or prohibit the use or distribution of a chemical substance if such restriction were necessary to protect health and the environment, and it would also authorize him to issue standards for tests to be performed and for results to be achieved from such tests for various classes and uses of new substances. Thus, in addition to providing the regulatory authority needed to deal with the PCB problem, it would also establish a system for preventing new chemicals from becoming similar problems. Action by the Congress to approve the Toxic Substances Control Act is the most important step which can be taken to deal with PCBs and similar problems.

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• U. S. GOVERNMENT PRINTING OFFICE : 1972—481-333 (759)

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