

Schalk Reports

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Physical Properties of PCBs

Introduction

Chlorinated biphenyls, more commonly known as "polychlorinated biphenyls" or "PCBs", are a group of 209 distinct chemical compounds, none of which are known to occur naturally. They were first synthesized and studied in Germany during the 1870's, and first produced on a commercial scale in 1929 by an American firm called Swann Chemical Company. The Monsanto Industrial Chemical Company, headquartered in Saint Louis, Missouri, bought Swann in 1935, and subsequently dominated the domestic PCB market until 1978, when all domestic production ceased. Approximately 99% of the PCBs used in the U.S. were produced by Monsanto at its plants in Sauget, Illinois and Anniston, Alabama. Approximately 1% were imported, and about 0.1% were manufactured by the now defunct Texas company, Geneva Industries. Various concerns offered PCBs for sale after Monsanto announced, as early as 1970, that they intended to initiate a policy of selling PCBs only for use in "closed" systems such as capacitors, transformers, and electromagnets. Monsanto implemented its restrictive sales policy by the end of 1972.

All but one of the PCBs are solids in their pure state; but in combination, as they are usually encountered, they take the form of oils and waxes. PCBs do not readily dissolve in water, so in the environment they are primarily found in sediments or in the fat of living organisms.

Over a billion pounds of PCBs have been distributed in the United States. Millions of pounds are free in the environment. Those which are still in use in electrical equipment have been the subject of recent regulatory activity because of the extremely toxic byproducts which are produced when they burn.

Mixtures of PCBs were initially produced for use by the electrical equipment manufacturing industry as wire coatings and fill for capacitors and transformers. Additional markets were later developed for the mixtures, and by the 1960's, these accounted for around half of the PCBs produced. The resistance of PCBs to destruction by chemicals, radiation, heat, fire or microbial degradation added to their appeal as lubricants, hydraulic fluids, waterproofing agents, flame retardants, heat transfer agents, plasticizers, and as components in a variety of products including surface coatings and carbonless copy-paper. Their resistance to destruction also accounts for their accumulation and persistence in the environment.

Reports of a mass poisoning incident in Japan, feed contamination in the United States, and global environmental contamination brought PCBs to the world's attention during the late 1960's and early 1970's, and provided the impetus for Monsanto's voluntary restriction

of PCB sales in 1971 and the banning of PCBs from commerce by the Toxic Substances Control Act of 1976. Evidence that PCBs exert insidious, long term effects on the body's immune and endocrine systems continues to mount, helping to explain a whole gamut of adverse effects ranging from reproductive failure to cancer.

PCBs have been the subject of wrongful death, personal injury, and property damage claims, as well as criminal proceedings. Sometimes the issues involved are simple; sometimes they are very complex. This report is intended to serve both as both a handy reference manual for the simple cases, and as a primer to prepare you for the experts and literature you will encounter on the complex ones.

The Properties and Uses of PCBs

This report provides background information concerning three important aspects of PCBs: the nature of the substance; how it was, and still is used; and how it behaves once it escapes into the environment. The nature of PCBs is addressed first on the gross, physical level, and then on the molecular level. The latter discussion is easy to understand, and provides a useful background for reading more advanced technical literature, dealing with experts, and interpreting laboratory test data.

The information concerning the uses and behavior of PCBs helps explain how some people became massively exposed, and why PCBs are still a matter of concern years after their manufacture has been banned.

The material presented in parts of this chapter is somewhat complex because PCBs themselves are complex and constantly changing mixtures. It is rare to find any one of the 209 distinct compounds known as PCBs in isolation, separate from at least some of the other 208 compounds which are also known as PCBs. It is also rare to find PCBs free of traces of the extremely toxic contaminants which are produced during manufacture, use, and burning. Thinking clearly about PCBs requires thinking in terms of components, contaminants, and byproducts.

Observable Characteristics of PCBs

Commercial PCBs include a clear, colorless free-flowing oil (Aroclor 1221); clear, practically colorless free-flowing oils (Aroclors 1232, 1242, and 1016); a clear, free-flowing oil which varied between being colorless to light yellow-green (Aroclor 1248); a light-yellow viscous liquid (Aroclor 1254); light-yellow sticky resins (Aroclors 1260 and 1262); and white powders, including Aroclors 1268 and 1270, and an imported Italian product called "Fenclor DK", or, more commonly, "deca". Monsanto also manufactured a series of products in the Aroclor series which contained chlorinated terphenyls (PCTs), often in combination with PCBs. One of these products, Aroclor 4465, was a light yellow, brittle resin containing 75% PCB and 25% PCT; another was a brittle black resin containing 60% PCB and 40% PCT. PCB oils were used primarily in their pure or in very concentrated form in electrical, hydraulic, and heat exchange equipment. Solid PCBs were used in numerous formulations, including asphalt, paint, and caulking compounds.

PCBs have a distinct odor which resembles the odor of carbonless copy paper (duplicate form paper). Between 1957 and 1971, such paper contained an average of 3.4% PCBs,¹ and it now contains similar compounds with odors similar to those of PCBs.

PCBs are denser than water and are virtually insoluble in it. If one were to attempt to mix PCBs with pure water, practically all the PCBs would eventually settle to the bottom.

PCBs mix readily with organic solvents such as paint thinners. They also mix readily with grease and oil. This explains the fact that while PCBs in lakes are normally found primarily in the sediment, they can accumulate on the surface when oil slicks are present.

Commercial PCB mixtures do not boil until they are heated well above the boiling point of water. They are very slow to evaporate at room temperature. The term "vapor pressure" is used to describe the propensity of substances to boil or evaporate. The low vapor pressures of PCBs added to the useful lifetime of PCB containing products such as coatings and resins, and minimized evaporation problems in hydraulic and electrical equipment, where PCBs were used in their liquid form.

Most PCBs reportedly burn only at elevated temperatures. The higher chlorinated products have the least tendency to ignite. They have little tendency to ignite or decompose when heated, and they are resistant to destruction by acids and other harsh chemicals. The stability of PCBs under a variety of conditions helps account for both their commercial success, and their persistence in the environment.

1. EPA, PCBs in the United States Industrial Use and Environmental Distribution, page 134, EPA 560/6-76-005 (1976).

UNDERWRITER'S LABORATORIES FLAMMABILITY RATINGS*

<u>Fluid</u>	<u>Flammability Rating</u>
Ether	100
Gasoline	90 - 100
Ethyl Alcohol	60 - 70
Kerosene (100 degrees)	30 - 40
Mineral Oil	10 - 20
Aroclor 1242	2 - 3

* Reprinted from: Department of Health Education and
Welfare, PCBs in the Environment, Report No. ITF-PCB-72-1,
COM-72-10419 (1972).

Uses of PCBs

Commercial production of PCBs began in 1929. The initial manufacturer, the Swann Chemical Company, was purchased in 1935 by the Monsanto Chemical Company, headquartered in Saint Louis, Missouri.¹ They were America's major manufacturer, producing about 1.3 billion pounds before they ceased production in 1979. Approximately 1% of America's PCBs were imported.¹

Initially, Monsanto produced PCBs to fill electrical transformers and capacitors and to coat electrical cable. PCBs are useful these applications because of their high resistivity; that is, their resistance to arcing in the presence of electrical fields. By way of comparison, salt water readily conducts electricity and therefore has a very low resistivity.

In addition to their high resistivity, PCBs also have high dielectric constants. To understand this, it is necessary to realize that capacitors are devices for storing energy, and the amount of energy which can be stored in a capacitor depends on the internal medium. When PCBs are used as the internal liquid medium, more energy can be stored in a capacitor of a given size than would be possible if a chemical with a lower dielectric strength were used. The U.S. government interdepartmental task force estimated that PCB impregnated capacitors are 1/6 the size, 1/5 the weight, and 1/4 the cost of comparable oil impregnated capacitors, providing the advantages of reliability, long life and compactness.² The chemical stability, fire resistance, high resistivity, and high dielectric constants of PCBs help explain why PCBs were used in most capacitors manufactured in the United States prior to the implementation of the Toxic Substances Control Act of 1976.

Most capacitors were impregnated with PCBs by submerging them in the hot liquid. Large capacitors and transformers were filled directly through orifices. Wire was coated by submerging it in PCBs prior to adding the insulation.

During the 1930's and 1940's, PCBs were often mixed with compounds called "chlorinated naphthalenes."³ These mixtures, as well as the chlorinated naphthalenes themselves, were known by the trade name "Halowax". Halowax appears to have been phased out after World War II, perhaps due to the fact that chlorinated naphthalenes can

rapidly destroy the liver.⁴ Chlorinated naphthalenes resemble PCBs molecularly, and have been identified as trace constituents of commercial PCB mixtures and as combustion byproducts formed during transformer fires.

In 1970, Monsanto began informing its customers that, because of persistent reports of the accumulation of PCBs in the general environment, they were restricting sales to companies using PCBs in "closed" systems.⁵ This meant that Monsanto would no longer sell PCBs to companies who ordered them for use as hydraulic fluids, heat exchange fluids, surface coatings, etc. After 1971, Monsanto sold PCBs only to capacitor and transformer manufacturers. Some former PCB customers found foreign suppliers, but most used substitute chemicals. Monsanto encouraged electrical equipment manufacturers to re-sell PCBs to electrical equipment repair shops, even though Monsanto was unwilling to sell directly to the shops.

Between 1929 and 1971, a variety of uses were found for PCBs. They mixed with other compounds to form malleable plastics for caulking and other applications, they effectively encapsulated ink in carbonless copy paper, and they were mixed with insecticides to inhibit evaporation, making them last longer.

In what turned out to be a particularly inappropriate application of PCBs, they were used as a component of coatings inside grain silos. The resulting contamination of dairy herds sometimes rendered their milk unfit for market. Numerous out of court settlements and at least one major lawsuit⁶ resulted from the loss of livestock, silage, and income and the expense of re-coating or replacing the affected silos. Coatings similar to those used in silos were also used in swimming pools.

PCBs were used as hydraulic fluids and lubricants, especially in applications involving high temperature and pressure. They were also used in numerous other products such as floor wax, adhesives, paints, lacquers, varnishes, and pigments.

Because of their stability and fire resistance, and because they can absorb and give off heat without undergoing large temperature changes (high heat capacity), PCBs were also used to transfer heat. Heat exchange applications involved both taking heat away from motors and transformers in order to cool them, and providing heat by circulating hot PCBs through coils or jackets. Heating PCBs at one location, and then pumping them to a location

where heat was needed, provided heat without without placing the substance being heated near flames or electrical coils. Whenever flammable liquids are used, there is a fire danger at the heat source.

The use of PCBs to provide heat led to two mass poisoning episodes, one in Japan and the other in Taiwan. In both cases, circulating hot PCBs were used to heat rice oil to remove odors prior to packaging. The heating apparatus developed small leaks, and the rice oil became contaminated with PCBs. Thousands of people were poisoned in these catastrophes, and while it is not clear that anyone was killed directly, great suffering, disfiguration (from chloracne), and disease resulted. *

1. EPA, PCBs in the United States Industrial Use and Environmental Distribution, page 207, EPA 560/6-76-005 (1976). Some estimates of overall imports place the total amount at slightly less than 0.5% of domestic production. According to the table on page 209, Supra, imports rose to 2.7% of domestic production in 1972 (Monsanto restricted sales in 1971), and declined during the next few years.
2. U.S. Department of Health Education and Welfare, PCBs and the Environment, Report No. ITF-PCB-72-1 (1972).
3. Schwartz, Dermatitis from Synthetic Resins and Waxes, 26 Dermatitis 590 (1936).
4. Flinn and Jarvik, Liver Lesions Caused by Chlorinated Naphthalene, 27 Am J Hygiene 19 (1938).
5. Haleys vs. Michigan Silo Company, C & B Silo Company, and Monsanto; Huron County, Michigan, File No. 77-00-2593-NP.
6. Kuratsune and Shapiro (eds), PCB Poisoning in Japan and Taiwan (1984).

MARKETING OF PCBs

During the 1960's, Monsanto expanded the market for PCBs using trade journal ad campaigns and brochures which stressed features such as the following:

Stability. PCBs can be boiled with concentrated lye, stirred with sulfuric acid, and heated for extended periods of time without appreciably decomposing. PCBs were marketed as non-reactive reaction media for high temperature chemical reactions. Chemicals could be combined and heated in PCBs, and the product removed once the reaction was complete. Monsanto's promotional literature does not indicate any concern on their part about the fact that extraordinarily toxic byproducts build up in PCBs which have been subjected to heat and certain chemicals.

Adhesiveness. Aroclors were said to adhere strongly to smooth surfaces such as glass, metal, and varnished surfaces.

Resistance to drying. When exposed to air in the form of thin films, PCBs evaporate extremely slowly. They do not tend to harden. Yet ads in trade journals of the painting industry pointed out that Aroclors do not retard the rate of drying of films, and that they can be used in quick drying varnishes and paints.

Electrical resistivity. PCBs do not conduct electricity.

Flame inhibition. Aroclors were marketed as fire retardants which could be mixed with resinous and pitch-like materials, and which could be incorporated into foam rubber and plastic films.

Monsanto not only pointed out the potentially useful properties of PCBs, they also recommended specific applications. Some of these are discussed below.

PCBs were said to be useful as fire retardants in asphalt. An illustration in one advertisement shows men applying material to a roof.

They were promoted in mechanical applications, such as raising lifts or actuating brakes. PCBs were said to be suitable hydraulic fluids in these applications because they do not attack metals, even at high temperatures; because they resist chemical and mechanical breakdown; and because of their good lubricating properties under high pressure.

Monsanto recommended PCBs for heat transfer applications, including space heating for offices. Just as radiators heat rooms by circulating water from a central boiler, so PCBs can be circulated through radiators, into submerged coils for large scale cooking operations, or

through jackets for high temperature processes such as rubber curing.

Other recommended mechanical application fo PCBs included their use as low cost substitutes for silicone oils in vacuum pumps, as mercury substitutes for temperature controls, and as roof seals for furnaces.

PCBs were promoted as paint additives. Monsanto said they greatly improve chemical resistance, weatherability, film strength, and adhesion to metal, wood, or masonry. They were specifically recommended for traffic road marking, marine and swimming pool paint, and applications and coatings for grain silos.

PCBs were recommended as swelling agents to tighten shrunken rubber seals. Monsanto said that instead of replacing seals in automatic transmissions, it was often only necessary to add PCBs to the transmission fluid.

Monsanto recommended adding a few tenths of one percent PCBs to bulk chemicals to prevent dusting. PCBs would help hold the chemicals together so that of potentially toxic or volatile chemical dusts would not rise during handling.

PCBs were also recommended as additives to insecticide formulations as vapor suppressants. Monsanto claimed that the U.S. Department of Agriculture reported that inclusion of between 5% and 25% PCBs increased the kill life of lindane spray up to ten times. PCBs were particularly recommended for use with chlordane and all the other chlorinated insecticides for uses not involving crops.

PCBs were recommended for use in precision casting waxes for dental castings, and for making precision aircraft parts and costume jewelry. The PCBs were said to impart hardness without brittleness, resistance to shrinking, sharp definition; sharp melting point; and fire resistance.

Monsanto recommended PCBs as lubricants or lubricant additives in a variety of situations. Where pressure is involved, such as in oxygen delivery systems, PCBs were said to be excellent additives to grease and pipe thread compounds. They were said to enhance the resistance of gear oils to sheer and temperature degradation. Where oils needed to be heavier than water, as in bridge rollers, PCBs mixed with about an equal weight of oil were recommended.

Tradenames Assigned to Monsanto's PCBs

<u>Tradename</u>	<u>Company</u>	
Aroclor 1221	Monsanto	
Aroclor 1232	Monsanto	
Aroclor 1242	Monsanto	
Aroclor 1248	Monsanto	
Aroclor 1254	Monsanto	
Aroclor 1260	Monsanto	
Aroclor 1262	Monsanto	
Aroclor 1268	Monsanto	
Aroclor 1270	Monsanto	
Aroclor 25(00) series	Monsanto	75% PCBs, 25% PCTs*
Aroclor 44(00) series	Monsanto	60% PCBs, 40% PCTs
Aroclor 6037	Monsanto	63% PCBs, 37% PCTs
Aroclor 6040	Monsanto	60% PCBs, 40% PCTs
Aroclor 6050	Monsanto	50% PCBs, 50% PCTs
Aroclor 6062	Monsanto	38% PCBs, 62% PCTs
Aroclor 6070	Monsanto	30% PCBs, 70% PCTs
Aroclor 6090	Monsanto	10% PCBs, 90% PCTs
Pydraul	Monsanto	(Hydraulic Fluid)
Pyroclor	Monsanto	
Therminol	Monsanto	(Heat transfer fluid)
Santovac	Monsanto	(Vacuum-pump oil)
Turbinol	Monsanto	(Compressor fluid)
Chlorextol	Allis-Chambers	
Dykanol	Federal Pacific	
Inerteen	Westinghouse	
No-Flamol	Wagner Electric	
Pyranol	General Electric	
Elemx	McGraw - Edison	

* PCT stands for "polychlorinated terphenyls." "Terphenyl" is a compound composed of three benzene rings attached to one another by single bonds.

SOME USES OF PCBs

Capacitors
Transformers
Hydraulic fluids
Heat-transfer fluids
Lubricants
Plasticizer in rubbers
Plasticizer in resins
Carbonless copy paper
adhesives
wax extenders
Dedusting agents
Inks
Immersion oils for microscopes
Pesticide extenders
Cutting oils
Sealants
Caulking compounds
Flame retardants

COMPOSITION OF COMMERCIAL PCB MIXTURES

There are 209 pure chemical compounds known collectively as polychlorinated biphenyls (PCBs). 208 of these are solids in the forms of white powders, flakes, or needles, and one is a clear liquid. When referred to individually, these compounds are called "PCB congeners". Thus, there are 209 distinct PCB congeners. Mixtures of PCBs are usually oils or waxes, despite the fact that the mixtures are made up of compounds which are solid in their pure form. Monsanto only marketed PCB mixtures.

These products were not, however, formulated from individual congeners; the mixtures were produced simultaneously in single batches. Solid biphenyl was heated beyond its melting point, and chlorine gas was introduced. The longer the chlorine was added, the more viscous the resulting PCB mixture would be. The manufacturing process provided no hint that the oily or waxy products were not single compounds, much less that they were mixtures of solids. Each PCB congener in the commercial mixtures has its own unique characteristics, such as tendency to evaporate, solubility in water (none are very soluble) and biodegradability. It is, therefore, not surprising that the PCB combinations found in lake sediments and human tissues seldom closely resemble commercial products. Nature takes its toll on different congeners at different rates, thereby changing their relative amounts in mixtures.

Trace Impurities

Two classes of compounds, known as chlorinated naphthalenes (PCNs) and chlorinated dibenzofurans (PCDFs), have been identified in unused PCBs. Members of another class, the chlorinated terphenyls, have been identified in PCBs which have been used as heat exchange fluid. When PCBs are subjected to heat, there is a tendency for a variety of toxic contaminants to form. Fires involving PCBs are considered very dangerous because of the potential for those products to form.

The PCDFs are among the most potent toxins known. It is possible that even the minute traces (1 to 10 ppm) of PCDFs reportedly found in some unused Aroclors are toxicologically significant. It is commonly believed that PCDFs found in PCB residues after accidental fires are at least as important as the PCBs themselves.

The extent to which PCDF levels varied from batch to batch is not well documented. Literature provided by Monsanto indicates that Aroclor 1248 was sometimes clear and sometimes had a yellow-green tinge. The fact that yellow-green byproducts were present in some batches but not in others suggest a fair degree of variability, but does not prove that some batches had extraordinarily high levels of PCDFs.

COMPOSITIONS OF MONSANTO'S AROCLOR PRODUCTS

Tadename

Aroclor 1221	21% Chlorine (by wt.)
Aroclor 1232	32% Chlorine
Aroclor 1242	42% Chlorine
Aroclor 1016	41% Chlorine
Aroclor 1248	48% Chlorine
Aroclor 1254	54% Chlorine
Aroclor 1260	60% Chlorine
Aroclor 1262	
Aroclor 1268	
Aroclor 1270	
Aroclor 25(00) series	75% PCBs, 25% PCTs**
Aroclor 44(00) series	60% PCBs, 40% PCTs
Aroclor 6037	63% PCBs, 37% PCTs
Aroclor 6040	60% PCBs, 40% PCTs
Aroclor 6050	50% PCBs, 50% PCTs
Aroclor 6062	38% PCBs, 62% PCTs
Aroclor 6070	30% PCBs, 70% PCTs
Aroclor 6090	10% PCBs, 90% PCTs

* Products beginning with "12" consist of over 99% PCBs; the digits after the "12" indicate the percentage of chlorine by weight.

** PCT stands for "polychlorinated terphenyls." "Terphenyl" is a compound composed of three benzene rings attached to one another by single bonds.

CHEMISTRY OF PCBs

Introduction

Knowing the molecular structure of PCBs is helpful in interpreting test data, visualizing the similarities between some PCB congeners and extraordinarily toxic compounds such as the dioxins, and understanding how PCBs are transformed by heat and oxygen to dioxin-like toxins such as chlorinated biphenylenes and dibenzofurans. Readers without strong backgrounds in science will find the following presentation easy to understand. This simplicity is not achieved by sacrificing accuracy, but by eliminating non-essential details.

Atoms and Molecules

There can be no simple answer to the question: What are atoms and molecules? Certainly, they are the very small (sub-microscopic) building blocks which comprise most matter as we know it on earth; but when we try to "picture" atoms and molecules, we run into trouble. The difficulty stems from the fact that reality on the atomic scale is not like the reality we experience with our senses. Existence is really quite different on that tiny level, and to visualize it is to create an imaginary model of something we cannot experience. For the purposes of this monograph, nothing is lost by viewing atoms as little hard spheres and bonds as light weight sticks which hold atoms together. Atoms held together by bonds are called molecules.

Two atoms can be joined by either one, two, or three bonds. Carbon atoms almost always have a total of four bonds. Oxygen usually has two, and chlorine and hydrogen usually have one bond. Examination of the following examples should make all of this clear. Readers are urged to construct each example using a ball and stick molecular model kit.

EXAMPLES

In the following examples, we will use the standard convention of letting "C", "O", "Cl", and "H" represent carbon, oxygen, chlorine, and hydrogen atoms respectively. Dashes between the letters represent bonds between atoms.

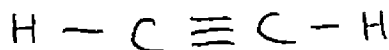
1. H_2O (water). Each hydrogen atom has one bond; oxygen has two.



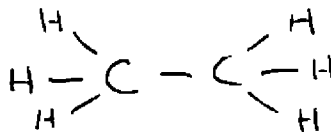
2. CO_2 (carbon dioxide). Carbon has four bonds, each oxygen has two.



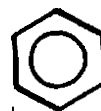
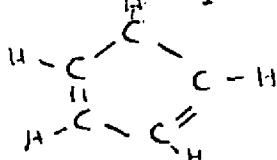
3. C_2H_2 (acetylene). Each hydrogen atom has one bond, each carbon has four. Note the carbon-carbon triple bond.



4. C_2H_6 (ethane). Ethane has all single bonds. Again, each carbon atom has four bonds and each hydrogen atom has one.



5. C_6H_6 (benzene). As usual, each carbon atom has four bonds and each hydrogen atom has one.

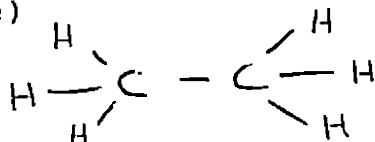


Short-hand
representation of
benzene

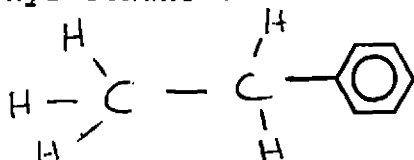
The six carbon atom of benzene form a very stable framework. Even under harsh chemical conditions in which the hydrogen atoms of benzene are readily replaced by other atoms, the six membered carbon ring remains intact.

Because benzene rings are so stable, they act as discrete units in chemical reactions. In other words, they attach to atoms and molecules under some conditions and disassociate from them under others, all the while remaining intact six membered rings of carbon atoms. These rings are often called phenyl groups when they are joined to other groups of atoms.

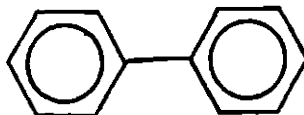
6. C_2H_6 (ethane)



7. C_8H_{10} (phenyl ethane). A benzene ring attached to another molecule is often called a "phenyl" group; thus, the name "phenyl ethane".

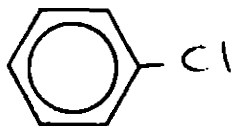


8. $C_{12}H_{10}$ (biphenyl). When benzene is heated in the presence of certain metals to over 700 degrees Centigrade, benzene rings attach together to form chains of varying length. The shortest chain consists of two rings, and is called "biphenyl", "phenylbenzene", or "diphenyl". The prefixes "bi-" and "di-", of course, both refer to the fact that there are two rings. "Biphenyl" is the most common name.

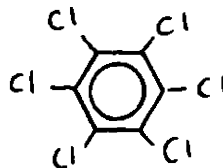


biphenyl
(also called "diphenyl" and "phenylbenzene")

9. Chlorobenzenes. Under a variety of chemical conditions, chlorine atoms will replace hydrogen atoms on benzene rings. Depending upon the amount of chlorine used, the conditions of the reaction, and the reaction time, 1 to 6 chlorine atoms can be added.



Chlorobenzene

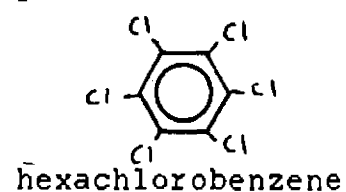
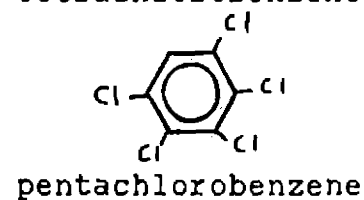
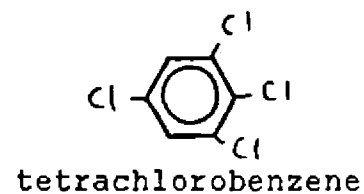
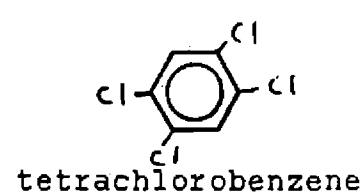
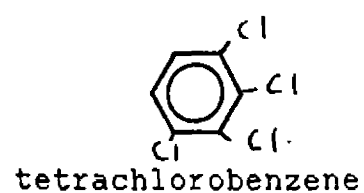
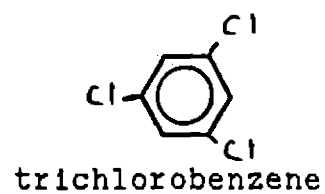
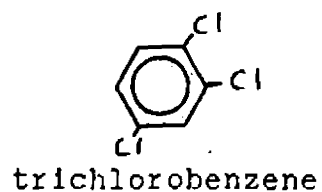
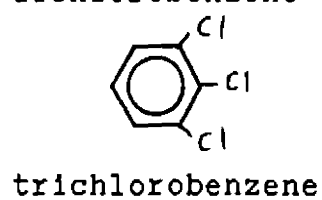
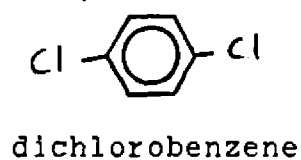
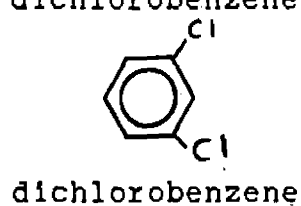
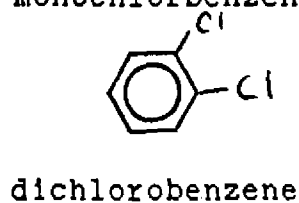
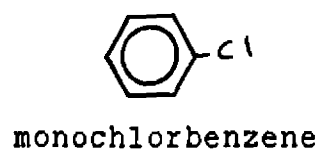


Hexachlorobenzene

When chlorine is scarce, or when the reaction is stopped before completion,, mixtures containing chlorobenzenes with fewer than six chlorine atoms are formed. The compounds in this series with 1, 2, 3, 4, 5, or 6 chlorine atoms per molecule are called mono-, di-, tri-, tetra-, penta-, and hexachlorobenzene respectively. There are three distinct dichlorobenzenes, three trichlorobenzenes, and three tetrachlorobenzenes. Each of these is a unique chemical compound, with its own characteristics and properties.

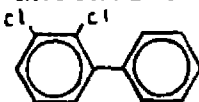
Ball and stick models are useful for visualizing the possible ways of substituting chlorine atoms on a benzene ring. These models seem like childrens toys, but professional chemists find them very useful for visualizing molecules. All twelve of the chlorobenzenes are shown in Table 1.

TABLE I

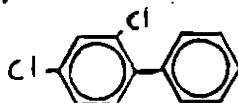


PCB Molecules

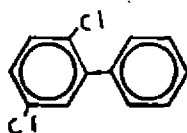
When biphenyl is heated in the presence of chlorine gas and certain metals, chlorine atoms gradually replace hydrogen atoms on the phenyl rings. Since PCBs (polychlorinated biphenyls) are, by definition, biphenyl molecules to which between one and ten chlorine atoms have been attached, this process is actually the manufacture of PCBs. As progressively more chlorine is added, mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decachlorobiphenyls are produced. The 15 possible dichlorobiphenyls are shown below. The system for naming them is explained in another report.



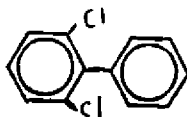
2,3-dichlorobiphenyl



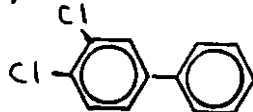
2,4-dichlorobiphenyl



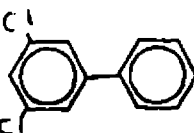
2,5-dichlorobiphenyl



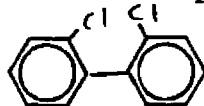
2,6-dichlorobiphenyl



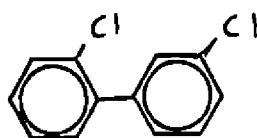
3,4-dichlorobiphenyl



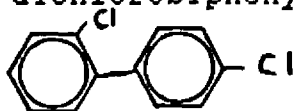
3,5-dichlorobiphenyl



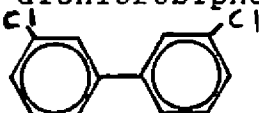
2,2'-dichlorobiphenyl



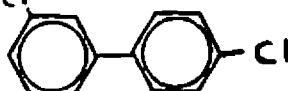
2,3'-dichlorobiphenyl



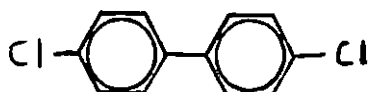
2,4'-dichlorobiphenyl



3,3'-dichlorobiphenyl

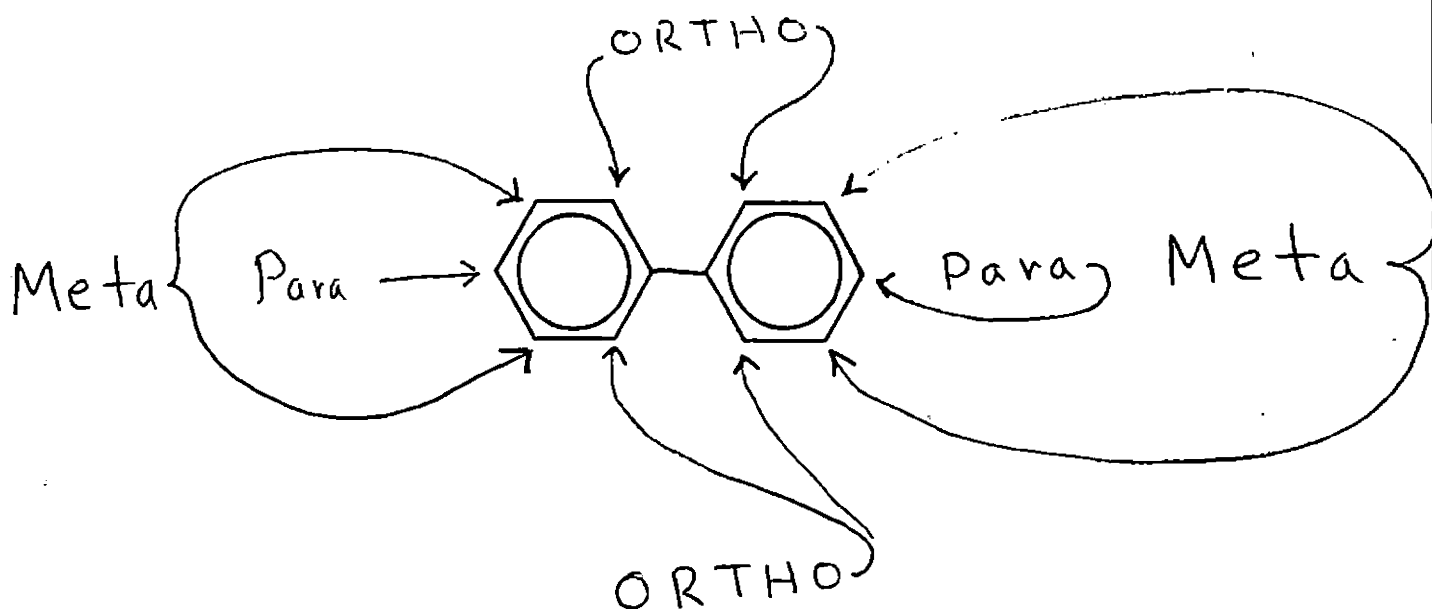


3,4'-dichlorobiphenyl



4,4'-dichlorobiphenyl

Biphenyl molecules are also designated by the terms "ortho," "meta," and "para." As indicated below, there are 4 ortho, 4 meta, and 2 para positions.



PCBs In Water

Because PCBs are practically insoluble in water, they are said to be "hydrophobic". Because they dissolve in fat (lipids), PCBs are said to be lipophilic. PCBs also dissolve in grease, oil, and organic solvents such as common paint thinners. Water contains varying amounts of small suspended particles. PCBs in water tend to be associated with these particles. A PCB molecule contained inside of, or adhering to the surface of a suspended particle is not dissolved in water, i.e., it is not a free molecule "swimming" among water molecules. PCB molecules avoid water by associating with suspended particles.

PCBs in lakes and streams waters tend to concentrate in sediment. The amount of PCBs in contaminated waters increases during heavy rainfall, when the sediment becomes stirred up and the amount of suspended particles increases.

Another result of the lipophilicity and hydrophobia of PCBs is their accumulation in fat tissue. PCBs remain in the body's tissues, particularly the fatty portions, for years. Congeners with fewer numbers of chlorine atoms tend to dissolve better in water. This helps explain why PCB mixtures found in the environment do not resemble commercial mixtures. PCBs which are more water soluble travel better in streams and are more available to microorganisms which can destroy them. Differences in water solubility are only one of many factors which help explain the fact that PCB mixtures in the body and in the wider environment differ from the mixtures which were initially distributed in commerce. The fact that living organisms destroy some PCB congeners much more rapidly than others also helps explain why PCB mixtures change with time.

PCBs in Air

As noted in Section 1:02, PCBs have low vapor pressures, i.e., they have little tendency to evaporate. This does not mean that individual PCB molecules never become airborne; it just means that they tend to dissolve in grease and oil or adhere to solid surfaces.

There are millions of free PCB molecules in practically every cubic inch of air over the surface of the earth. These molecules eventually leave the air to settle on surfaces or in liquids, or they remain airborne by adhering to particles of dust. Conversely, PCB molecules on surfaces or in liquids exposed to the air occasionally take flight. This process is known as "volatilization" or "vaporization." PCBs mixed with solid material also become airborne in the form of dust.

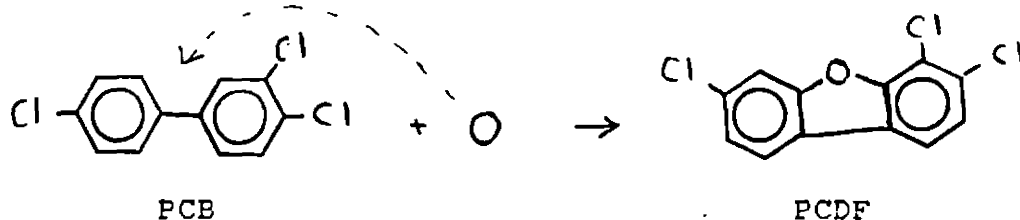
Individual PCB molecules on the earth's exposed surface spend most of their time adhering to solids, and a very small fraction of their time in the vapor state; i.e., in free flight. Volatilization and the movement of dust help explain the fact that PCBs have spread over the world's entire surface.

PCDF AND DIOXIN CHEMISTRY

Polychlorinated dibenzofurans (PCDFs) are a class of extraordinarily toxic compounds which are universal PCB contaminants. They were present in freshly manufactured PCBs, and they are known to increase in concentration when PCBs are exposed to heat and oxygen. Fires involving PCBs are considered to be extremely hazardous because of these byproducts.

It has been suggested that minute traces (usually between 1 and 10 parts per million [ppm]) of PCDFs might account for much of the documented toxicity of commercial PCBs. It is though, in the aftermath of the Japanese mass poisoning catastrophe known as Yusho, that when PCDF concentrations are as high 1000 ppm (0.1%), the toxicity of PCBs is significantly increased.

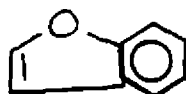
The following diagram shows how a PCDF molecule can form from a single oxygen atom and a PCB molecule.



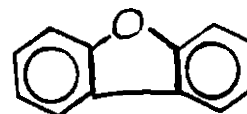
"Benzo-" in chemical nomenclature signifies benzene rings which are attached to other groups of atoms at two adjacent locations. "Benzo-" is very similar to "phenyl-", only phenyl groups are benzene rings attached to other groups of atoms at only one location.



furan



benzofuran



dibenzofuran

There are 135 chlorinated dibenzofuran congeners. These are often referred to as "polychlorinated dibenzofurans" and abbreviated "PCDFs". The PCDFs are similar in structure and in toxicity to the PCDDs. One particularly deadly PCDF is 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF).

2,3,7,8-TCDF

PCDF formation during incineration and in accidental PCB fires is a matter of major concern because of the extreme toxicity of these compounds. Complete combustion of PCBs usually requires the addition of 20 oxygen atoms to the ten carbon atoms of biphenyl to form ten molecules of carbon dioxide.¹ Oxygen also adds to hydrogen atoms during combustion to form water. Complete combustion requires plenty of oxygen, very high temperatures, and sufficient reaction time. At higher temperatures, less time is required.

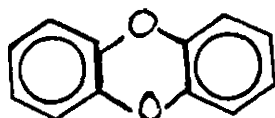
PCDFs are one of the many products of incomplete PCB combustion. They result from the addition of only one oxygen atom to a PCB molecule. When an oxygen atom reacts with a PCB molecule to form a dibenzofuran molecule, the two PCB rings are prevented from spinning relative to one another. The molecule becomes locked into a planar (flat) configuration in which it closely resembles the family of very toxic compounds known as the dioxins.

1. Complete combustion can also result in the formation of phosgene, which has the formula CCl_2O . The other biproducts of PCB combustion are hydrogen chloride (HCl) and water (H_2O).

Dioxins

The dioxins are relevant to a discussion of PCBs because their toxicities are currently being investigated more extensively than the toxicities of PCBs, yet it is widely thought that dioxins and many of the PCB congeners share a similar mode of toxic action.¹ Moreover, dioxins are formed in PCB transformer fires.² Since dioxins strongly resemble PCBs and PCDFs, and are found in combination with them after transformer fires, it is appropriate for those interested in PCBs to gain some familiarity with them.

A dibenzo-p-dioxin molecule has the structure:

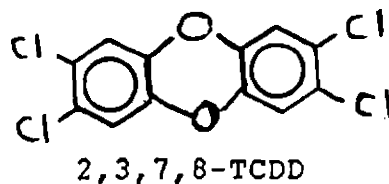


The "p" in dibenzo-p-dioxin stands for "para", signifying that the oxygen atoms are on opposite sides of the central ring. The central ring never has oxygen atoms at other locations.

There are 75 chlorinated dibenzo-p-dioxin congeners (dioxins). These are often called "polychlorinated dibenzo-p-dioxins" and given the abbreviation "PCDD's". The tetrachlorodibenzo-p-dioxins (TCDDs) are thought to be the most toxic dioxins. One of these, 2,3,7,8-tetrachlorodibenzo-p-dioxin is the most acutely toxic of them all for many species of animal. Some say it is the most potent man made poison, although people also manufacture plutonium, an even more deadly, naturally occurring substance.

The atoms in dioxin molecules are locked into a planar (flat) configuration. Considerable discussion has been generated by the observation that when the two rings of a PCB molecule are in the same plane, i.e., when a PCB molecule is flat, it resembles a dioxin molecule. The more chlorine atoms which are placed on carbon atoms adjacent to the carbon-carbon double bonds, the more strained a planar configuration becomes due to crowding by the large chlorine atoms. Chlorine in these positions tends to reduce the dioxin-like acute toxicity of PCBs. PCB congeners containing four or five chlorine atoms, but no chlorine adjacent to the carbon-carbon bond, are potent acute toxins, comparable in strength to the dioxins

themselves.



1. Safe, Polychlorinated Biphenyls (PCBs) and Polybrominated Biphenyls (PBBs): Biochemistry, Toxicology, and Mechanism of Action, 13 CRC Crit Rev Toxicol 319 (1985).
- 2.. Hutzinger et al., Formation of Polychlorinated Dibenzofurans and Dioxins during Combustion, Electrical Equipment Fires and PCB Incineration, 60 Environ Health Perspect 3 (1985).

Tradenames Assigned to Monsanto's PCBs

Aroclor	Monsanto	Monsanto
Pydraul	Monsanto	(Hydraulic Fluid)
Pyroclor		Monsanto
Therminol	Monsanto	(Heat transfer fluid)
Santovac	Monsanto	(Vacuum-pump oil)
Turbinol	Monsanto	(Compressor fluid)
Hyvol		Aerovox
Hyvol	Universal Manufacturing Co.	
Asbestol	American Corporation	
Chlorextol	Allis-Chalmers	
Clorphen	JARD Corp.	
Clorphen	York Electronics	
Dykanol	Cornell Dubilier	
Eucarel	Electric Utility Co.	
Eucarel	Capacitor Specialists, Inc.	
EEC-18	Niagra Transformer Corp.	
EEC-18	Helena Corp.	
EEC-18	Hevi-Duty Electric	
Dykanol	Federal Pacific	
Pyranol	General Electric	
Pyranol	Research-Cottrell	
Saf-T-Kuhl	Kuhlman Electric	
Saf-T-Kuhl	Electro Engineering Works	
Saf-T-Kuhl	Envirotech Buell	
Saf-T-Kuhl	R.E. Uptegraff Mfg. Co.	
Saf-T-Kuhl	H.K. Porter	
Saf-T-Kuhl	Van Tran Electric Co.	
Elemex	McGraw Edison	
Elemex	RF Interonics	
Elemex	Axel Electronic, Inc.	
Elemex	Tobe Deutschmann Labs	
Elemex	Cine-Chrome Lab, Inc.	
Aroclor B	P.R. Mallory & Co.	
Diaclor	Sangamo Electric	
Chlorinol	Sprague Electric Co.	
No-Flamol	Wagner Electric	
Inerteen	Westinghouse	
Sanotherm		

ENVIRONMENTAL EXPOSURE TO PCBs

Introduction

Everyone is contaminated by PCBs. Evidence for this includes the results of widespread testing of blood, adipose (fat) tissue and milk;¹ and the fact that the earth's entire lower atmosphere is contaminated.² A report which appeared in the Annals of the New York Academy of Science in 1979 states: "At some time or other in the last 50 years, PCBs have become current constituents of the human body and the biologic environment."³ 99% of all Americans reportedly have over 500 ug/kg (ppb) PCBs in their adipose tissue.⁴

This report deals with the spread of PCBs throughout the world. Revelations about global contamination help explain why Congress placed severe restrictions on the use of PCBs in 1976, and why Monsanto limited sales in 1972 and completely stopped production in 1977.

1. Slorach and Vaz, PCB Levels in Breast Milk: Data from the UNEP/WHO Pilot Project on Biological Monitoring and Some Other Recent Studies, 60 Environ Health Perspect 121 (1985); Kreiss, Studies on Populations Exposed to Polychlorinated Biphenyls, 60 Environ Health Persp 193 (1985).

2. Oehme and Mano, The Long-Range Transport of Organic Pollutants to the Arctic, 319 Anal Chem 141 (1984); Doskey and Andren, Modeling the Flux of Atmospheric Polychlorinated Biphenyls across the Air/Water Interface, 15 Environ Sci Tech 705 (1981); Atlas and Giam, Global Transport of Organic Pollutants: Ambient Concentrations in the Remote Marine Atmosphere, 211 Science 163 (1981); Tanabe et al, PCBs and Chlorinated Hydrocarbon Pesticides in Antarctic Atmosphere and Hydrosphere, 12 Chemosphere 277 (1983); Harvey and Steinhauer, Atmospheric Transport of Polychlorobiphenyls to the North Atlantic, 8 Atmos Environ 777 (1974); Chlorinated Hydrocarbons in the Sargasso Sea Atmosphere and Surface Water, 183 Science 516 (1974); Biddleman et al., Atmospheric Transport of Organochlorines in the North Atlantic Gyre, 39 J Mar Res 443 (1981); Giam et al., Phthalate Esters, PCB, and DDT Residues in the Gulf of Mexico Atmosphere, 14 Atmos Environ 65 (1980).

3. Wasserman, World PCBs Map: Storage and Effects in Man and His Biologic Environment in the 1970s, 320 Ann NY Acad Sci 69 (1979). This article is an excellent review of the topic.

environment."

One of Monsanto's slides at the presentation reads:

CAPACITOR FLUIDS

WHY CONVERT

TO AROCLOR 1016

- 9-fold reduction in PCBs of pentachloro and higher
- 10-fold reduction in PCBs of hexachloro- and higher
- minimum industry testing required

No mention was made of the fact that Aroclor 1016 provided a 1000 fold reduction in PCDFs.¹

1. Milby et al, PCB-Containing Transformer Fires: Decontamination Guidelines Based on Health Consideration, 27 J Occup Med 351 (1985).

4. U.S. EPA, Environmental News, May 9, 1983.

PCBs in the General Environment

Over 10% of the approximately 1.3 billion pounds of PCBs which were produced in the United States between 1929 and 1979 may be free in the environment.¹ These PCBs, along with an approximately equal amount produced in and released from other countries, have spread throughout the earth's lower atmosphere and over its entire surface. They are likely to remain a feature of our planet for centuries to come.

"Background" PCB levels are the concentrations typically found in the absence of any particular source of pollution. Background air levels in the most remote parts of the world reportedly are about 0.1 ng/cu.m.² Background levels in biota³ and human beings⁴ have also been documented. Typical human levels are around 5ug/l in blood and 1mg/kg in fat, varying with geographic location, diet, and work history.

1. U.S. EPA, PCBs in the United States Industrial Use and Environmental Distribution, EPA 560/6-76-005 (1976).
2. Atlas and Giam, Global Transport of Organic Pollutants: Ambient Concentrations in the Remote Marine Atmosphere, 211 Science 163 (1981).
3. Wasserman, et al., World PCBs Map: Storage and Effects in Man and His Biologic Environment in the 1970's, 320 Ann NY Acad Sci 69 (1979).
4. Jensen and Sundstrom, Structures and Levels of Most Chlorobiphenyls in the Technical PCB Products and in Human Adipose Tissue, 3 Ambio, 70 (1974); F.W. Kutz and S.C. Stressman, Residues of Polychlorinated Biphenyls in the General Population of the United States, National Conference on Polychlorinated Biphenyls, Chicago, p 139 (1975); New York State Planning Commission, Report of the Ad Hoc Committee on the Health Implications of PCBs in Mothers' Milk. (1977); Holdrinet et al., Organochlorine Residues in Human Adipose Tissue and Milk From Ontario Residents, 1969-1974, 68 Can J Publ Health 74 (1977); Mes et al., Polychlorinated Biphenyl and Organochlorine Pesticide Residues in Adipose Tissue of Canadians, 17 Bull Environ Contam Toxicol 581 (1977); Mes and Davies, Presence of Polychlorinated Biphenyl and Organochlorine Pesticide Residues and the Absence of Polychlorinated Terphenyls in Canadian Human Milk Samples, 21 Bull Environ Contam Toxicol 381 (1978); Cordle et al., Human Exposure to Polychlorinated Biphenyls and Polybrominated Biphenyls, 24 Environ Health Perspect 157 (1978); Mes and Davies, Variations in the Polychlorinated Biphenyl and Organochlorine Pesticide Residues During Human Breast Feeding and its Diurnal Pattern, 7 Chemosphere 699 (1978); Barr, Environmental Contamination of Human Breast Milk, 71 Am J Public Health 124 (1981); Eckenhausen et al., Organochlorine Pesticide Concentrations in Prenatal Samples from Mothers and Babies, 36 Arch Environ Health 81 (1981); Safe, Halogenated Hydrocarbons and Aryl Hydrocarbons Identified in Human Tissues, 5 Toxicol Environ Chem 153 (1982); Baluga et al., Presence of Organochlorine Pesticides, Polychlorinated Biphenyls and Mercury in Spanish Human Milk Samples, 28 Bull Environ Contam Toxicol 573 (1982).

Worldwide Contamination

The discovery of PCBs in the general environment stemmed partly from pesticide research which was being conducted during the 1960s. Knowledge of the ubiquitous presence of DDT and other chlorinated pesticides in our environment became common after Rachel Carson's *Silent Spring* was published in 1962. This knowledge was being gathered by the same procedures which are used to analyze environmental specimens for PCB content: extraction followed by GC/EC. So while they were analyzing samples for pesticides, researchers were also unknowingly recording the presence of PCBs in the environment.

Then, in 1966, the annoying interference peaks which routinely appeared on strip chart recordings during pesticide analyses were positively identified as PCBs by the Swedish researcher Soren Jensen. What had been considered merely a nuisance to analytical chemists became a matter of serious concern to researchers and public health officials throughout the world.

The first news of Jensen's discovery appeared in the "Notes on the News" feature in New Scientist magazine in 1966,¹ where it was reported that Soren Jensen was finding PCB contamination throughout Sweden. Dr. Jensen reportedly suggested that PCBs were being deposited from the air into lakes, and that fish were passing it on to humans. The anonymous author also reported that PCBs had been detected in the air over London and Hamburg, and in seals taken from the coast of Scotland. His concluding remark is, "It can therefore be presumed to be widespread throughout the world." Jensen's work was published the following year.² Subsequent research showed that PCBs have become dispersed over the entire surface of the earth,³ presumably by means of atmospheric transport.⁴

PCB levels in some relatively contaminated geographical areas appear to be diminishing, but "pristine" areas are reportedly becoming increasingly contaminated.⁵ This can be attributed to the fact that PCBs are mobile, traveling over the surface of the earth through the air.

A major, and perhaps the major source of PCBs in lake sediment is the air, and now a major source of PCBs in the air may be lake sediment.⁶ Thus, while lakes may have originally served as sinks for PCBs which were released into the environment, at some stage an equilibrium may have been reached, and then the situation reversed and the lakes

became a source of atmospheric PCBs.

Tests involving filtration of particulates and adsorption of vapor phase organics suggest that PCBs move through the atmosphere partly on dust, and partly as free molecules. More chlorinated congeners have a greater tendency to travel on dust. PCBs also flow into the environment from municipal and industrial discharges, as well as from rainwater runoff. They find their way into our food, water, and air through many routes. Leaking or exploding electrical equipment - such as power grid transformers and capacitors - probably account for most of the PCBs currently being released into the environment.

Widespread human contamination was reported in 1972.⁷ One article indicated that 41 - 45% of the general population had more than 1 ppm combined penta-, hexa-, hepta-, and octachlorobiphenyl in their adipose tissue.* The following year, a number of human milk samples collected in Colorado were found to contain high levels of PCBs.*

In order to curb the rate of environmental contamination by PCBs, Monsanto, America's sole producer, notified its customers in 1971 that sales were being restricted to firms using PCBs in closed systems for which there were no practical PCB substitutes. This eliminated its use in paint, asphalt, putty, and pesticide formulations; its use as a petroleum product additive; and even relatively closed uses in heat exchange and hydraulic fluid applications. etc. Monsanto narrowed its customer list to capacitor, transformer, and electromagnet manufacturers, but this did not eliminate dissemination into the environment. It was estimated in 1976 that 5% of the PCBs used in electrical equipment eventually escapes into the environment.¹⁰

There are indications that background PCB levels are declining in some parts of the world, presumably as a result of curtailed production, use, and dumping.¹¹ It is, however, difficult to draw conclusions from the test data because results are very imprecise, and methods vary from lab to lab and year to year.¹²

As part of their effort to minimize environmental contamination, the U.S. EPA requires that the liquids containing more than 500 ppm PCBs be incinerated¹³ unless an equally effective alternative is used.¹⁴ Such alternatives might be necessary in view of the fact that incineration of PCBs and PCB contaminated equipment releases harmful combustion byproducts into the environment, including PCDFs,

dioxins,

and

heavy

metals.

1. Anon., Report of a New Chemical Hazard, 32 New sci, 621 (1966).
2. Wildmark, Possible Interference by Chlorinated Biphenyls, 50 J Assoc Off Anal Chem 1069 (1967).
3. Ballschmiter, Baseline Studies of the Global Pollution, 306 Fres Z Anal Chem 323 (1981); Ballschmiter et al, Studies of The Global Baseline Pollution, 309 Fres Z Anal Chem 1 (1981); Wasserman, et al., World PCBs Map: Storage and Effects in Man and His Biologic Environment in the 1970's, 320 Ann NY Acad Sci 69 (1979); Kutz, et al., Survey of Selected Organochlorine Pesticides in the General Population of the United States: Fiscal Years 1970-1975, 320 Ann NY Acad Sci 60 (1979); Higuchi, Ed., PCB Poisoning and Pollution. Kodansha, Tokyo (1976); Fishbein, Chromatographic and Biological Aspects of Polychlorinated Biphenyls, 68 Journal of Chromatography 345 (1972); Risebrough, et al., Polychlorinated Biphenyls in the Global Ecosystem, 220 Nature 1098 (1968).
4. U.S. EPA Project Report R 805325 (1981). See also: Tanabe, et al., PCBs and Chlorinated Hydrocarbon Pesticides in Antarctic Atmosphere and Hydrosphere, 12 Chemosphere 277 (1983); Atlas and Giam, Global Transport of Organic Pollutants: Ambient Concentrations in the Remote Marine Atmosphere, 211 Science 163 (1981); Eisenreich et al., 15 Environ Sci Tech 30-38; Harvey and Steinhauer, Atmospheric Transport of Polychlorobiphenyls to the North Atlantic, 8 Atmos Environ 777 (1974); Biddleman and Olney, 183 Science 517-518.
5. Stout, What is Happening to PCBs, p. 164, in: Waid, PCBs and the Environment, Vol I (1986).
6. It has recently been estimated (Murphy et al., Polychlorinated Biphenyl Emissions to the Atmosphere in the Great Lakes Region. Municipal Landfills and Incinerators, 19 Environ Sci Tech 942 [1985]) that well over 18,000 kg of PCBs are in the air over the United States at any given time, and that 900,000 kg annually cycle through the atmosphere over the United States. This implies that the average deposition rate is 60 g/(km²/year), a figure which is in agreement with deposition rates into the Great Lakes estimated in: Murphy et al., Duluth Environmental Research Laboratory, U.S. EPA Project Report R-805325 (1981); See also: Murphy et al., Physical Behavior of PCBs in the Great Lakes. pp 49+ (1983); Eisenreich et al., Eisenreich Ed., Atmospheric Inputs of Pollutants to Natural Waters, pp 398-

407 (1981); Strachan and Huneault, 5 J Great Lake Res. 61 (1979); Swain, 4 J Great Lakes Res 398 (1978); Murphy and Rzeszutko, 3 J Great Lakes Res 305 (1977).

7. A.R. Yobs, Levels of Polychlorinated Biphenyls in Adipose Tissue of the General Population of the Nation, 1 Environ Health Persp 79 (1972).

8. H.A. Price and R.L. Welch, Occurance of Polychlorinated Biphenyls in Humans, 1 Environ Health Perspect 73 (1972).

9. Savage et al., Organochlorine Pesticide Residues and Polychlorinated Biphenyls in Human Milk, Colorado - 1971-1972, 7 Pesticide Monitoring J 1 (1973).

10. U.S. EPA, PCBs in the United States Industrial Use and Environmental Distribution 286 (1976).

11. Passivirta and Linko, Environmental Toxins in Finnish Wildlife. A Study of Trends of Residue Contents in Fish During 1973-1978, 9 Chemosphere 643 (1980); also, On May 9, 1983, EPA's "Environmental News" reported the results of a study involving 8,593 human beings whose fat tissue or milk were tested beginning in 1972. The Study showed that 99% of the population had at least 0.5ppm in their fat, and that the percentage of the population with over 3ppm in their fat declined from 8% in 1977 to about 1% in 1981. That number had risen from 2.7% in 1972 to 8% in 1977.

12. Stout, What is Happening to PCBs, p. 167, in: Wald (ed), PCBs and the Environment, Vol I (1986). Referring to: Great Lakes Water Quality Board, 1981 Report on Great Lakes Water Quality, Appendix, Great Lakes Surveillance, Report to the International Joint Commission, November, 1981.

13. 40 CFR Part 761.60 (a)(1).

14. 40 CFR Part 761.60 (e).

Bioaccumulation

PCBs are not readily metabolized or excreted by living organisms, and hence they tend to build up. This buildup is called "bioaccumulation". Bioaccumulation has been widely observed in nature, and has been studied in animal experiments. A diet containing 25 ppm PCBs caused levels in monkey adipose tissue to rise to 127 ppm after two months.¹

Many toxins are either rapidly excreted from the body, or rapidly destroyed by metabolic processes. Large chlorinated hydrocarbons,² however, tend to resist both of these processes. They are not rapidly eliminated because they are lipophilic (attracted to fats) and hydrophobic (repelled by water), and hence become stored in fat tissue. Storage in fat deposits also helps prevent metabolism by keeping chlorinated hydrocarbons out of circulation and away from important centers of detoxification such as the liver, lungs, and skin. Metabolism is also slow because most chlorinated hydrocarbons have been introduced to our planet during the past fifty years, so enzyme systems which can efficiently metabolize them have not had time to evolve.

Bioaccumulation is reversible.³ Once exposure is reduced, the combined rates of PCB metabolism and excretion can overtake accumulation. Since each PCDF and PCB congener declines at its own rate, the mixture PCBs in each person's body bears little resemblance to the mixtures to which they were exposed. Gas chromatograms (see Section 1:14) of PCBs extracted from human blood and adipose tissue have larger later eluting (see see Section 1:14) peaks and smaller earlier eluting peaks than gas chromatograms of the PCB mixtures to which the tissue donors have been occupationally or environmentally exposed. The main reason for this is that the earlier eluting peaks tend to contain fewer chlorine atoms, and PCBs with fewer chlorine atoms tend to be metabolized and excreted more rapidly by the body.

But while there is a tendency for higher chlorinated PCBs to accumulate and remain longer in fat tissue, it is only a gross tendency; something which is observed on the average, but for which exceptions are common. A striking exception is decachlorobiphenyl (DCB), the most chlorinated of all the PCBs. DCB does not readily bioaccumulate and, once in fatty tissue, it has little tendency to remain.⁴

1. Allen and Norback, Pathobiological Responses of Primates to Polychlorinated Biphenyl Exposure, National Conference on Polychlorinated Biphenyls 43, EPA - 560/6-75-004 (1976).

2. A "hydrocarbon" is a chemical compound composed of carbon and hydrogen atoms. A "chlorinated hydrocarbon" contains chlorine atoms in addition to carbon and hydrogen. PCBs, DDT, and dioxin are examples of chlorinated hydrocarbons.

3. For a discussion of the dynamics of body burden versus level of exposure, see Gaffy, The Epidemiology of PCBs, in PCBs: Human and Environmental Hazards, page 279 (1983).

4. Platinow and meads, Distribution and Excretion of Two Chlorinated Biphenyl Isomers: 4-Chlorobiphenyl and Decachlorobiphenyl in Lactating Bovine, 39 Can J Comp Med 104.

Differential Bioaccumulation

Each of the 209 PCB congeners has its own, unique propensity to accumulate in any given organism. Differences depend upon the congeners, the species, and the species member. Individual biochemistry varies perhaps to a greater degree than personal appearance, so patterns of PCB accumulation are different for different people. Individual metabolisms change with age and exposure to chemicals. But despite individual idiosyncrasies, there are tendencies in PCB bioaccumulation which apply throughout nature. Some observations concerning bioaccumulation listed below.

- 1) As stated above, higher chlorinated congeners have a greater tendency to bioaccumulate.¹
- 2) Those congeners which have the greatest tendencies to accumulate also tend to elute latest from gas chromatographs. As time goes on, chromatograms of specimens taken from occupationally exposed workers exhibit progressively less resemblance to chromatograms of the PCBs to which they were exposed. In particular, the proportion of later eluting peaks to earlier eluting ones increases with time.
- 3) Congeners which have the greatest tendencies to bioaccumulate also tend to elute latest from analytical devices called "high pressure liquid chromatographs."²
- 4) Octanol floats on water. The tendency of a compound to go into the octanol layer correlates with its solubility in fat. Compounds which tend to enter the octanol layer above water tend to bioaccumulate.³
- 5) PCB congeners with adjacent hydrogen atoms tend to be metabolized faster than those in which each hydrogen atom is straddled by chlorine and/or carbon atoms.

As is the case with the other "rules", there are exceptions.⁴

It is possible that gas chromatography could be used to prove past massive exposure to PCBs, even in cases in which total PCB levels have returned to the normal range. The evidence would lie in the patterns; in the proportions of the individual congeners, one to another. Perhaps there are barely detectable, persistent congeners which add little to total PCB content after massive exposure, but which are significantly more abundant in previously exposed people,

even after total levels have nearly returned to normal.

Years after the poisoning episode at Yusho, PCB levels in the livers of some victims did nearly return to normal, while levels of some PCDF congeners remained drastically elevated.⁵ The ratio of PCB to PCDF in the contaminated rice oil was about 2,000:1, while the ratio reported in some liver biopsy specimens taken years later was 4:1. Similar ratio enrichments involving only PCBs probably exist, but researchers have yet to provide detailed documentation.

There is a strong correlation between age and PCB levels. This might be because metabolism rates tend to become slower with age, possibly due to liver impairment. It could even be because younger people lived a smaller percentage of their lives during the years of maximum environmental contamination.

It is not true that each of us accumulates a little more PCB each day; in fact, average body burdens appear to be declining.⁶ When the rate of PCB intake is constant, there is a tendency for levels in the body to reach a plateau and remain constant⁷ At this stage, the rate of excretion is equal to the rate of intake. Subsequent lowering of intake levels would be expected to result in declining levels in body tissues.

1. Kreiss et al, Association of Blood Pressure and Polychlorinated Biphenyl Levels, 245 JAMA 2505 (1981). Also, The results of the National Human Adipose Tissue Survey conducted by the U.S. EPAs office of Toxic Substances, released on May 9, 1983, show declining levels.

2 Rats reached equilibrium in six months according to Burse et al., Polychlorinated Biphenyls. Storage, Distribution, Excretion, and Recovery: Liver Morphology After Prolonged Dietary Ingestion, 29 Arch Environ Health 301 (1974).

5. Platinow and meads, Distribution and Excretion of Two Chlorinated Biphenyl Isomers: 4-Chlorobiphenyl and Decachlorobiphenyl in Lactating Bovine, 39 Can J Comp Med 104.

4. Matthews et al, Effects of Chlorination on the Distribution and Excretion of Polychlorinated Biphenyls, 3 Drug Metab Disp 371 (1975); Sugiura et al, Accumulation of Polychlorinated and Polybrominated Biphenyls in Fish: Limitation of Correlation Between Partition Coefficients and Accumulation Factors, 7 Chemosphere 731 (1978); Gage and Holm, The Influence of Molecular Structure on the Retention and Excretion of Polychlorinated Biphenyls by the Mouse, 36 Toxicol Appl Pharmacol 555 (1976); Morales, Pharmacokinetics of Multiple Oral Doses of Selected Polychlorinated Biphenyls in Mice, 48 Toxicol Appl Pharmacol 397 (1979); Bungay et al, Pharmacokinetics of Halogenated Hydrocarbons, 320 Ann NY Acad Sci 257 (1979). For information concerning the degree of chlorination on rates of metabolism, see: Hutzinger et al, Polychlorinated Biphenyls: Metabolic Behavior of Pure Isomers in Pigeons, Rats and Brook Trout, 178 Science 312 (1972); De Freitas and Norstrom, Turnover and Metabolism of PCBs in Relation to Their Chemical Structure and the Movement of Lipids in the Pigeon, 52 Canadian J Physiol 1080 (1974); Burse et al, PCB Metabolism in Rats Following Prolonged Exposure to Aroclor 1242 and Aroclor 1016, 15 Bull Environ Contam Toxicol 122 (1976).

5. Sugiura et al, Accumulation of Polychlorinated and Polybrominated Biphenyls in Fish: Limitation of Correlation Between Partition Coefficients and Accumulation Factors, 7 Chemosphere 731 (1978).

6. Kreiss et al, Association of Blood Pressure and Polychlorinated Biphenyl Levels, 245 JAMA 2505 (1981). Also, The results of the National Human Adipose Tissue Survey conducted by the U.S. EPAs office of Toxic

Substances, released on May 9, 1983, show declining levels.

7. Rats reached equilibrium in six months according to
Burse et al., Polychlorinated Biphenyls. Storage,
Distribution, Excretion, and Recovery: Liver Morphology
After Prolonged Dietary Ingestion, 29 Arch Environ Health
301 (1974).

Biomagnification

Because organisms accumulate PCBs, their tissues contain higher concentrations of PCBs than their environment. Fish contain much higher levels than the sediment and water of their habitat. Birds which eat fish have higher levels than their prey.¹ The process whereby living organisms build up higher PCB concentrations than their environments is called "biomagnification".

The concept of "food chain" is often used in discussions of biomagnification. Vegetation is low on the food chain, fish which eat vegetation are higher, and fish which eat other fish are higher yet. Humans who are not vegetarians are high on the food chain. The blood of people who eat fish from Lake Michigan has been measured at levels as high as 366 ppm, a concentration which is around 50 times the average level and considerably higher than concentrations found in the fish.²

1. Tulp and Hutzinger, Some Thoughts on Aqueous Solubilities and Partition Coefficients of PCB and the Mathematical Correlation Between Bioaccumulation and Physico-Chemical Properties, 7 Chemosphere 849 (1978).

2. Hutzinger et al, Chemicals With Pollution Potential. In: Hutzinger et al (eds), Aquatic Pollutants: Transformations and Biological Effects, Page 13 (1978).

Conclusion

Litigation concerning the contamination of persons, livestock, or property by PCBs often involves laboratory tests to determine PCB concentrations. The results of these tests can be crucial, particularly when their purpose is to establish whether or not a cause of action exists; and if so, what its settlement value might be. An understanding of background PCBs - both total levels and congener distribution - is crucial in assessing the significance of the test results.

Background levels in humans vary depending upon geographic location and eating habits. Plaintiffs attempting to demonstrate that the actions or negligence of specific defendants caused increases in their PCB levels must take geography and diet into account in gauging what their levels would be without the alleged exposure. Since everyone is contaminated by PCBs, the mere presence of PCBs in the human body is not sufficient to bring a cause to trial. PCB levels become legally significant only when they are above background.

Total PCB levels decline after exposure ceases, so the absence of above average PCB levels years after exposure does not prove that exposure did not occur. Future litigation will likely involve not just total PCB levels, but levels of persistent congeners. Gas chromatographic patterns contain information which is rarely examined, but which could prove very useful in disposing of claims involving alleged exposure.

In environmental cases, gas chromatographic patterns can provide critical evidence. Gas chromatograms of background PCBs do not resemble commercial PCB products. If the pattern produced by a soil or sediment sample is nearly identical to one of Monsanto's Aroclors, it is likely that PCBs were spilled or a nearby source of the contamination can be found.

EXAMPLE: The Bloomington, Indiana area contains numerous sites which are contaminated with PCBs originating from a local capacitor manufacturing plant. When PCBs were discovered in lake sediment at the city's main water intake pumps, some local residents were inclined to blame the capacitor manufacturer. It was shown, however, that the lake sediment contained Aroclor 1254, the PCB mixture which was in the hydraulic fluid used to actuate the water pump valves. Aroclor 1254 is not found at any of the identified

capacitor dump sites, and is not thought to have been used by the Bloomington manufacturing facility. No action against the manufacturer was called for, and the city employee who discovered the problem was reprimanded.

ALLEGATIONS OF FRAUD

Plaintiffs in civil suits have alleged that Monsanto and other companies fraudulently concealed from their customers and the public the dangerous nature of PCBs. At least one plaintiff, the Electric Power Board of Chatanooga, alleges that manufacturers of PCB contaminated electrical equipment fraudulently advertized and sold the equipment as PCB free.² The Board alleges that transformers were filled with PCB contaminated mineral oil at the time of manufacture.

The Electric Power Board of Chatanooga further alleges that the Monsanto Company fraudulently concealed the toxicity of PCBs from government regulators and from purchasers of PCB containing electrical equipment. The Board's contentions parallel those of the City of Bloomington,² which amended its environmental pollution complaint to allege that Monsanto violated the Racketeer Influenced and Corrupt Practices Act by misleading the U.S. EPA and FDA.³ Plaintiffs allege that Monsanto made misleading representations to the federal government in its report to the Interdepartmental Task Force,⁴ that Monsanto and electrical equipment manufacturing industry attempted to sidetrack governmental regulatory efforts through the American National Standards Institute C-107 Committee, and that the alleged pattern of concealment persisted up unto recent times. The reliance by Monsanto on the test results of the discredited Industrial Bio-test Laboratories is a major component of the fraud allegations.

1. Electric Power Board of Chatanooga, et al. vs. Monasanto et al, U.S. Dist East Tenn, Southern Div, Civ 1-85-634.
2. The City of Bloomington, Indiana et al. vs. Westinghouse Electric Corporation and Monsanto Company, U.S. Dist South Ind, Indianapolis Div, IP 83-9-C.
3. Title 18 U.S.C. Section XXXXXXXX
4. Monsanto Company, Presentation to the Interdepartmental Task Force on PCBs, Washington, D.C., May 15, 1972

INDUSTRIAL BIO-TEST SCANDAL

A special 1980 federal grand jury brought charges mail and wire fraud and against officials of Industrial Bio-Test Laboratories (IBT), a contract research laboratory which conducted animal studies to determine the safety and/or effectiveness of various drugs, pesticides, chemicals, and other substances. The grand jury charged former IBT president Joseph Calandra, former IBT Manager of Toxicology Moreno Keplinger, former Section Head for Rat Toxicology (who returned to Monsanto in 1972) Paul Wright, and former Assistant to the Manager of Toxicology James Plank with mail fraud, wire fraud, and making false statements to the U.S. Food and Drug Administration (FDA). The laws cited were 18 USC Section 1341, 18 USC Section 1343, and 18 USC 1001. The charges stemmed from IBT's alleged submission of false laboratory studies to the FDA. A mistrial was declared in Calandra's case, and the other three men were convicted. The convictions were upheld in 1985.²

During their trials, IBT officials were not accused of falsifying PCB tests. It was only later, in civil cases, that such accusations were formally lodged.³ Plaintiffs contend that Monsanto knowingly submitted fraudulent test results to the U.S. Environmental Protection Agency in order to prevent or delay regulatory action. In Electric Power Board et al. vs. Monsanto et al., plaintiffs hope to reverse summary judgement based on expiration of Tennessee's 10 year statute of repose by persuading the court that "Monsanto engaged in a conspiracy with its principal testing laboratory to falsely conceal the harmful effects of PCBs with the purpose and effect of concealing from the Plaintiffs the existence of the claims set forth in this case, as well as concealing like claims from others similarly situated." In support of this allegation, Chattanooga contends that in order to conceal the dangerous characteristics of PCBs, Monsanto used poorly executed, "fudged", and altered test results.

Photocopies of alleged correspondence between IBT and Monsanto indicates that in 1971, IBT granted Monsanto's request that a report stating PCBs are "slightly tumorigenic" be changed to read "does not cause cancer." Another letter indicates that in 1975, IBT offered to assist Monsanto "in any adversary situation in or out of government."

1. United States of America vs. Joseph C. Calandra, Moreno L. Keplinger, Paul L. Wright and James B. Plank, U.S. District Court, Northern District of Illinois, Eastern Division, No. 81 CR 335 (1980).

2. 776 F.2d 678 (7th Cir. 1985)

MONSANTO'S REPORT TO THE INTERDEPARTMENTAL TASK FORCE

The Interdepartmental Task force which was established between various departments of the federal government to investigate PCBs sought information from industrial sources. Monsanto made a presentation to the Task Force in which it did not discuss the toxicity of PCBs. They have subsequently been criticized for discussing animal bioaccumulation tests without mentioning that test animals developed tumors.

Monsanto's presentation was geared to show that only PCB congeners having five or more chlorine atoms accumulate in the environment. E.S. Tucker concluded his remarks on behalf of Monsanto by stating that "...the bulk of the PCB homologs released to the environment (PCBs with less than 5 chlorines) are subject to environmental degradation of one sort or another at measurable rates and as such have not accumulated."

C. Patton informed the Task Force on behalf of Monsanto that Monsanto had a program to prevent environmental pollution by PCBs. The program was summarized as follows:

- no more sales world-wide except dielectric users;
- restrict over 97% capacitor fluid sales to Aroclor 1016 only;
- eliminate Aroclor 1260 from transformer fluids;
- continue incineration facilities for scrap PCBs;
- encourage dielectric industry through ANSI C-107 to enforce strict environmental control over PCBs.

Monsanto spokesman W.B. Papageorge, in summarizing the presentation, stated:

"For the capacitor application the development of Aroclor 1016, which satisfied all of the industry's requirements relating to dielectric characteristics and handling properties as well as having Underwriter Laboratory fire-resistance approval, permits the continued use of PCBs in this important hermetically sealed application but with a fluid that has significantly lower content of the slower degrading isomers. The use of this material accompanied by proper handling and disposal of the scrap fluid by high temperature incineration represents in our considered opinion a significant step forward in our efforts to control the impact of PCBs on the

MONSANTO CORRESPONDENCE

Some Monsanto correspondence has been placed in government dockets or submitted as plaintiff's exhibits in PCB cases. Some of these documents are described below.

1) A December 22, 1949 letter from the Barnard Frek Skin and Cancer Hospital to Monsanto's medical director, Emmet Kelly, informing him that skin patch tests indicated that "it would be safe to assume that individuals may be exposed to Aroclor."

2) A July 14, 1951 letter from the Barnard Frek Skin and Cancer Hospital to Emmet Kelly of Monsanto stating that skin patch tests indicated "it would be safe to assume that individuals may be exposed to Pydraul."

3) A letter dated March 3, 1969, signed by Monsanto's Manager of Environmental Health, Elmer P. Wheeler, was mailed to some purchasers of PCB. Mr. Wheeler questions Swedish scientists Gunnar Widmark's and Soren Jensen's 1966 announcement of widespread environmental PCB contamination, and Robert Risebrough's 1969 discovery of PCBs in the San Francisco Bay. He states that their conclusions are "puzzling" and that it is "difficult to comprehend how commercially produced PCB can show up in wildlife as DDT and other pesticides appear to be." Wheeler also states that PCBs are "not toxic unless they are mishandled or misused."

4) On June 1, 1970, Monsanto's director of plasticizer sales, Walter Schalk, notified customers that the use of PCBs in synthetic resins may be a source of "the alleged" environmental contamination. Mr. Schalk informed the customers that PCBs would cease to be available from Monsanto for modifier and plasticizer applications effective August 30, 1970.

5) John Mason, whose title at Monsanto was "Assistant General Manager," signed a June 30, 1970 letter to then Congressman William F. Ryan, informing him that Monsanto considered PCBs to be an environmental threat and enumerating steps Monsanto was taking to minimize the problem. Mr. Mason wrote that:

a) effective August 30, 1970, Monsanto would no longer sell PCBs for use in general plasticizer applications where disposal of the end product cannot be controlled;

b) PCBs would be eliminated from Hydraulic fluid formulations by the end of 1970;

c) Monsanto would continue to sell PCBs for use in "closed system applications, e.g. transformers, capacitors, and heat transfer fluids ... for the

safety and well being of the population of this country"

d) Monsanto would continue to accept used PCBs for regeneration or incineration; e) Monsanto was developing a biodegradable PCB mixture;

f) Monsanto would not comply with Congressman Ryan's request for bulletins on PCBs referred to in Monsanto bulletin O/PL-306 because the requested bulletins dealt with applications for which Monsanto no longer provided PCBs;

g) Monsanto would not release a complete list of the uses of PCBs;

h) information concerning annual production and usage of PCBs would only be provided if assurances of confidentiality and restricted use of the data were met.

i) Monsanto officials were meeting with leading research scientists and government officials around the world to discuss PCBs.

6) Early in 1972, Monsanto sent letters to its PCB customers informing them that in order to continue to receive PCBs, they would have to "defend, indemnify, and hold harmless" Monsanto for any problems arising from PCBs. Customers were also asked to provide proof that they held sufficient insurance to back up such an agreement.

EFFECTS ON THE LIVER

Changes in liver chemistry and morphology - including enzyme induction and progression through microsomal proliferation, cellular hypertrophy, focal necrosis, fibrosis, and tumors - are consistently observed in PCB exposed experimental animals.¹ Changes in liver chemistry have been observed in PCB exposed humans but they are often described as mild and "sub-clinical". Morphological changes in the livers of Yusho victims were not readily apparent, even by conventional microscopy, although they were quite obvious under the more powerful electron microscope.²

Morphological manifestations observed in test animals and humans exposed to toxic chlorinated hydrocarbons can serve as a reliable means of documenting exposure. Electron microscopic examination of liver biopsy specimens taken from people exposed to smoke and soot from a transformer fire revealed proliferation of the endoplasmic reticulum.³

Some studies of occupationally exposed workers suggest liver damage may not be a common manifestation of their exposure. This could be because the tests are inadequate, or it could be because humans are resistant to liver damage. While it is to be hoped that the latter is the case, it still would not be generally reassuring. Top species which are extraordinarily susceptible to PCB toxicosis, the mink and the guinea pig, both are resistant to PCB mediated liver damage.⁴

A crucial question regarding biochemical manifestations of exposure is: where do we draw the line between liver damage and sub-clinical biochemical manifestations of exposure? Enzyme induction is a case in point. Industry experts say that drug metabolizing enzymes merely indicate that a healthy liver is ridding the body of unwanted chemicals. There are other experts who are more inclined to interpret elevated enzyme production as a sign of disease. In any event, they are a danger sign that the body will respond abnormally to certain other chemicals.

The latter point raises three important questions. One, are enough enzymes produced to significantly speed up the metabolism of therapeutic drugs, thereby interfering with medical treatment? Two, can PCB induced enzymes react with the body's natural chemicals in a harmful way, and three, can PCB induced enzymes react with foreign chemicals to produce greater amounts of toxic byproducts than otherwise would be expected? No reference to the first two points has surfaced, but evidence of powerful enzyme mediated synergistic effects has appeared.

Such a synergism exists between PCBs and halothane, an anesthetic which usually does not harm the liver. After mice are given a single dose of PCBs, halothane becomes a powerful liver toxin.⁵ Moreover, it has been suggested that an apparent case of halothane induced liver damage

might have been the result of previous occupational exposure to PCBs.⁶ A similar effect involving carbon tetrachloride also exists.⁷

1. (a) Vos and Koeman, Comparative Toxicologic Study with Polychlorinated Biphenyls in Chickens with Special Reference to Porphyria, Edema Formation, Liver Necrosis and Tissue Residues, 17 Toxicol Appl Pharmacol 656 (1970);
- (b) Vos and Beems, Dermal Toxicity Studies of Technical Polychlorinated Biphenyls and Fractions Thereof in Rabbits, 19 Toxicol Appl Pharmacol 617 (1971);
- (c) Allen and Abrahamson, Morphological and Biochemical Changes in the Liver of Rats Fed Polychlorinated Biphenyls, 1 Arch Environ Contam Toxicol 205 (1973);
- (d) Kimbrough et al, Changes in Livers of Rats Fed Polychlorinated Biphenyls, Light Microscopy and Ultrastructure, 25 Arch Ind Health 354 (1972).
- (e) Kimbrough et al., Induction of Adenofibrosis and Hepatomas of the Liver in BALB/CJ Mice by Polychlorinated Biphenyls (Aroclor 1254), 53 J Natl Cancer Inst 547 (1974).
- (f) Kimbrough et al., Induction of Liver Tumors in Sherman Strain Female Rats by Polychlorinated Biphenyl Aroclor 1260, 55 J Natl Cancer Inst 1453 (1975).
- (g) Kimbrough et al, Adenofibrosis in the Rat Liver, 27 Arch Environ Health 390 (1973);
- (h) Kimbrough, The Toxicity of Polychlorinated Polycyclic Compounds and Related Compounds, 2 Crit Rev Toxicol 445 (1974);
- (i) Bruckner et al, Effect of Prolonged Ingestion of Polychlorinated Biphenyls on the Rat, 12 Food Cosmet Toxicol 323 (1974);
- (j) Fishbein, Toxicity of Chlorinated Biphenyls, 14 Annu Rev Pharmacol 139 (1974);
- (k) Allen et al., Responses of Rats Exposed to Polychlorinated Biphenyls for 52 Weeks, Comparison of Tissue Levels of PCBs and Biological Changes, 4 J Arch Environ Contam Toxicol 409 (1976);
- (l) Mathews, Metabolism and Biochemical Toxicity of PCBs and PBB's, 24 Environ Health Perspect 147 (1978);
- (m) Koller and Zinkl, Pathology of Polychlorinated Biphenyls In Rabbits, 23 Environ Health Perspect 153

(1978);

(n) Higuchi Ed., PCB Poisoning and Pollution (1979);

(o) McConnell and Moore, Toxicopathology Characteristics of the Halogenated Aromatics, 320 Ann NY Acad Sci 138 (1979);

(p) McConnell, Acute and Chronic Toxicity, Carcinogenesis, Reproduction Teratogenesis and Mutagenesis in Animals, in Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products, p. 109 (1980);

(q) Parkinson et al., Reconstituted Breast Milk PCBs as Potent Inducers of Aryl Hydrocarbon Hydroxylase, 96 Biochem Biophys Res Commun 882 (1980);

(r) Parkinson and Safe, Aryl Hydrocarbon Hydroxylase Induction and its Relationship to the Toxicity of Halogenated Aryl Hydrocarbons, 4 Toxicol Environ Chem Rev 4 (1981);

(s) Jonsson et al., Effects of Prolonged Exposure to Dietary DDT and PCBs on Rat Liver Morphology, 10 Arch Environ Contam Toxicol 1.1 (1981).

2. Kuratsume, Yusho, in Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products, p. 298 (1980).

3. Schechter and Tiernan, Occupational Exposure to Polychlorinated Dioxins, Polychlorinated Furans, Polychlorinated Biphenyls, and Biphenylenes After an Electrical Panel and Transformer Accident in an Office Building in Binghamton, NY., 60 Environ Health Perspect 305 (1985).

4. Safe, Polychlorinated Biphenyls (PCBs) and Polybrominated Biphenyls (PBB's), 13(4) CRC Critical Reviews 319, 330 (1985).

5. Sipes and Brown, An Animal Model of Hepatotoxicity Associated with Halothane Anesthesia, 45 Anesthesiology 628 (1979).

6. Rosenblatt, A Possible Interaction of PCB and Halothane in Man, 51 Anesthesiology 95 (1979).

7. Dinker et al, The Problem of Possible Systemic Effects from Certain Chlorinated Hydrocarbons, 19 Journal of Industrial Hygiene and Toxicology 283 (1937).

Porphyria

A condition called "chronic hepatic porphyria (CHP)" can develop after massive exposure to PCBs and related compounds.¹ In its initial stages and in its milder forms, CHP is a subclinical condition manifested primarily by an unusual balance of substances called "porphyrins" in the urine. The test for porphyria is routine.

CHP can progress into its most severe form, porphyria cutanea tarda (PCT). This condition is manifested by increased mechanical fragility of the skin; the appearance of blistering with vesicles and ulcerative lesions on the hands, neck, face, and feet after exposure to sunlight; excessive growth of hair, particularly facial hair; hyperpigmentation of the skin; and the presence of not only an unusual porphyrin balance in the urine, but also excessive amounts resulting in pink to brown discoloration. PCT indicates a diseased condition of the liver. Gradual recovery will usually occur after exposure has ceased.

1. Sano et al., Toxicity of Polychlorinated Biphenyl with Special Reference to Porphyrin Metabolism, 59 Environ Health Perspect 137 (1985); also see Crow, Chloracne - An Up To Date Assessment, 21 Ann Occup Hyg 38 (1978). Also, R.A. Neal, Mechanisms of the Biologocal Effects of PCBs, Polychlorinated Dibenzop-dioxins and Polychlorinated Dibenzofurans in Experimental Animals, 60 Environ Health Perspect 41 (1985): "The mechanism by which PCBs cause hepatic porphyria is the best understood of all the biological effects of this mixture of compounds." (citing: (a) Elder, Porphyria caused by hexachlorobenzene and other polyhalogenated aromatic hydrocarbons. In: Heme and hemoproteins (Matteis and Aldridge, Eds.), Springer-Verlag, Berlin, pp. 15.-200 (1978); and (b) Elder and Sheppard, Immunoreactive Uroporphyrinogen Decarboxylase is Unchanged in Porphyria Caused by TCDD and Hexachlorobenzene, 109 Biochem Biophys Res Commun 113 [1982]).

Significance of Hepatic Porphyria

Hepatic porphyria is useful in documenting recent massive exposure to PCBs and related compounds. Persons who receive sudden exposure (firefighters, for example) should be tested for porphyria as soon after the incident as is convenient. Since the onset of porphyria is gradual, the initial test will serve to document the subject's background condition before exposure. If the exposure was high, subsequent tests may show slight changes in the balance of urinary porphyrins. The degree of porphyria which develops will help document the degree of exposure, and the extent to which the liver is able to withstand it.

Exposed persons should be carefully monitored. Potential defendants in lawsuits which might result from accidents are often willing to pay for testing in exchange for some input into the selection of a doctor and medical facility.

Regular monitoring of workers at high risk of exposure has been recommended.¹ People at risk include firefighters and other emergency response personnel, electrical equipment maintenance and repair workers, salvagers, and waste disposal personnel.

1. Strik et al. (Chemical Porphyria, page 192 of Halogenated Biphenyls, Terphenyls, Naphthalenes, and Dibenzodioxins and Related Products [1980]) state: "The routine measurement of urinary porphyrin patterns may represent a valuable tool for human monitoring of workers and of groups in the general population with special exposure, in instances such as the PBB case in Michigan and the TCDD cloud in Seveso. It may even replace some of the parameters currently in use such as serum enzymes like transaminases which certainly cannot be considered as "early indicators" of liver damage."

See also: Strik et al, Coproporphyrinuria and Chronic Hepatic Porphyria Type A Found in Farm Families from Michigan (U.S.) Exposed to Polybrominated Biphenyls (PBB's), in Chemical Porphyria in Man, Strik and Koeman, Eds. Elsevier/North-Holland, Amsterdam (1979) p. 29.

Biochemical Basis of Porphyria

Porphyrins are involved in the production of vital, iron containing substances known collectively as "heme". 85% of heme production takes place in circulating cells and in the bone marrow where heme in the form of the familiar blood pigment hemoglobin is produced.

The other 15% of heme production takes place in the liver. Hepatic heme is involved in enzyme production. The drug metabolizing enzymes cytochrome P-450 and P-448 are based on heme. It is not clear whether the induction of these enzyme systems is related to porphyria.

Hereditary porphyria involves heme production in the bone marrow and in the liver. Chemically induced porphyria involves only the liver. This distinction is part of the basis for making a diagnosis.

Chemicals can cause porphyria by inhibiting the enzyme uroporphyrinogen decarboxylase (UPD). Insufficient activity of this enzyme leads to insufficient heme production, and the body compensates by producing elevated amounts of delta-aminolevulinic acid (ALA). High levels of UPD are responsible for increased porphyrin production and the onset of porphyria. The most potent microsomal enzyme inducers appear to be the most porphyrinogenic of the PCBs. 3,4,3',4'-tetrachlorobiphenyl and 3,4,5,3',4',5'-hexachlorobiphenyl are potent inhibitors of uroporphyrinogen decarboxylase and hence potent inducers of hepatic porphyria.¹

PCB metabolizing enzymes require heme in the form of cytochrome P-450 and cytochrome P-448. The fact that the most potent enzyme inducers are the most potent porphyrinogens may one day help explain exactly how PCBs affect the human body.

1. Sano et al., Toxicity of Polychlorinated Biphenyl with Special Reference to Porphyrin Metabolism, 59 Environ Health Perspect 137 (1985).

EARLY DOCUMENTAION OF PCB TOXICITY

Literature describing the toxicity of chlorinated hydrocarbons, particularly chlorinated naphthalenes, dates back to the late nineteenth century. Documentation of PCB toxicity appeared in the literature as early as 1936. In that year, Louis Schwartz reported that workers involved in PCB manufacture developed chloracne, digestive problems, burning of the eyes, impotence, and hematuria.¹ He wrote that laws should be passed requiring good ventilation, fresh work clothes, frequent washing of the work areas, screening of job applicants and new employees, and periodic examination for symptoms of systemic poisoning for all who work with PCNs and PCBs.

Also in 1936, Jones and Alden² reported an outbreak of chloracne in 1933 among workers involved in manufacturing PCBs. The authors, however, concluded that PCBs were not the major cause, since there appeared to be a relationship between the source of benzene used to make biphenyl and the outbreak of disease.

The first report of animal experiments involving PCBs appeared in 1937.³

1. Schwartz, Dermatitis from Synthetic Resins and Waxes, 26 Am J Pub Health 586 (1936).
2. Jones and Allen, An Acneform Dermatosis, 33 Arch Dermat & Syph 1022 (1936).
3. Drinker, The Problem of Possible Systemic Effects from Certain Chlorinated Hydrocarbons, 19 J Ind Hyg Toxicol 283 (1937).

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Fulton and Matthews, Preliminary Report of the Dermatological and Systemic Effects of Exposure to Hexachloronaphthalene and Chlorodiphenyl, Pennsylvania Department of Labor and Industry Special Bulletin #43 (1936).

Flinn and Jarvik, Action of Certain Chlorinated Naphthalenes on the Liver, 35 Prec Soc Exper Bio Med 118 (1936).

Schwartz, Skin Hazards in American Industry, U.S. Public Health Bulletin No. 229, part II (1936).

Schwartz, Dermatitis from Synthetic Resins and Waxes, 26 Am J Pub Health 586 (1936).

Jones and Allen, An Acneform Dermatosis, 33 Arch Dermat & Syph 1022 (1936).

Drinker et al, The Problem of Possible Systemic Effects from Certain Chlorinated Hydrocarbons, 19 J Ind Hyg Toxicol 283 (1937).

Mayers and Silverberg, Skin Conditions Resulting from Exposure to Certain Chlorinated Hydrocarbons, 20 J Ind Hyg Toxicol 244 (1938).

Bennett et al, Morphological Changes in the Livers of Rats Resulting from Exposure to Certain Chlorinated Hydrocarbons, 20 J Ind Hyg and Toxicol 97 (1938).

Greenberg et al., The Systemic Effects Resulting from Exposure to Certain Chlorinated Hydrocarbons, 21 J Ind Hyg Toxicol 29 (1939).

Schwartz, An Outbreak of Halowax Acne "Cable Rash" Among Electricians, 122 J Am Med Assoc 158 (1943).

Schwartz and Peck, Occupational Acne, 43 N Y State J Med 1711 (1943).

Good and Pensky, Halowax Acne (Cable Rash), 48 Arch Dermat & Syph 251 (1943).

Greenberg, Chlorinated naphthalenes and diphenyls, 12 Ind Med 520 (1943).

Cranch, Chlorinated Compounds - Precautions in Handling, 13 Ind Med 1000 (1944).

Miller, Pathologic Changes in Animals Exposed to a Commercial Chlorinated Diphenyl, 59 Public Health Reports 1085 (1944).

Miller, Pathological Changes in Animals Exposed to a Commercial Chlorinated Diphenyl, 59 Pub Health Reports 1085 (1944).

Meigs et al, Chloracne from an Unusual Exposure to Aroclor, 154 J Am Med Assoc 1417 (1953).

Treon, The Toxicity of the Vapors of Aroclor 1242 and Aroclor 1254, 17 Am Ind Hyg Assoc Quart 204 (1956).

Gafafer (ed), Chlorinated Diphenyls and Naphthalenes, in: Occupational Diseases -- A Guide to their Recognition, PHS Publication No. 1097, US Dept of HEW, Public Health Service, Pages 117 - 118 (1966).

SOME FOREIGN PCB MANUFACTURERS

<u>Producer</u>	<u>Trademark Nationality</u>	<u>Trademark</u>
Kanegafuchi Industries Co.	Japanese	Kanachlor
Caffaro* (Italy) Marketed by Apirollo after 1974	Italian	Fenclor
Bayer (Leverkusen)	West German	Clophen
Prodelec**	French	Phenoclor Pyrallene
Mitsubishi-Monsanto	Japanese	Aroclor Santotherm
Soval	U.S.S.R.	Soval
Chemko	Czechoslovakia	Delor

* Millions of pounds of Fenclor DK, a technical grade of decachlorobiphenyl, were imported into the United States to make wax molds for use in die casting; smaller amounts were imported for other purposes.

** Prodelec exported PCBs to the United States; specifically, to the Joy Manufacturing Co. (Pittsburgh, Pa.) for use as a motor coolant, and possibly to other U.S. companies.

REGULATION OF PCBS BY U.S.EPA

Introduction

Regulatory authorities paid little attention to PCBs prior to the late 1960's. Only then did the U.S. Food and Drug Administration (FDA) begin the regulatory process by setting internal "action levels." In 1970, FDA announced guidelines for fish and milk, and in 1972 they published proposed tolerances in the Federal Register. Nearly all FDA activity regarding PCBs during the past few years has focused upon fish and shell fish.

The Federal Insecticide, Fungicide, and Rodenticide Act cancelled the registration of all pesticides which contained PCBs either as an active ingredient, or as an extender to retard evaporation, effective November 29, 1970.

The U.S. EPA gathered information during the early 1970's, but did little to regulate PCBs before they were forced to administer the PCB phase-out mandated by Section 6(e) of the Toxic Substances Control Act of 1976 (TSCA). EPA currently regulates PCBs which are still in service in electrical equipment or which are distributed in commerce for special uses. They also regulate PCB discharges into the environment, and the disposal or destruction of discarded PCBs.

The U.S. Department of Labor has not become very involved in PCB regulation; in fact, the Occupational Health and Safety Administration (OSHA) still permits levels in workroom air which are 500 to 1000 times the maximum levels recommended in 1977 by the National Institute of Occupational Health and Safety.² Workers must rely upon state regulations, their unions, and their own diligence for protection from PCB exposure.

Public Law 94-469, found at 15 U.S.C.A. Sections 2601 et seq. 2. 29 CFR 1910.1000, Table G-1, contains the OSHA standard. The NIOSH recommendation is in: U.S. Department of Health Education and Welfare, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 77 - 225, "Criteria for a Recommended Standard, Occupational Exposure to Polychlorinated Biphenyls (PCB's).

The Interdepartmental Task Force

On September 1, 1971, representatives of several agencies of the Federal Government formed an interdepartmental task force to study PCBs. The major sponsoring agencies were the Environmental Protection Agency and the Departments of Agriculture; Health, Education, and Welfare; Interior; and Commerce. Industry representatives played a crucial role in helping the task force develop its recommendations, particularly those relating to the continued use of PCBs in electrical equipment.

On March 20, 1972, the task force issued the following findings, conclusions, and recommendations:¹

1. PCBs should be restricted to essential or non-replaceable uses which involve minimal direct human exposure since they can have adverse affects on human health.

2. PCBs have been used so widely over such a long period that they are ubiquitous.

3. PCBs were first identified as potential food contaminants in 1966. The three principal dietary sources are fresh water fish, food packaging material, and accidental PCB leaks into food or feed.

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 task force went on to state that: "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 task force recommended passage of the Toxic Substances Control Act.

5. Housekeeping is particularly important in the

manufacture, use, and disposal of PCBs.

6. The use of PCBs should not be banned entirely. The task force elaborated as follows: "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.

8. PCBs are manufactured in countries other than the United States.

9. More scientific information about PCBs is needed, and several Government agencies are seeking it through research.

1. Interdepartmental Task Force on PCBs, PCBs and the Environment, Department of Health Education and Welfare, March 1972.

The National Bureau of Standards Report

The report of the Task Force included a three page review of the benefits and utility of PCBs prepared by the National Bureau of Standards and based partly upon information supplied by the National Industrial Pollution Control Council and other unspecified associations. The National Bureau of Standards report stated that fluorocarbons are the other class of non-flammable fluids which are comparable to PCBs. They pointed out that the fluorocarbons are more volatile than PCBs. They concluded that "If codes did allow flammable materials in (buildings in which flammable fluids in capacitors are prohibited by insurance companies and building codes), 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."

The Bureau concluded that adequate substitutes for PCBs were available for use as hydraulic fluids, lubricants, heat transfer fluids, and "plasticizers". This report could be used to support the position that the electric power industry was not negligent in using PCBs, at least not after society had become accustomed to using equipment containing them. Should the report be entered into evidence, plaintiffs' attorneys are likely to allege that too much weight was given to information provided by Industry. They may also draw attention to technical inadequacies, such as the statements that fluorocarbons typically have low vapor pressures and that PCBs are good plasticizers for use with copy paper are erroneous. Fluorocarbons typically have high vapor pressures, and while PCBs make good dye carriers for copy paper, this application has nothing to do with plasticity.

4:04. PCB Regulation pursuant to TSCA

The Toxic Substances Control Act of 1976¹ requires the EPA to gather information about toxic substances from manufacturers, regulate chemical substances and mixtures which "present an unreasonable risk of injury to health or the environment", and to take action with respect to "imminent hazards". In addition to these general purposes, TSCA specifically requires that EPA take action regarding PCBs.² PCBs are the only chemicals which are specifically mentioned in the Act. Section 6(e) of TSCA bans the manufacture, processing, and use of all PCBs which are not "totally enclosed" after January 1, 1979, bans the distribution of PCBs in commerce after July 1, 1979, and requires proper disposal of PCBs. The EPA is authorized to make renewable one-year exceptions upon petition.³

1. 15 U.S.C.A. Sections 2601 et seq.
2. 15 U.S.C.A. Sections 2601 et seq.
3. 15 U.S.C. 2605(e)(2)

The PCB Ban Rule

Pursuant to the requirements of TSCA, EPA promulgated regulations in 1979 which prohibited the manufacture, import, processing, distribution in commerce, or export of PCBs unless an exemption was granted.² Eleven specific exemptions were granted for uses which did not seem to pose a significant threat, and where adequate substitutes were unavailable. These included the use of PCBs in servicing transformers, the use of small quantities for research, and as a microscopic mounting medium.

In the 1979 promulgation, EPA also exempted from regulation all material containing less than 50 ppm PCBs; all intact, nonleaking capacitors and electromagnets; and all intact, nonleaking transformers which were not being used on railroads.

The following section describes the Environmental Defense Fund's successful challenge to the 50 ppm cutoff, and to EPA's effort to exclude capacitors, transformers, and electromagnets from regulation.

1. 44 FR 31514 (May 31, 1979).

EDF V EPA

TSCA allows the EPA to authorize the use of PCBs in any way that it finds "will not present an unreasonable risk of injury to health or the environment." Section 6(e)(2) specifically provides that EPA may allow the continued use of PCBs in a "totally enclosed manner", that is, "any manner which will ensure that any exposure of human beings or the environment to a polychlorinated biphenyl will be insignificant, as determined by the Administrator by rule." Pursuant to these provisions in the statute, EPA included in its initial regulations, published on May 31, 1979 (44 FR 31514), the designation of all intact, non-leaking capacitors, electromagnets, and transformers other than railroad transformers as "totally enclosed." EPA also set 50 parts per million (ppm) as the minimal level of PCB concentration which would be regulated and granted exemptions to 11 petitioners.

The Environmental Defense Fund petitioned the U.S. Court of Appeals for the District of Columbia Circuit to review these decisions, ¹ and on October 30, 1980, the court ruled that there was insufficient evidence in the record to support the EPA's classification of transformers, capacitors, and electromagnets as "totally closed", and remanded the matter to EPA for further action. In its decision, the court pointed out that PCBs are specified in TSCA not only because they are dangerous, but because of EPA's history of less than vigorous action. The court commented: "Human beings have finally come to recognize that they must eliminate or control life threatening chemicals, such as PCBs, if the miracle of life is to continue and if earth is to remain a living planet" and that knowing the small amount of PCBs which had been eliminated from service since the passage of TSCA, "timid souls have good reason to question the prospects for our continued survival, and cynics have just cause to sneer at the effectiveness of governmental regulations." On January 21, 1981, EPA, EDF, and certain industry interveners in EDF V. EPA filed a joint motion asking the court to stay its mandate setting aside the classification of transformers, capacitors, and electromagnets as totally enclosed. EDF agreed with the other parties' contention that immediately taking all of the PCB containing equipment out of service would be impractical.

On February 12, 1981, the court issued an order staying its mandate regarding capacitors, transformers, and electromagnets pending further rulemaking.² On April 13,

1981, the court stayed its mandate with respect to activities involving PCBs in concentrations of less than 50 ppm pending further rulemaking.³ The court orders contained several provisions, including the following:

1) The EPA would publish an Advance Notice of Proposed Rulemaking, and follow up by developing a rule.

2) The Edison Electric Institute (EEI) and the Utility Solid Waste Activities Group (USWAG) would develop some of the factual material necessary for the rulemaking.

3) The parties would abide by a specified Interim Measures Program.

4) The EPA would promulgate a final rule within six months of receipt of the study from EEI and USWAG.

The Electrical Equipment Rule was published on August 25, 1982 (47 FR 37342), and became effective on September 28, 1982. These regulations were revised on July 17, 1985, to help minimize the danger of PCB, PCDF, and PCDD contamination resulting from transformer fires. The Closed and Controlled Waste Manufacturing Processes Rule was published on October 21, 1982 (47 FR 46980). That rule permits the manufacture, processing, and distribution in commerce of PCBs without an exemption, provided that (1) the PCBs are released only in concentrations below the practical limits of quantitation for PCBs in air emissions, water effluents, products, and process wastes and (2) the wastes from these manufacturing processes are controlled and disposed of in accordance with the methods for disposal specified in the rule. The Uncontrolled PCB Rule was published on July 10, 1984 (49 FR 28172). Uncontrolled PCBs are also referred to as "inadvertently generated" or "non-aroclor" PCBs. In the rulemaking, EPA estimated that 100,000 pounds of PCBs are inadvertently generated in the U.S. each year, and of these, 1000 pounds escape into the environment. They compare this to the 10,000,000 pounds which they estimate entered the environment annually before PCB controls were instituted, and the 150,000,000 pounds they estimated were currently in the environment as free PCBs. This rule introduced a "discounting factor" for monochlorinated and dichlorinated biphenyls whereby concentrations of these compounds are divided by 50 for regulatory purposes.

1. The Environmental Defense Fund, Inc. (Petitioner) v. Environmental Protection Agency (Respondent), 636 F.2d 1267 (1980). Interveners were: Ad Hoc Committee on Liquid Dielectrics of the Electronic Industries Association et al., Joy Manufacturing Company, Edison Electric Institute et al., and Aluminum Company of America. The Color Manufacturers Association filed an amicus curiae brief.

2. 46 FR 16090 (March 10, 1981). 3. 46 FR 27615 (May 20, 1981).

EPA RULEMAKING UNDER TSCA

February 17, 1978 (43 FR 7150) Disposal and Marking Rule.

August 2, 1978 (43 FR 33918) Technical amendments to the Disposal and Marking Rule.

November 1, 1978 (43 FR 50905) Interim Procedural Rules for PCB Manufacturing Exemptions.

January 2, 1979 (44 FR 108) All Petitioners for PCB manufacturing exemptions granted permission to continue their activities until further notice.

May 31, 1979 (FR 44 31524) Prohibited the manufacture (without specific exemption), sale, distribution in commerce, processing, and use (outside of "totally enclosed systems") of PCBs after July 2, 1979.

May 31, 1979 (44 FR 31558) Interim Procedural Rules for PCB Processing and Distribution in Commerce Exemptions.

May 31, 1979 (44 FR 41564) Proposed Rule for PCB Manufacturing Exemptions.

July 20, 1979 (44 FR 42727) Extended the public comment period regarding the proposal in 44 FR 41564.

March 5, 1980 (45 FR 14247) Clarification of points in 44 FR 108 May 1, 1980 (45 FR 29115) Reiterated the policy of not permitting PCBs to be imported or exported for disposal.

March 5, 1980 (45 FR 14247) Extended exemptions to petitioners whose activities were underway before January 1, 1979 (manufacturers) or July 1, 1979 (processors and distributors).

March 10, 1981 (46 FR 16090) Court Ordered Interim Measures Program

May 20, 1981 (46 FR 27615) The Court Order staying its mandate regarding PCBs at concentrations less than 50 ppm.

May 20, 1981 (46 FR 17617) ANPR on the 50 ppm limit for closed and controlled waste manufacturing processes.

May 20, 1981 (46 FR 17619) ANPR announcing rulemaking activities on the 50 ppm limit for uncontrolled PCBs.

May 6, 1982 (47 FR 19527) Recodification of the May 31, 1979 PCB ban.

August 25, 1982 (47 FR 37342) Electrical Equipment Rule

October 21, 1982 (47 FR 46980) Closed and Controlled Waste Manufacturing Processes Rule

November 1, 1983 (48 FR 50486) Proposed PCB Exemptions Rule

July 10, 1984 (49 FR 28154) Response to Individual Class Petitions

July 10, 1984 (49 FR 28172) Inadvertently generated PCB final rule.

July 10, 1984 (49 28193) Permanent exemptions promulgated for microscopy and research and development.

48 FR 55079 EPA explains why it adopted the approach to PCB regulations involving the concept of "unreasonable risk" recommended by CMA, EDF, and NRDC.

48 FR 55079 The concept of "unreasonable risk" is further explained December 8, 1983 proposed rulemaking for inadvertently generated PCBs.

July 10, 1984 (49 FR 28154) Individual and Class Exemptions

July 17, 1985 (50 FR 29171) Transformer Regulations

1. 15 U.S.C.A. Section 2601 et seq. 2. Federal Register of May 31, 1979 (44 FR 31514). 3. Environmental Defense Fund, Inc v. Environmental Protection Agency, 636 F. 2d 1267.

Highlights of the Electrical Equipment Regulations

Following the Environmental Defense Fund's 1980 victory, EPA published interim regulations regarding the inspection and maintenance of transformers.¹ On August 25, 1982, EPA promulgated final rules (they have since been changed) concerning PCBs.² The 1982 rules banned the use of PCB Transformers (electrical transformers containing more than 500 ppm PCBs) in facilities involved in the handling of food or feed effective October 1, 1985, and allowed the use of all other non-railroad electrical transformers for the remainder of their useful lives.

New rules were promulgated on July 17, 1985, after EPA recognized the danger posed by PCDFs and other products of incomplete PCB combustion. Highlights of these rules are as follows:

- 1) Prohibition of the continued use of some types of high voltage PCB transformers near commercial buildings beyond October 1, 1985.
- 2) Prohibition of future installation of some types of high voltage PCB Transformers near commercial buildings.
- 3) Installation by 1990 of protective equipment on certain high voltage PCB transformers located in or near commercial buildings.
- 4) Required registration of all PCB Transformers with fire departments and building owners by December 1, 1985.
- 5) Required marking of all PCB Transformer locations (except grates and manholes) by December 1, 1985.
- 6) Required removal of combustible material from the vicinity of PCB Transformers by December 1, 1985.
- 7) Required precautions in the event of a PCB Transformer fire. 8) Required notification of the National Response Center in the event of a PCB Transformer fire.

Responsibility for compliance with TSCA regulations rests primarily with owners of transformers. Users of transformers posing a risk to food are responsible for required inspection, maintenance, and record keeping until they notify the owner of the risk. EPA holds users at least partly responsible when the user has agreed to perform the owner's duty, the user has traditionally taken

responsibility, the owner's access to the equipment has been limited, or there is an emergency."

1. Federal Register on March 10, 1981 (46 FR 16090).
2. 47 FR 37342 codified in 40 CFR Part 761.
3. TSCA Compliance Program Policy No. 6-PCB-1, available from EPA.

Exemptions Granted by EPA

Section 6(e)(3)(B) of TSCA provides that any person may petition the Administrator of the EPA for an exemption from the prohibition against the manufacture, processing, and distribution in commerce of PCBs. The Administrator is authorized to grant exemptions of no more than one year in duration if he finds that no unreasonable risk would result and that an effort has been made to find a safe substitute for PCBs.

EPA has published separate rules for granting exemptions involving manufacturing¹ and processing and distribution in commerce.² These interim procedural rules describe the required content of exemption petitions, and the procedures EPA follows in ruling upon them.

On July 10, 1984, EPA published its final rule regarding 109 petitions for exemptions to TSCA's prohibition against the manufacturing, processing, distribution in and commerce and use of PCBs.³ Also in that issue of the Federal Register, EPA published a proposed ruling on the remaining 50 petitions for exemptions which it had received up to that time.

1. 43 FR 50905 (November 1, 1978). 2. 44 FR 31558 (May 31, 1979). 3. 15 U.S.C. 2605(e)(3)(B)

Requirements for PCB Disposal Under TSCA

When PCBs and PCB items are removed from service, they must be disposed of in accordance with regulations promulgated by the U.S. EPA pursuant to TSCA. PCBs, PCB items, and PCB contaminated dirt and debris which are in disposal sites are considered "in service" for the purposes of the regulations. PCBs which were landfilled prior to February 17, 1978, are not regulated under the TSCA regulations unless they are removed from the site.

PCBs which have been removed from service may be stored for no more than one year prior to disposal.¹ This requirement is not literally interpreted when PCBs are placed into bulk storage tanks. In those cases, levels in the tanks are not permitted to rise during any 12 month period.²

EPA has established detailed requirements concerning the disposal of various types of materials.³ Diluting contaminated material does not affect its regulatory category. Material containing over 500 ppm PCBs may not be landfilled; it must be disposed of in a way which EPA deems to be at least as efficient as incineration.⁴

EPA has also set performance standards for incinerators which burn PCBs.⁵ These standards have been criticized because they do not deal directly with what may be the greatest threat from PCB combustion, the formation of PCDFs. The standards do, however, offer at least some degree of protection from PCDFs and other products of incomplete combustion by requiring that certain minimal operating conditions be maintained.

1. 40 CFR Section 761.65. 2. TSCA Compliance Program Policy No. 6-PCB-10, August 13, 1985. 3. 40 CFR 761.60. 4. 40 CFR 761.60(a)(1) requires incineration, 40 CFR 761.60(e) provides for alternatives which are at least as efficient. 5. 40 CFR 761.70.

Preemption of Local Disposal Restrictions

The U.S. EPA allows state and local governments to impose regulatory standards which are more stringent than federal standards, particularly when state and local standards do not conflict with federal regulations. EPA's policy has been to encourage the states to take responsibility for environmental protection, which is usually possible when state regulatory standards are not lower than the U.S. EPA's.

Under the Commerce Clause of the Constitution and the preemption clause of TSCA, the United States has broad authority to preempt local bans on PCB disposal. In one important decision¹, the Fifth Circuit Court of Appeals ruled that state and local governments must obtain an exemption under Section 18(b) of TSCA before implementing restrictions on the disposal of PCBs. Local authorities will have greater success in restricting PCB disposal if they do not specifically address PCBs, cite specific local conditions which make disposal inappropriate, and are prepared to show that their regulations do not unduly restrict interstate commerce. The Fifth circuit ruling attempts to place the burden of proving the validity of local restrictions of PCB disposal upon local government, with EPA, not a federal district court, as the initial judge of reasonableness.²

1. Rollins Environmental Services (FS), Inc. v. Parish of St. James, 15 ELR 21044.
2. Lewis, National Interest

PCB Spills

While specific requirements for cleaning up PCB spills have not been established under TSCA, the U.S. EPA Office of Toxic Substances does maintain authority pursuant to TSCA by classifying spills as improper disposal under 40 CFR 761.30(a)(1)(iii) and 40 CFR 761.60(d). In most cases, such as capacitor explosions or transformer leaks, EPA Regional Offices merely maintain telephone contact with the responsible party. Inspectors may be dispatched in case of large spills, or where it is apparent that remedial action or post-cleanup activities are inadequate.

Persons in charge of vessels or facilities from which over 10 pounds of PCBs spill within a 24 hour period must report the incident immediately upon discovery to the National Response Center in Washington D.C. (800 - 424-8802).¹ This satisfies the reporting requirements of all applicable programs administered by the U.S. EPA and the U.S. Department of Transportation. The reportable quantity would have been 1 pound under Section 102(b) of CERCLA,² but pending reevaluation,³ that level is superseded by the 10 pound reportable quantity set by EPA pursuant to Section 311(b)(4) of the Clean Water Act.⁴ Newspaper notification of potentially injured parties is required by Section 111(g) of CERCLA.

1. 50 FR 13456 (April 4, 1985)

2. The Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (Pub. L. 96-510), 42 U.S.C. 9601 et seq.

3. EPA proposed lowering the limit to 1 pound in 48 FR 23552 (May 25, 1983), but has not made a final ruling.

4. Federal Water Pollution Control Act as amended in 1976, 33 U.S.C.A. et seq.

EPA Regulations Pursuant to RCRA And CERCLA

The Resource Conservation and Recovery Act of 1976 (as amended in 1984) gives the EPA the authority to regulate waste disposal facilities, and to initiate judicial action where waste disposal presents an "imminent hazard". Because Section 6(e) of the Toxic Substances Control Act specifically requires the regulation of PCBs, they were not listed as hazardous substances in the regulations which were promulgated pursuant to the enactment of RCRA.¹ Because of this, wastes which contain no hazardous constituent other than PCBs are not regulated under RCRA. The RCRA regulations are likely to be changed to include pure PCBs in the near future. They already include them as constituents of waste and subject them to special handling, reporting and record-keeping requirements.² The Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA)³, commonly known as the "Superfund" act, does not specifically mention PCBs, but it contains provisions which are relevant to environmental contamination by PCBs. Section 103(b) provides for penalties of up to \$10,000 in fines and one year in prison for failure to notify the Emergency Response Center of a spill involving the release of more than 10 pounds of PCBs within a period of 24 hours. The reportable amount would have been 1 pound under CERCLA alone, but the 10 pound limit set pursuant to Section 311(b)(4) of the Clean Water Act takes precedence. EPA is considering lowering the reportable quantity to 1 pound.

Section 103(c) provides for fines and imprisonment of up to \$10,000 and 1 year for failure to report stored or discarded PCBs within 180 days of December 11, 1980.

Section 103(d) provides for fines and imprisonment up to \$20,000 and one year for knowingly destroying, mutilating, erasing, disposing of, concealing, or otherwise rendering unavailable or unreadable or falsify any records concerning stored or discarded PCBs.

Sections 106 et seq. give the president of the United States the authority to require the Attorney General of the United States to protect the public from any actual or threatened release of hazardous substances, including PCBs. This authority allows the federal government to clean up PCB contaminated sites, and force the liable parties to pay punitive damages of three times the government's expense. This has the effect of encouraging potentially liable parties to cooperate in clean-up efforts.

EPA Regulations Pursuant to RCRA And CERCLA

The Resource Conservation and Recovery Act of 1976 (as amended in 1984) gives the EPA the authority to regulate waste disposal facilities, and to initiate judicial action where waste disposal presents an "imminent hazard". Because Section 6(e) of the Toxic Substances Control Act specifically requires the regulation of PCBs, they were not listed as hazardous substances in the regulations which were promulgated pursuant to the enactment of RCRA.¹ Because of this, wastes which contain no hazardous constituent other than PCBs are not regulated under RCRA. The RCRA regulations are likely to be changed to include pure PCBs in the near future. They already include them as constituents of waste and subject them to special handling, reporting and record-keeping requirements.² The Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA)³, commonly known as the "Superfund" act, does not specifically mention PCBs, but it contains provisions which are relevant to environmental contamination by PCBs. Section 103(b) provides for penalties of up to \$10,000 in fines and one year in prison for failure to notify the Emergency Response Center of a spill involving the release of more than 10 pounds of PCBs within a period of 24 hours. The reportable amount would have been 1 pound under CERCLA alone, but the 10 pound limit set pursuant to Section 311(b)(4) of the Clean Water Act takes precedence. EPA is considering lowering the reportable quantity to 1 pound.

Section 103(c) provides for fines and imprisonment of up to \$10,000 and 1 year for failure to report stored or discarded PCBs within 180 days of December 11, 1980.

Section 103(d) provides for fines and imprisonment up to \$20,000 and one year for knowingly destroying, mutilating, erasing, disposing of, concealing, or otherwise rendering unavailable or unreadable or falsify any records concerning stored or discarded PCBs.

Sections 106 et seq. give the president of the United States the authority to require the Attorney General of the United States to protect the public from any actual or threatened release of hazardous substances, including PCBs. This authority allows the federal government to clean up PCB contaminated sites, and force the liable parties to pay punitive damages of three times the government's expense. This has the effect of encouraging potentially liable parties to cooperate in clean-up efforts.

1. The original proposal (43 FR 243, p.58946 [12/18/78]) did specify PCBs.
2. 40 CFR 261.11, promulgated 5/19/80.
3. The Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (Pub. L. 96-510), 42 U.S.C. 9601 et seq.

Judicial Action Pursuant to RCRA and CERCLA

A judicial action involves or is intended to lead to the filing of papers in court. The action begins only after EPA has filed a written proposal with the U.S. Department of Justice (DOJ), preferably addressed to the Assistant Attorney General of the Land and Natural Resources Division. Activities such as the issuance of notices, complaints, requests, or orders under various environmental statutes which do not involve the courts are called administrative actions. Administrative actions can be very effective. For example, violation of a presidential order¹ under Section 106 of CERCLA can result in court imposed fines of up to \$5,000 per day.

Judicial action is usually initiated by EPA Regional Administrators in consultation with EPA Regional Counsels. Regional Administrators are responsible for identifying parties against whom judicial action might be appropriate, and for referring cases to the DOJ, accompanied by a concurrence from the Regional Counsel. The Regional Administrator must first obtain approval from the Assistant Administrator for the EPA Office of Enforcement and Compliance Monitoring. Relief sought in this manner can include:

- declaratory judgements on points of law.
- money judgements for the recovery of civil penalties, out of pocket expense, or damages.
- injunctive or similar specific relief such as an order to perform certain acts.
- interem relief; for example, seizing or condemning land, vessels, or goods.

In criminal matters, cases are referred to the DOJ by the Regional Counsel or the Special Agent In Charge. The EPA's Criminal Enforcement Division is independent from Regional control. Criminal convictions under many environmental statutes can carry penalties of imprisonment, probation, and fines.

Pleadings, motions, and briefs are prepared by the DOJ in consultation with EPA. Technical allegations in complaints are prepared by EPA. Lead trial attorneys are provided by the DOJ. Inquiries concerning particular cases are referred to the lead trial attorney who designates

contact people for opposing parties, the press, and other parties outside EPA. The lead trial attorney speaks for the government in negotiations, although settlements must be approved by the Regional Administrator, the DOJ, and EPA's Office of Enforcement and Compliance Monitoring.

1. The President's function were transferred to the Administrator of EPA in 46 FR 42237. Specific Presidential authority was held unnecessary in U.S. v. Reilly Tar, 546 F. Supp. 1100 (D. Minn. 1982).

Technical Documents

Numerous technical documents may be drafted by the United States and its consultants during a judicial action. The two most important such documents are the endangerment assessment and the feasibility study for enforcement.

The endangerment assessment evaluates the nature and extent of contamination at a site, along with the potential for harm to humans and the environment. It is a prerequisite for administrative orders under Section 106 of CERCLA and Sections 3013 and 7003 of RCRA, and it provides the "imminent and substantial endangerment" count of the complaint in a judicial action. At Superfund sites, an endangerment assessment is part of the remedial investigation/feasibility study.

The feasibility study for enforcement determines the relief that will be sought from defendants at trial. At Superfund sites, it also assures compliance with Section 300.68 (c) of the National Contingency Plan, which requires that enforcement sought remedies consider the same factors as Fund financed remedies.

The Bloomington, Indiana CERCLA Action

The Westinghouse Corporation has operated an electrical equipment manufacturing plant in Bloomington, Indiana since 1957. The major product is large, power grid capacitors. PCBs were used to fill these capacitors prior to 1979.

Large numbers of failed capacitors found their way to various dumps and abandoned stone quarries in and near Bloomington. Some of these capacitors were assembly line rejects; some reportedly had failed in service and were sent to Bloomington for dumping.

A Bloomington sewer line and sewage treatment plant were also heavily contaminated with PCBs. Contaminated sludge from the sewage treatment plant was used by nearby farmers and gardeners as fertilizer. The creek into which treated sewage was discharged became contaminated with PCBs.

The city of Bloomington waited until 1981 to file suit against Westinghouse to remedy PCB contamination of the sewer and the sewage treatment plant. Later that year, they amended their complaint to include their abandoned dump, the Lemon Lane Landfill, and to include Monsanto as a defendant. The city filed in federal court based on diversity of citizenship, but relied solely on Indiana common law theories of nuisance, trespass, strict liability, and negligence in their complaint.

In January, 1983, the United States filed a separate action against Westinghouse to force the cleanup of two PCB dump sites not by Bloomington and to recover their costs. The United States based their action on section 7003 of RCRA, and sections 106 and 107 of CERCLA, asserting that the sites posed an imminent and substantial endangerment to the public health and the environment. The State of Indiana intervened as a plaintiff and Westinghouse joined Monsanto as a third party defendant.

The two suits were consolidated, and the court entered a consent decree approving an out of court settlement on August 22, 1985. EPA officials promoted the settlement as a model of cooperation between industry and government, and called it their biggest Superfund victory.

In Bloomington, the settlement is a matter of bitter controversy. In March of 1986, the Bloomington city

chemist and his predecessor jointly filed a motion for leave to intervene in the consolidated lawsuits in order to pursue their allegations of collusion among the parties, and fraud by several principal attorneys. The Indiana Public Interest Research Group (InPIRG) filed a brief in August, 1986, asking the court to vacate its approval of the consent decree and its decision barring their intervention. InPIRG alleged that the parties fraudulently misled the court regarding key issues, including the safety of the proposal to incinerate PCB contaminated material in a refuse fired incinerator.

The former and current city chemists allege in their motion and supporting briefs that the proposed incinerator would be unsafe, and that setting the precedent of legally burning hazardous waste in a municipal trash incinerators would be ill-advised. They also allege that the parties concealed from the court the nature and extent of the contamination problem in Bloomington, and that EPA and Westinghouse entered into a "sweetheart" arrangement whereby Westinghouse would use the Bloomington location to launch a lucrative incinerator business and EPA would claim an environmental victory.

1. Public Law 96-510 (11 December 1980), 42 U.S.C. Section 9601 et seq.
2. Federal Water Pollution Control Act as amended in 1976, 33 U.S.C.A. et seq.
3. 50 FR 13471
4. EPA, Westinghouse Agree on Waste Cleanups, Chem Eng News, May 27, 1985, page 7.
5. Trewhitt, EPA's Biggest Waste Win Ever, Chemical Week, May 29, 1985, page 13.
6. Collins and Zordan, PCBs Destroyed by Burning with Municipal Waste, Modern Power Systems, August, 1985m page 50.
7. Rogers, Westinghouse Cleans Up, Fortune, June 24, 1985, page 9.

FIFRA Regulations

The Federal Insecticide, Fungicide, and Rodenticide Act¹ is currently administered by the U.S. EPA; but it was in 1970, while the Act was being administered by the Agriculture Department that PCBs were banned from all pesticide formulations.²

1. 7 U.S.C. 135-135k 2. PR Notice 70 - 25, issued on October 29, 1970, gave pesticide formulators six months to remove PCBs from their products.

HALOWAX

During the early years of PCB use, between 1930 and 1950, PCBs were often mixed with compounds called chlorinated naphthalenes.

"Halowax" was a brand name for chlorinated naphthalenes and mixtures of chlorinated naphthalenes (CNPs) and PCBs. CNPs are relevant to this discussion for numerous reasons, including: (1) they have been identified as PCB contaminants; (2) they have been identified as PCB combustion byproducts; (3) They were used in formulations containing PCBs; and (4) they are so similar in structure to PCBs that industry had reason to suspect PCBs were harmful when CNPs were shown to be. Indeed, PCNs seem to act on the body much in the same way PCBs do. There is also evidence that exposure to PCBs increase the toxicity of PCNs. It seems ironic that although CNPs are generally thought to be more toxic than PCBs, they are still being manufactured and used.¹ Since the properties, uses, and effects of CNPs parallel those of PCBs, learning about either necessarily increases one's understanding of the other.

Literature dating back to the turn of the century reports chloracne and liver disease among workers exposed to CNPs.² According to Louis Schwartz, unreported cases of CNP induced chloracne occurred in the United States as early as 1912.³ Many of these workers had been exposed to PCBs also, since both types of mixture were used primarily as fill for capacitors and transformers; as insulators on electric wires; and in paints, varnishes and lacquers. Popular commercial forms of CNPs known as "Halowax", manufactured by a subsidiary of Union Carbide, contained up to 20% PCBs. Koppers has been America's major producer of PCNs for many years.

In 1943, an article titled "Halowax Acne (Cable Rash)" described pervasive chloracne, complaints of burning and sore eyes, lassitude, anorexia, nausea, headaches, vague abdominal pains, impotence, weight loss, insomnia, alopecia and disturbance of taste among marine electricians. They had been installing Halowax coated cable in poorly ventilated crawl spaces.⁴ Other articles concerning the toxicity of chlorinated hydrocarbons appeared around this time.⁵

1. Kimbrough, Occupational Exposure, page 373 of Kimbrough (ed), Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products, (1980)
2. Kimbrough, Occupational Exposure, page 373 of Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products, (1980) citing: Herxheimer, Uber Chloracne, 46 Munch Med Wochenschr 278, (1899); and Bettmann, Chloracne, Eine Besondere Form von Professioneller Hauterkrankung, 27 Dtsch Med Wochenschr 437 (1901).
3. Schwartz, Skin Hazards in American industry, U.S. Public Health Bulletin No. 229, part II (1936).
4. Good and Pensky, Halowax Acne (cable rash), 48 Arch Dermat & Syph 251 (1943).
5. Flinn and Jarvik, Liver Lesions Caused by Chlorinated Naphthalene, 27 Am J Hyg 19 (1938); citing Wauer, Gewerbliche Erkrangingen Durch Gechlorte Kohlenwasserstoffe (Pernakrankheit), 6 Zentralblatt f. Gewerberg 100, 1918. Also see the bibliography in Section 3:05.

THE YUSHO CATASTROPHE

Although the toxicity of PCBs had long been known within industries involved in their manufacture and use, it took the large outbreak of disease which came to be known as "Yusho" - Japanese for "rice oil disease" - to bring the world's attention to the dangers of PCBs.

Cases of chloracne began to surface in northern Japan in the summer of 1968. By January, 1969, Kyushu University Hospital had seen at least 138 cases of what came to be known as "Yusho", Japanese for "oil disease". It later became apparent that about 1800 people had been poisoned by PCBs which had leaked from heat exchange equipment into cooking oil which was being processed for market. The batches of contaminated oil contained 2000 ppm Kenechlor 400, a Japanese brand of PCBs.

Many years after the poisoning episode, it became apparent that PCDFs and chlorinated quaterphenyls (PCQs) had formed in the Kenechlor 400, presumably due to the combination of heat and air in the heat exchange equipment. It is now widely accepted that Yusho was caused by the combination of PCBs, PCQs and PCDFs or else primarily by PCDFs. In examining the history of PCBs, it is significant that for years Yusho was thought to be nothing but PCB poisoning. The revelation that other compounds were present in the Yusho rice oil does not diminish the significance of the tragedy in a discussion of PCBs because PCDFs, the most toxic component of the contaminated oil, are universal PCB contaminants. Levels of PCDFs in PCBs vary depending on the extent to which the PCBs have been exposed to heat and air. PCDFs are the primary cause of concern after PCB fires.

Press reports of the suffering of the Yusho victims sparked world-wide concern about PCBs. Early signs of poisoning included weakening of eyesight, fatigue, nausea and vomiting. Disfiguring chloracne, swollen limbs, enlargement of follicular openings, and pigmentation of the gingivae, lips, knuckles, and nails followed. Victims suffered from swelling, pigmentation, and cheese-like discharges from their eyes. Infected acneform lesions became abscesses. Sputum producing coughs were a symptom. Lumbago, joint pains, and heel pains were common. Soles and palms became hardened and discolored. Numbness, anorexia, and impotence were reported. Menstrual cycles were disrupted and discolored and sickly babies were born. Benign tumors were observed in some victims' joints.

REVIEWS

Kuratsune and Shapiro (eds), PCB Poisoning in Japan and Taiwan (1984).

Higuchi, PCB Poisoning and Pollution, Academic Press (1976).

Autopsy Reports

For most victims, Yusho symptoms gradually diminished, although in 1984 it was reported that some Yusho victims were still severely ill.¹ A few people (including one stillborn infant) may have died from acute PCB poisoning. As of 1982, 12 victims were known to have died, but the possible role of PCBs in these deaths could not be definitely ascertained.

Autopsies of the 12 victims revealed that two people had developed pulmonary carcinomas, two had developed liver cell carcinomas, and one developed an esophageal carcinoma. The autopsies also revealed PCB accumulations, lesions, and degeneration around the hearts of the victims. It has been postulated that the heart conditions resulted from disturbances of the endocrine system.²

Esophageal and bronchial abnormalities were also observed in the first autopsied Yusho victims. These findings are thought to be at least partly attributable to ingestion of the contaminated cooking oil.²

Changes in the fine structure - the structure seen through an electron microscope as opposed to the larger view seen through conventional optical microscopes - of the liver are characteristic of exposure to PCBs and related compounds. The changes - most notably a proliferation of the smooth endoplasmic reticulum - can be observed in biopsy specimens from living patients.²

1. Kikuchi, Autopsy of Patients with Yusho, 5 Amer J Ind Med 19 (1984), reprinted in Kuratsune and Shapiro (eds), PCB Poisoning in Japan and Taiwan (1984).

2. Kikuchi, Autopsy of Patients with Yusho, 5 Am J Ind Med 19 (1984), reprinted in Kuratsune and Shapiro (eds), PCB Poisoning in Japan and Taiwan (1984).

MORTALITY STATISTICS

Statistics published in 1984¹ show above average death rates for various causes. The most statistically significant results include total liver cancer among men, liver cancer among men over 40 years of age who died after 1976, ischemic heart disease among women who died after 1976, and cancer of the esophagus among men in Fukuoka prefecture who died after 1976.

The results associating Yusho with liver cancer are consistent with experimental data and some epidemiological studies involving occupational exposure.² The evidence that PCBs cause liver cancer in humans is becoming increasingly compelling with the passage of time.

1. Amano et al., Statistical Observations about the Causes of the Death of Patients with Oil Poisoning, 39 Japan Hygiene 1 (1984); translated for the U.S. EPA by SCITRAN.
2. Brown, Mortality of Workers Exposed to Polychlorinated Biphenyls - an Update, NIOSH, Cincinnati (1986); also: Zach and Musch, A Retrospective Mortality Study of Polychlorinated Biphenyl Manufacturing Workers, Monsanto Company (1983).

Update - much new info
Schell

TOXICOLOGICAL OVERVIEW

Poisons induce illness by first interacting with natural chemicals inside the body. These interactions set off a complex chain of reactions which culminate in a change in the body's chemical balance, either in the bloodstream or in specific organs or types of cells. Overt effects of poisons and drugs are a consequence of these often little understood chemical events.

Numerous overt adverse health consequences of PCB exposure have been identified. Interactions between PCBs and natural molecules inside the body have also been identified. In addition to this information at the two extremes - the manifestation of symptoms and the underlying chemical basis for disease - there is also information about intermediate biochemical abnormalities and imbalances which can be induced by exposure to PCBs. Some of these abnormalities and imbalances are part of the chains of events linking molecular interactions between PCBs and the body's chemicals with the symptoms of disease.

As experimental scientists continue to generate information, medical practitioners will be increasingly able to link the biochemical changes induced by PCBs with specific disorders and diseases. This increased knowledge could help in the treatment of people whose maladies are induced or exacerbated by exposure to PCBs and related compounds; it could also provide the basis for expert testimony in toxic tort lawsuits.

Chlorinated Aromatic Hydrocarbons

PCBs are a group of 209 chemical compounds. They are a sub-group of a larger group of compounds called "chlorinated aromatic hydrocarbons," a group which includes notoriously potent toxins such as the dioxins and the chlorinated dibenzofurans (also called "furans" or "PCDFs"). The dioxins and furans are members of a larger group of compounds called "chlorinated polycyclic aromatic hydrocarbons" or "chlorinated polynuclear aromatic hydrocarbons." PCBs can be included among the polycyclic aromatic hydrocarbons, even though that designation is usually reserved for compounds whose carbon framework is locked in a planar configuration.

Dioxins, furans, and PCBs are the three groups of chlorinated polycyclic aromatic hydrocarbons which have received the most scientific and regulatory attention. These compounds are frequently treated simultaneously because they share so many chemical, environmental, and toxicological properties. In fact, some PCB congeners have much more in common with some of the furans and dioxins than they do with many of the other PCBs.

The biochemical effects of the most potent dioxins, furans, and PCBs are receiving a great deal of attention from the scientific community. Proteins to which these compounds bind are being isolated and identified, and it is evident that many of the dioxins, furans, and PCBs interact with molecules fundamentally involved with the endocrine (hormone) and immune systems. Future research will elucidate how people vary in their reactions to PCB exposure, and how PCB induced imbalances in the body's internal chemistry are linked to illness.

Partial List of Reported Adverse Health Effects

There is much controversy and wide-ranging opinions concerning the adverse health effects of exposure to PCBs. The following is a partial list of diseases, conditions, and symptoms which have been linked to PCB exposure:

- harm to the immune system
- harm to the endocrine system
- heart disease
- lung disease
- gastro-intestinal disease
- liver disease
- cancer
- eye disease
- skin disease
- diseases of the joints
- damage to the nervous system
- pathological changes in sweat glands
- profuse sweating
- sexual dysfunction
- chronic headaches
- disorientation
- dizzy spells
- irritability
- phobias
- tingling and numbness in the extremities
- increased sensitivity to many chemicals

PCB Contaminants and Byproducts

Due to the non-specific manufacturing process, which produced a variety of PCB congeners in each batch, PCBs are nearly always encountered in the form of complex mixtures. Traces of highly toxic PCB congeners in the mixtures, and the trace contaminants which form during manufacturing and use, together may account for a significant portion of their toxicity.

PCBs are like "Agent Orange" in that toxicity varies from specimen to specimen, and may depend on the quantities of the trace toxins present. In the case of "Agent Orange", the most significant contaminants are thought to be traces of dioxins. In the case of PCBs, the contaminants include dioxin-like compounds such as furans and biphenylenes, and after transformer fires, dioxins themselves. In a sense, PCB contaminants can be said to include some of the PCBs themselves.

It is odd, of course, to view PCBs as PCB contaminants. It is just one way of expressing the fact that toxicities vary widely among the 209 PCB congeners, and that the most devastatingly acute poisons among them tend to be only minor constituents of commercial and environmental mixtures. A detailed review dealing with research into the properties of some of the most toxic PCB congeners has been published.¹ Some researchers are now analyzing oils, human adipose tissue, and environmental specimens to determine the content of only three PCB congeners: 3,3',4,4'-tetrachlorobiphenyl, 3,3',4,4',5-pentachloro-biphenyl, and 3,3',4,4',5,5'-hexachlorobiphenyl.² These three compounds are among the most toxic and environmentally persistent PCBs.

The PCB contaminants which have received the most attention are the chlorinated dibenzofurans, often called "polychlorinated" dibenzofurans and abbreviated PCDFs. Like PCBs, PCDFs do not rapidly degrade in the environment, and hence contaminate the entire surface of planet Earth. They were constituents of commercial PCB mixtures,³ and are, therefore, probably present at higher than background levels wherever PCB contamination is found (see table I). The combination of heat and oxygen transforms PCBs into PCDFs. As is described in another report, a single oxygen atom changes a PCB molecule into a dibenzofuran molecule.⁴

INSERT TABLE - MILBY

Personnel responsible for emergency response and testing in incidents involving PCB combustion should be acutely aware of the potential threat from PCDFs, chlorinated biphenylenes, dioxins, etc.⁵ Significant dioxin contamination results when PCBs mixed with chlorinated benzenes are burned. Many PCB containing transformers were filled with this combination, which is often referred to as "askarels". (Some use "askarel" as a generic term referring to any commercial PCB mixture.)

Chlorinated naphthalenes (polychlorinated naphthalenes, PCNs) are another group of extremely toxic compounds which have been identified in commercial PCBs⁶ and in residues remaining after PCB fires. PCNs preceded PCBs in the electrical equipment industry where PCNs, often mixed with lesser amounts of PCBs, were used to fill transformers and capacitors, and to coat electrical cable. These commercial PCN mixtures were called "halowax" and were manufactured by the Halowax Corporation and later by Coppers.

PCNs became decreasingly popular as it became clear that they were responsible for occupational disease and a number of deaths due to acute yellow atrophy of the liver. Salvage yard workers should be aware that in addition to PCBs and PCB contaminants, some older equipment might contain primarily PCNs.

While it is difficult to ascertain everything that has been mixed with PCBs,⁷ it is known that intentionally added capacitor and transformer fluid preservatives include potent carcinogens such as 1-ethylenoxy-3,4-epoxycyclohexane.⁸ When PCBs are burned, potentially significant byproducts include chlorinated phenols, biphenylenes, pyrenes, and chrysenes.

1. Safe, Polychlorinated Biphenyls (PCBs) and Polybrominated Biphenyls (PBBs), 13(4) CRC Crit Rev Toxicol 319 (1985).

2. Tanabe et al., Potentially Significant Residues of Highly Toxic Non-ortho Substituted PCBs in Human Adipose Tissues and in Terrestrial and Marine Animals, Paper given at Dioxin '86, the 6th International Symposium on Chlorinated Dioxins and Related Compounds, held in Fukuoka, Japan, between September 16th and 19th.

3. Perhaps due to an extra purification step, Aroclor 1016

may have been relatively free of PCDF contamination. For further information, see: (a) Miyata and Kashimoto, The Finding of Polychlorodibenzofurans in Commercial PCBs (Aroclor, Phenclor and Clophen), 17 Food Hyg Soc Japan 443 (1976) and (b) Bowes et al, Identification of Chlorinated Dibenzofurans in American Polychlorinated Biphenyls, 256 Nature (London) 305 (1975).

4. (a) The Yusho (contaminated rice oil) tragedy which occurred in Japan in 1976 involved PCBs which leaked from heat exchange equipment. Elevated PCDF levels were found in the heat exchange fluid, the rice oil, and in the victims, and it has been widely conjectured that they are primarily responsible for the symptoms which appeared; also see: (b) Lee, Assessment of PCDFs and PCDFs from PCB Transformer and Capacitor Fires, EPA/600/2-85/036(1985); NTIS Order # PB 85-188 837/AS (summarized in EPA document EPA/600/S2-85/036) and "Polychlorinated Biphenyls in Electrical Transformers; Final Rule.", Fed Reg, July 17, 1985 (40 CFR Part 761) for a discussion of several recent fires.

5. Schecter and Tiernan, Occupational Exposure to Polychlorinated Di8oxins, Polychlorinated Furans, Polychlorinated Biphenyls, and Biphenylenes After an Electrical Panel and Transformer Accident in an Office Building in Binghamton, NY, 60 Environ Health Persp 305 (1985).

6. Bowes et al, Identification of Chlorinated Dibenzofurans in American Polychlorinated Biphenyls, 256 Nature (London) 305 (1975).

7. Lee, Assessment of PCDFs and PCDFs from PCB Transformer and Capacitor Fires, EPA/600/2-85/036(1985); NTIS Order # PB 85-188 837/AS (summarized in EPA document EPA/600/S2-85/036) and "Polychlorinated Biphenyls in Electrical Transformers; Final Rule.", Fed Reg, July 17, 1985 (40 CFR Part 761) states: "In the last 60 years of commercial use, the formulation of askarels has undergone various changes and few manufacturers have made or kept records of the type and quality of PCBs and other chemical substances used in the formulating process."

8. Lawrence, Letter, 269(2) New Eng J Med 108 (1977), citing: Van Duren et al, Carcinogenicity of Epoxides, Lactones and Peroxy Compounds, 3 J Natl Cancer Inst 41 (1963).

Complexity of PCB Toxicity

Assessing the toxicity of a given PCB specimen is of limited general value. One reason is that each brand is a unique PCB mixture, as was each batch of a given brand. Another reason is that the amounts of toxic contaminants increase with some kinds of use, with PCDFs reaching thousands of parts per million in heat transfer equipment, but not always significantly increasing in capacitors. Byproduct formation depends on heat, availability of oxygen, and other factors; no two fires will produce precisely the same array of chlorinated polycyclic hydrocarbons.¹

Further complicating the picture is the fact that PCB mixtures found in environmental samples and human tissues often bear little resemblance to the original commercial formulations. It now appears that the PCB mixtures which accumulate in fish, and also those which contaminate human milk, might be more toxic than the commercial mixtures originally provided by Monsanto. If this is the case, it could be because the most biologically active PCBs bind most tightly to some of the body's proteins, and are therefore not available for destruction by other proteins known as "enzymes."

Current research is focusing on individual PCB and contaminant congeners. Modern analytical techniques are making identification of individual congeners routine. But even if a physician knew the effects of each PCB and contaminant congener individually, and a patient's burden of each now and in the past, along with the concentrations of all the potentially harmful metabolic byproducts within a patient's body, the significance would still be obscured by the fact that the individual congeners and byproducts work sometimes independently, sometimes competitively, and sometimes synergistically to exert their various effects. Add to this the likelihood that individuals vary over a wide range in their responses to individual PCBs, PCB contaminant, and combinations of PCBs and contaminants,² and in their histories of exposure to other chemicals, and it becomes apparent why doctors have difficulty diagnosing PCB poisoning.

But causality is seldom iron-clad in toxic tort litigation, and public policy regarding toxic substances is not contingent on the ability to make a diagnosis in individual cases. PCB exposure has specific adverse consequences in every species of laboratory animal which

has been tested.³ The effects of various doses on particular species are well documented. No experiments to determine dose/response relationships in humans should be attempted because of the long delay in eliciting responses and the cumulative effect of the poison. (Dioxin has actually been administered to prisoner "volunteers" in the United States.)⁴ The final word on this subject still has not been written. The anecdotes, analogies, and tragedies documented in this chapter will lead each reader to his and her own conclusions regarding the consequences of PCB exposure.

1. Milby et al., PCB-Containing Transformer Fires: Decontamination Guidelines Based on Health Considerations, 27 J Occup Med 351 (1985).

2. Large variations among individuals in cellular response to PCDFs is discussed in: Nagayama, Inducing Potency of Aryl Hydrocarbon Hydroxylase Activity in Human Lymphoblastoid Cells and Mice by Polychlorinated Dibenzofuran Congeners, 59 Environ Health Perspec 107 (1985).

3. The following is from Safe et al., PCBs: Structure-Function Relationships and Mechanism of Action, 60 Environ Health Perspect 47 (1985):

"Commercial PCBs, in common with other halogenated aromatics such as the polychlorinated dibenzo-p-dioxins (PCDFs) and polybrominated biphenyls (PBBs) elicit a number of common toxic and biologic effects. PCBs typically cause thymic atrophy, a wasting syndrome, immunotoxic responses, reproductive problems, porphyria, and related liver damage. In PCB-exposed animals, these effects are preceded by the induction of numerous enzymes including the hepatic and extrahepatic drug-metabolizing enzymes."

4. American Scientific, January, 1986.

Uniqueness of each of the 209 PCB congeners

Each of the 209 individual compounds known collectively as PCBs is unique. The six PCB congeners with at least two chlorine atoms at the meta and/or para positions of each ring (the 3, 3', 4, 4', 5, and 5' positions), but none at the ortho (2, 2', 6, or 6') positions, can readily assume a flat conformation which makes them approximately the size and shape of the most toxic dioxins. Compounds with only one or two chlorine atoms in the ortho positions and chlorine atoms in at least 4 of the meta and para positions also significantly resemble dioxins.

These six toxic PCB congeners have a strong tendency, as do the most potent dioxins, to bind to certain of the proteins involved in regulating the highly complex chemistry of life.¹ Other PCB congeners, particularly those with at least two chlorine atoms at the meta and/or para positions of each ring and only 1 or 2 chlorine atoms at the ortho positions, are also biologically active, and similar to dioxin in their modes of action. It has been suggested that chlorine atoms in the para positions are more important than chlorine atoms in the meta positions for potentiating the enzyme inducing ability, and presumably the toxicity by PCBs.² On the other hand, studies correlating PCB toxicity with similarities to the thyroid hormone, thyroxine, suggest that the meta positions are more important.³

It is believed that chlorine atoms in the ortho positions render PCBs less like dioxin by making it difficult, due to crowding by large chlorine atoms, for the two rings of the PCB molecule to assume a co-planar configuration. All the dioxins are planar.

The modes of action of PCB congeners which do not

resemble the dioxins are not well understood. While they are not potent acute toxins, their long term adverse effects could conceivably be worse than those of dioxin and the dioxin-like PCB congeners.

1. Safe et al., PCBs:Structure-Function Relationships and Mechanism of Action, 60 Environmental Health Perspectives 47 (1985).

2. Safe et al., Effects of Structure on Binding to the 2,3,7,8-TCDD Receptor Protein and AHH Induction - Halogenated Biphenyls, 61 Environmental Health Perspectives 21 (1985). Also: Safe et al., PCBs:Structure-Function Relationships and Mechanism of Action, 60 Environmental Health Perspectives 47 (1985), citing: Poland and Glover, chlorinated biphenyl Induction of Aryl Hydrocarbon Hydroxylase Activity: a Study of the Structure Activity Relationship, 13 Mol Pharmacol 924 (1977); Goldstein et al., Separation of Pure Polychlorinated Biphenyl Isomers Into Two Types of Inducers on the Basis of Induction of Cytochrome P-450 or P-448, 17 Chem-Biol Interact 69 (1977); and Parkinson et al., Polychlorinated Biphenyls as Inducers of Hepatic Microsomal Enzymes: Structure-Activity Rules, 30 Chem-Biol Interact 271 (1981).

PCB INDUCED ENZYMES

Enzymes are among the proteins which the body produces to help regulate itself at the molecular level. The function of enzymes is to determine the rates at which chemical reactions occur.

Some enzymes serve to speed up the rate at which the body destroys foreign chemicals. These enzymes are often referred to as "detoxifying" enzymes, despite the fact that they frequently change toxic chemicals into compounds which are even more toxic.¹ Compounds which are destroyed with the help of enzymes are called "substrates", and the products which are created in the process are called "metabolites". These metabolites are often more water soluble than the substrates, and hence more readily excreted in the urine. The most common metabolic pathway for PCBs involves addition of hydroxide groups (-OH). Hydroxylated PCBs may be excreted by becoming attached to sulfate or glucuronic acid to form a water soluble complex.² Metabolites which are not excreted are stored in the body or further metabolized into other, possibly more readily excreted compounds.

Foreign chemicals in the body stimulate the production and/or activity of detoxifying enzymes; or, as one is likely to encounter it in the technical literature, "induce" or "activate" detoxifying enzyme systems. In some situations, enzymes are activated by compounds which bind to them, so enzyme activity can increase without the actual number of enzyme molecules increasing.

PCBs are metabolized inside cells located in various parts of the body, but primarily located in the liver. Significant PCB metabolism takes place in the skin,³ and in the lungs. A variety of natural compounds are involved in the systems which oxydize PCBs. Oxygen atoms are provided in these systems by a variety of proteins which each have a flat, iron-containing part resembling hemoglobin, and which are known collectively as cytochrome P-450. These complete enzyme systems are sometimes called "mixed function oxygenase systems (MFO's)." PCB exposure induces the P-450 MFO system,⁴ including a sub-system involving cytochrome P-448 which is thought to be involved in transforming certain chemical compounds into carcinogens.⁵ There is evidence that traces of chlorinated dibenzofurans in PCB specimens contribute significantly to enzyme induction.⁶

Enzymes may be related to PCB toxicity in several

ways:

(1) By indicating that foreign chemicals are in the body, they help document exposure to PCBs.

(2) Prior exposure to PCBs increases the activities of enzymes which transform certain chemicals into toxic metabolites. For example, the anesthetic halothane is not, in itself, particularly toxic, but it is very toxic when certain enzymes are present at elevated levels. A woman may have been seriously injured by the anesthetic haloform as a consequence of occupational exposure to PCBs.⁷ Animal experiments show that vinyl chloride and fluorene become more toxic after exposure to PCBs.⁸

(3) Enzyme systems which have been induced by PCBs can interfere with therapeutic drug treatment by ridding the body of the drug more rapidly than anticipated by the prescribing physician.

(4) Some PCBs and PCB byproducts induce enzymes such as P-448 monooxygenases which can transform PCBs and other chemicals into reactive and potentially carcinogenic compounds called "arenes oxides" or "epoxides." PCB epoxides react with protein, and to some extent with genetic material, by becoming attached to it. Reactive PCB epoxides may help account for PCB-induced liver damage.⁹

(5) PCB induced enzymes can destroy hormones, the body's molecular messengers and regulators.

(6) The molecular events leading up to enzyme induction play a key role in scientific research into the biochemical basis of PCB toxicity. This research indicates that before some PCB induced enzymes can be manufactured inside the body's cells, PCBs must be transported inside the cell nucleus after binding to a protein called the "Ah-receptor." Species, as well as individuals within species, which have the most Ah-receptor protein tend to be the most sensitive to the ill effects of PCBs, dioxins, and related compounds.¹⁰ It has been found that some people have the Ah-receptor but others don't.¹¹ If this result is confirmed by other studies, then a genetic predisposition to PCB poisoning may have been found. Researchers in the field of PCB toxicity have already suggested that characterizing the extent of human pathology caused by PCBs and related compounds may require determining not only the levels of these toxic compounds have been in the body, but also whether or not the patient is "dioxin receptor positive" or "dioxin receptor negative."¹² PCDF induction

of one of the mixed function oxydases, known as "aryl hydrocarbon oxydase (AHH)," varies over a wide range in some human cells, suggesting that PCDFs are much more toxic to some people than to others.¹³

Experts sponsored by the chemical and electrical power industries acknowledge that PCBs induce enzyme production, but assert that this phenomenon merely demonstrates that the body is dealing with intruding molecules in a natural and harmless way. As General Electric puts it: "Such effects are also produced by many other substances, and are generally regarded as physiological rather than pathological processes".¹⁴

1. Rickenbacher et al., Structurally Specific Binding of Halogenated Biphenyls to Thyroxine Transport Protein, 29 J Med Chem 641 (1986) - citing Yoshimura and Yoshihara, PCB Poisoning and Pollution, P. 57 (1976) - suggest that 3, 3', 4, 4'-tetrachlorobiphenyl becomes significantly more toxic inside the body after the enzyme assisted substitution of an OH (oxygen and hydrogen) group for the hydrogen atom in the 5 position.

2. Bickers et al., (1982).

3. Calabrese, Methodological approaches to deriving environmental and occupational health standards, Wiley (1977)

4. Ryan et al., Separation and characterization of highly purified forms of liver microsomal cytochrome P-450 from rats treated with polychlorinated biphenyls, phenobarbital, and 2-methylchloanthrene, 254 J Biol Chem 1365 (1979); also: Parkinson et al., polychlorinated biphenyls as inducers of hepatic microsomal enzymes: Structure-activity rules, 30 Chem Biol Interac 271 (1980).

5. Alvares et al., Polychlorinated biphenyls: A New Type of Inducer of Cytochrome p-448 in the liver, 70 Proc Nat Acad Science USA 1321; also: Bickers, Microscope Immersion Oils: Effects of Skin Application on Cutaneous and Hepatic Drug-Metabolizing Enzymes, 24 Biochem Pharmacol 779 (1975).

6. Goldstein et al., 2,3,7,8-Tetrachlorodibenzofuran in a Commercially Available 99% Pure Polychlorinated Biphenyl Isomer Identified as the Inducer of Hepatic Cytochrome P-448 and Aryl Hydrocarbon Hydroxylase in the Rat, 6 Drug Metab Disp 258 (1978); also: Nagayama et al., Inducing Potency of Aryl Hydrocarbon Hydroxylase Activity in Human Lymphoblastoid Cells and Mice by Polychlorinated

Dibenzofuran Congeners, 59 Environ Health Persp 107 (1985).

7. Rosenblatt, A Possible Interaction of PCB and Halothane in Man, 51 Anesthesiology 95 (1979).

8. Jaegar, Chemical Modification of Acute Hepatotoxicity of Vinyl Chloride Monomer in Rats, 41 Toxicol 597 (1977); also: Murphy, Potentiation of Fluroxene Toxicity with Polychlorinated Biphenyls, 48 Toxicol Appl Pharmacol 87 (1979).

9. Shimada and Sato, Covalent Binding of Polychlorinated Biphenyls to Rat Liver Microsomes, 55 Toxicol Appl Pharmacol 490.

10. Safe et al., PCBs:Structure-Function Relationships and Mechanism of Action, 60 Environmental Health Perspectives 47 (1985).

11. Roberts et al., The Ah Receptor and Dioxin Toxicity: From Rodent to Human Tissues, 14 Chemosphere 661 (1985).

12. Schecter et al., Chlorinated Dioxin and Dibenzofuran Levels in Human Adipose Tissues from Exposed and Control Populations In: Rappe et al., (eds), Chlorinated Dioxins and Dibenzofurans in Perspective, page 51 (1986).

13. Nagayama et al., Inducing Potency of Aryl Hydrocarbon Hydroxylase Activity in Human Lymphoblastoid Cells and Mice by Polychlorinated Dibenzofuran Congeners, 59 Environ Health Persp 107 (1985).

14. Brown et al., Human Health Effects of Electrical-Grade PCBs, General Electric Company, Fairfield, Conn. (1981).

Research Concerning Enzymes and Disease

People who have suffered exposure to high levels of PCBs may be at increased risk of being harmed by other chemicals. One reason for this is that the victims of PCB exposure produce high levels of enzymes which can rapidly change other chemicals into potent toxins. Supporting this conclusion is the fact that PCBs can drastically increase the teratogenicity of dioxin in rodents.¹ There is also a study which shows that PCBs make grilled hamburger extract mutagenic to certain bacteria.² A possible case of PCBs making a usually harmless anesthetic into a potent liver toxin, involving a woman who worked in a capacitor manufacturing plant, has appeared in the medical literature.³ The enzymes which PCBs induce change some compounds to liver toxins,⁴ some into mutagens,⁵ some into embriotoxins,⁶ and others into carcinogens.⁷ It has been shown that relatively non-toxic polybrominated biphenyls greatly increased the toxicity of 1,2,4,6,7-pentabromonaphthalene.⁸

Another proposed explanation of observed synergisms involves the binding of PCBs to liver cell proteins which also binds toxins such as dioxin. It is speculated that by tying up the receptor protein binding sites, PCBs leave the other toxins free to do harm.⁹ Another explanation involves impairment of the immune system by PCBs.¹⁰

A great deal of evidence that PCB exposure makes people more vulnerable to the adverse effects of other chemicals comes from a procedure called the "Ames Salmonella/microsome mutagenicity test," a procedure which relies on PCBs or other compounds to induce enzymes.¹¹ In its simpler forms, the Ames test involves adding chemicals to bacterial cultures to determine whether the chemicals are mutagenic. The Ames Salmonella/microsome mutagenicity test involves adding to the bacterial culture not only the chemical being tested, but also an extract of the livers of rodents which had been pre-exposed to PCBs or other enzyme inducing compounds. In many cases, the compound being tested is mutagenic only in combination with PCB induced enzymes.

Trichloroethylene, a once very popular de-greasing solvent, may be especially dangerous to persons who have been exposed to PCBs.¹² Trichloroethylene was used in quantity at many capacitor and transformer manufacturing facilities.

1. Birnbaum et al, Toxic Interaction of Specific Polychlorinated Biphenyls and 2,3,7,8-tetrachlorodibenzo-p-Dioxin: Increased Incidence of Cleft Palate in Mice, 77 Toxicol Appl Pharmacol (1985); Also, Bandiera et al., Binding of Polychlorinated Biphenyl Classified as Either Pheno-barbitone, 3-methylcholentrene or Mixed-type Inducers to Cytosolic Ah Receptor, 39 Chem Biol Interact 259 (1982).
2. Felton et al, Mutagens in Cooked Foods -- Metabolism and Genetic Toxicity, 177 Adv Exp Med Biol 555 (1985).
3. Rosenblatt, A Possible Interaction Of PCB and Halothane in Man, 51 Anesthesiology 95 (1979).
4. Osimitz and Conolly, Mixed-function Oxidase System Induction and Propylene Hepatotoxicity, 15 J Toxicol Environ Health 39, (1985); Rosenblatt, A Possible Interaction of PCB and Halothane in Man, 51 Anesthesiology 95 (1979).
5. Welsh, Effects of Acute Chronic Polychlorinated Biphenyl Ingestion on Embryotoxicity Caused by Cyclophosphamide in Mice, 57 Arch Toxicol 104 (1985); Wild et al., Differential Mutagenic Activity of IQ (2Amino-3-methylimidazo[4,5-f]quinoline) in Salmonella Typh-imurium Strains in Vitro and in Vivo, in Drosophila and in Mice, 156 Mutat Res 93 (1985).
6. Greim et al, 39 Falk Symp. (Hepatology) 177, (1985); Talcott et al., The Effect of Lead and Polychlorinated Biphenyl Exposure on Rat Natural Killer Cell Cytotoxicity, 7(2) Int J Immunopharmacol 225 (1985).
7. Rosenblatt, A Possible Interaction Of PCB and Halothane in Man, 51 Anesthesiology 95 (1979).
8. McKinney et al, Structure-Induction Versus Structure-Toxicity Relationships for Polychlorinated Biphenyls and Related Aromatic Hydrocarbons, 60 Environ Health Perspect 57 (1985).
9. McKinney et al, Structure-Induction Versus Structure-Toxicity Relationships for Polychlorinated Biphenyls and Related Aromatic Hydrocarbons, 60 Environ Health Perspect 57 (1985).
10. Talcott et al., The Effect of Lead and Polychlorinated Biphenyl Exposure on Rat Natural Killer Cell Cytotoxicity,

7(2) Int J Immunopharmacol 225 (1985).

11. McCann et al, Detection of Carcinogens as Mutagens in the Salmonelly/Microsome Test: Assay of 300 Chemicals, 72 Proc Natl Acad Sci USA 5135 (1975).

12. Sato and Nakajima, Enhanced Metabolism of Volatile Hydrocarbons in Rat Liver Following Food Deprivation, Restricted Carbohydrate Intake, and Administration of Ethanol, Phenobarbital, Polychlorinated Biphenyl and 3-Methyl-cholanthrene: a Comparative Study, 15 Xenobiotica 67 (1985).

Enzyme Tests for Documenting Exposure

Physicians attending persons with known or potential PCB exposure commonly order routine blood and urine analyses. Where litigation is involved, the plaintiff's attorney as well as the defendants attorney have an interest in making sure such tests are performed. The tests often include enzyme analysis.

Enzyme analysis can serve two functions: it can provide evidence that exposure has occurred by documenting changes in enzyme levels after an incident involving potential exposure; and it can provide evidence of liver impairment after already documented past exposure. After a transformer fire, or any incident involving potential exposure to PCBs and related compounds, enzyme tests should be performed immediately so that subsequent changes, some of which occur over a period of weeks, can be monitored. Each individual has a unique balance of enzymes, so it is important to observe changes within individuals from as soon after an incident as possible.

The tests for urinary d-glucyric acid, 6-b-hydroxycortisol, and porphyrin balance are not direct enzyme tests - these compounds are merely byproducts of enzyme activity - but they can provide indirect evidence of enzyme system abnormalities. Measuring aminoantipyrine half life is another indirect test; the greater the amount of certain enzymes present, the faster aminoantipyrine is metabolized.

Enzymes which are routinely measured in PCB exposure cases are serum γ -GTP, SGOT, SGTP, and alkaline phosphatase. This is not because these are the enzymes of greatest interest; it is because they are easy to measure during blood tests. Hepatic mixed function oxydases are more interesting, but they do not circulate in the blood to an appreciable extent.

Theoretical Enzyme Research

Enzyme determinations are usually performed on experimental animals after they have been sacrificed. When the liver of the experimental animal is homogenized and centrifuged (spun in a test tube), solid material goes to the bottom. The liquid on top is called cytosol or cytosolic fluid; the particles on the the bottom are called microsomes.²

Some of the enzymes whose production is induced by PCBs are found in the cytosol. These include the group of enzymes called "cytochrome P-450", which are also involved in microsomal processes. "Cyto-" tells where it is found, "-chrome" refers to the fact that it is based on the iron containing pigment heme, and "450" refers to a frequency of radiation which it readily absorbs.

Enzymes found in the solids at the bottom of the centrifuge tube are called "microsomal enzymes". Thus, references to "hepatic microsomal enzymes" are not so much descriptions of the enzymes themselves as they are descriptions of where the enzymes were found in the animal (hepatic refers to the liver) and in the centrifuge tube. The microsomal portion is added to the cultures in the Ames Salmonella/microsome mutagenicity test mentioned earlier.

The degree to which various dioxin and PCDF congeners bind to certain proteins found in the cytosolic fluid correlates with their ability to induce the activities of various drug metabolizing enzymes; and it also correlates with their acute toxicity. Thus 2,3,7,8-TCDD, the most toxic dioxin congener, is the PCDD which binds most readily to the cytosolic protein, and is also the most potent inducer of enzymes.

How and where enzyme induction takes place is unknown. Some researchers believe that enzyme induction occurs after the toxin-protein complex migrates toward the center of the cell into what is called the cell "nucleus". (This would have been before the researcher removed, homogenized, and centrifuged the tissue or organ, of course, and isn't easily verified in the lab.) Others think it happens within minute bodies inside the cell called mitochondria.

The PCB congeners which bind to the cytosolic proteins are extremely toxic. They are the congeners which most resemble 2,3,7,8-TCDD in structure, i.e., they have chlorine atoms on either end and some ability to assume a

planar configuration.

Little is known about how the dioxins and the most toxic PCB congeners cause sickness and death. It appears that the enzymes which are induced are not the toxic agents because some potent enzyme inducers are virtually non-toxic. Scientists continue to investigate the puzzling link between toxicities of PCBs and related compounds and their abilities to stimulate the production of certain enzymes in the hope of solving the mystery of how these compounds cause sickness and death in living organisms.

FOOTNOTE AND RECENT REVIEWS

1. The microsomes are primarily fragments of cell membranes and minute structures called ribosomes and mitochondria. Genetic material is concentrated in the ribosomes, and it is there that most protein synthesis takes place. The mitochondria are responsible for producing energy and are rich in fats, proteins, and enzymes.

Safe, Polychlorinated Biphenyls and Polybrominated Biphenyls: Biochemistry, Toxicology, and Mechanism of Action, 13(4) CRC Crit Rev Toxicol 319 (1985).

McKinney et al., Structure - Induction Versus Structure - Toxicity Relationships for Polychlorinated Biphenyls and Related Aromatic Hydrocarbons, 60 Environ Health Perspect 57 (1985).

Neil, Mechanisms of the Biological Effects of PCBs, Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans in Experimental Animals, 60 Environ Health Perspect 41 (1985).

Safe et al., Structure-Function Relationships and Mechanism of Action, 60 Environ Health Perspect 47 (1985).

PCB METABOLITES

Metabolism is the sum of all the chemical processes inside the body which release energy from ingested substances; mainly, food. These reactions are the basis of life. Some of these reactions involve the addition of oxygen atoms to organic (carbon containing) compounds in order to produce energy. This is similar to the way oxygen adds to burning substances to produce energy, only the reactions in the body are controlled by enzymes.

In a broader sense, "metabolism" can include all chemical reactions inside the body, including removing or rearranging atoms in a molecule. As was mentioned earlier, whenever a molecule is transformed into another molecule inside the body, the original molecule is called a "substrate" and the new molecule is called a "metabolite".

PCB congeners with hydrogen atoms in adjacent positions tend to be most readily metabolized. 8 hours after injection of highly toxic 3,3',4,4'-tetrachlorobiphenyl into rats, 90% of the compound had been transformed into two metabolites. One of these metabolites was found to very active in suppressing levels of Vitamin A and thyroxin, a hormone produced by the thyroid gland.²

Sulfur containing PCB metabolites accumulate in the lungs, and may be responsible for the respiratory distress and reduced vital capacity associated with exposure to PCBs. Other PCB metabolites, called arene oxides, are of interest because some arene oxides are proven carcinogens.

PCB metabolites are primarily of interest to research scientists. Levels of these compounds are not commonly measured in exposure victims.

1. Brouwer and Berg, Binding of a Metabolite of 3,4,3',4'-tetrachlorobiphenyl to transthyretin Reduces Serum vitamin A Transport by Inhibiting the Formation of the Protein Complex Carrying Both Retinol and Thyroxin, 85 Toxicol Appl Pharm 301 (1986).

MAINTENANCE OF PCBs

PCBs can deteriorate in use. For example, extremely toxic compounds known as chlorinated dibenzofurans - more commonly known as polychlorinated dibenzofurans or "PCDFs" - can build up in the presence of oxygen and heat. PCDFs are generally thought to be a major contributing factor in the the world's worst documented PCB mass poisoning incidents, the 1968 Japanese tragedy known as Yusho, and the 1979 Chinese tragedy known as Yu-cheng. In both cases, PCBs being used as heat exchange fluid leaked into rice oil which was being heated. The use of PCBs as heat exchange fluids (except in transformers) in the United States is prohibited by the Toxic Substances Control Act of 1976. The major current domestic use for PCBs is in electrical transformers and capacitors which were manufactured prior to 1979.

Little is known concerning the possible build up of extremely toxic PCB byproducts in electrical equipment. Transformers get hot, but they do not normally contain air so it is unlikely that PCDF formation is common. Chlorinated biphenylenes are very toxic compounds which form from PCBs in the absence of oxygen, but little is known about their possible presence in electrical equipment fluids. Since toxic PCB byproducts are not likely to effect the performance of transformers or capacitors, no effort is made to monitor for their presence.

PCB maintenance is largely concerned with the buildup of compounds which conduct electricity. Such compounds are called "electrolytes", and the ones which occur most commonly in PCBs are water and hydrogen chloride. Electrolytes in PCBs can lead to electrical arcing which can damage equipment and further degrade the PCBs. Electrical arcing can also cause equipment to explode and catch fire.

Attorneys representing plaintiffs in cases involving PCB contamination due to arcing will use PCB maintenance reports to help determine possible negligence on the part of the owner of the equipment. The following guidelines are reproduced from the U.S. Department of the Interior, Bureau of Reclamation's Power O. & M. Bulletin No. 11 titled "Maintenance of Liquid Insulation."

EPIDEMIOLOGICAL FINDINGS

Other reports in this series deal with biochemical phenomina which are induced by PCB exposure, and which are sometimes associated with disease. This report takes a very different approach; it considers attempts to learn about the effects of PCB exposure on human health by describing poisoning incidents and observing trends among groups of people.

Because each person's biochemical makeup is unique, each person is affected uniquely by toxic chemicals. In a small group of people exposed to PCBs, it is possible that no specific adverse health trends would be observed.

For example, PCB exposure might cause a specific disease in 1 out of 200 people. If that disease afflicts 1 out of 600 unexposed people, then PCBs triple the risk of developing that disease. Such a situation would be apparent if thousands of exposure victims were examined; there would be no clue if a group of, say, 50 exposure victims were examined. In some situations, it might be easier for a physician to observe ways in which the known interactions between PCBs and the body's natural chemicals are affecting victims, than for a statistician to detect trends.

The mass poisoning incidents in Japan and Taiwan provide information concerning massive exposure to PCBs and related compounds. Symptoms of Yusho - which, in Japanese, means "rice oil poisoning" - are largely what would have been predicted based on studies involving experimental animals. Yusho was caused by rice oil which was contaminated by PCBs and PCDFs.

Very little information has resulted from attempts to survey the general population. Not enough people have been sampled and technical problems, such as fluctuating serum PCB levels in individuals over the course of a day, have compromised the data.

The situation involving occupational health surveys is also unsatisfactory. Information has been gathered, but it is inconclusive by the statistical measures commonly applied by epidemiologists. This has lead some industry representatives and government officials to assert that there is no conclusive evidence that PCBs and related compounds are associated with serious occupational disease. Other observers - many of whom have observed the victims of PCB exposure first-hand - are convinced that these compounds take a serious toll on the human body. Concerning the lack of dose-response relationships in data gathered from Yusho patients, Yoshimura and Hayabuchi wrote:¹

"Other subjective symptoms, such as fever, headache, dizziness, abdominal pain, swelling in the joints, changes in menstruation, and loss of hair failed to show consistent dose-response relationships. It

should be noted, however, that for these symptoms which failed to show dose-response relationships, it is impossible to deny a causal relationship."

1. Yoshimura and Hayabuchi, Relationship Between the Amount of Rice Oil Ingested by Patients With Yusho and Their Subjective Symptoms, 59 Environ Health Perspect 47 (1985).

Industry Position Regarding PCB Toxicity

Monsanto and some of the companies which used PCBs maintain that PCBs are virtually harmless. Spokesmen for these concerns will only say that heavy exposure can cause reversible skin conditions, but deny that there are any long term consequences of exposure. Their position rests partly upon the following assertions:

- 1) the effects of PCBs vary widely among strains and species, so only human studies are relevant;
- 2) the amounts of PCBs administered to experimental animals in most studies far exceeds the amounts to which humans have been exposed;
- 3) humans who apparently exhibit symptoms of PCB toxicosis have, in many instances, been shown to have been exposed to other substances, such as chlorinated naphthalenes or chlorinated dibenzofurans;
- 4) Excessive incidence of cancers or other adverse conditions are not found consistently from one study to another, hence they must be attributable to other factors;
- 5) Where excessive average ill health is reported among exposed populations, no statistically significant correlation can be found between PCB levels and the prevalence of symptoms, hence PCBs must not be to blame.

Working in the electrical equipment manufacturing industry and consuming fresh water fish have been the major documented routes of exposure to PCBs. It is clear that these activities have led to elevated PCB levels in blood, adipose tissue, and milk, but there is disagreement concerning the effects of exposure. Industry and government spokesmen, - relying heavily upon statistics - have observed the biochemical effects of PCBs on groups of workers, and observed that some of these workers are ill, but declined to connect biochemical changes with the symptoms of disease. The following is from William Gaffey of Monsanto (quoting Alexander Blair Smith of NIOSH)¹:

"The weight of evidence, as Smith et al. (1982) conclude, is that no studies to date "have shown that occupational exposure to PCBs is associated with any adverse health outcome, to be distinguished from demonstrable subclinical biochemical alterations."²

1. Smith et al., Metabolic and Health Consequences of occupational Exposure to Polychlorinated Biphenyls, 39 Br J Ind Med 361 (1982).

2. Gaffey, The Epidemiology of PCBs, in PCBs: Human & Environmental Hazards, page 279 (1983).

Possible Plaintiffs' Responses

Point "1" suggests that people, too, might vary widely in their susceptibility to the effects of PCBs.¹ This would have the following implications:

A) genetically resistant people might have an easier time maintaining employment at places of high exposure, hence there may be a tendency to selectively survey such people in workplace epidemiological studies;

B) if a population whose members vary widely in susceptibility is surveyed, the adverse affects on susceptible individuals could result in elevations in the average numbers of health complaints, even in the absence of a correlation between number of complaints and degree of exposure. In other words, those who are susceptible get sick, even at low levels of exposure, and no dose-response curve fits the data from the limited number of people observed.

The more diverse people are in their habits and physiologies, the more of them must be sampled to detect possible dose-response correlations. The populations studied to date may not have been large enough to detect such correspondences where they exist, or rule them out where they don't. This relates to point "5" above.

Point 2 does not apply to all studies. Reproductive and other effects have been studied at low levels of exposure.

Point 3 raises the issue of synergistic effects. By increasing enzyme levels, for example, PCB exposure can increase the body's susceptibility to harm from other chemicals. Point 4 also suggests the possibility that PCBs may be working in combination with other chemicals.

Also complicating epidemiological studies is the fact that current PCB levels are not good indicators of past exposure. For example, blood levels in Yusho victims nearly returned to normal while their symptoms persisted. In the absence of a reliable method for determining levels of past exposure, epidemiological studies can provide information of only limited value.

Another complication may be the possible reluctance of workers to complain of poor health to the company doctor and the people conducting the survey. It is possible that

workers felt that complaining might jeopardize their jobs.

1. Recent experiments using human lymphoblastoid cells suggest that human susceptibility to the effects of PCBs might parallel susceptibility to AHH induction by 3-methylcholanthrene. This suggests that by measuring AHH after administering a relatively non-toxic AHH inducer, humans could be screened for susceptibility to PCB toxicosis. See Nagayama et al., Inducing Potency of Aryl Hydrocarbon Hydroxylase Activity in Human Lymphoblastoid Cells and Mice by Polychlorinated Dibenzofuran Congeners, 59 Environ Health Perspect 107 (1985).

Chronology of PCB Studies

This report describes studies reported after 1950. Earlier studies are described elsewhere in this monograph.

1954 Meigs et al., Chloracne from an Unusual Exposure to Arochlor, 154 JAMA 1417. This report states that seven of fourteen workers developed chloracne between five and fourteen months after PCBs began to escape into the air of their workplace. It states that PCB levels in the air were only 0.1 mg/m³ and that only one worker exhibited abnormal liver function. That worker exhibited increases in cephalin flocculation and thymol turbidity. No changes in serum bilirubins, and SAP activity were reported among the workers.

1968 Yusho. Thousands were exposed to cooking oil contaminated with PCBs, PCDFs, and chlorinated quaterphenyls. Hyperpigmentation and severe chloracne, along with vomiting, diarrhea, and neurological abnormalities appeared after approximately three months of exposure. Victims complained of symptoms such as numbness of the extremities, headache, stomach pain, respiratory problems, and joint pain years after exposure ended. Elevated cancer and infant mortality were later reported.

1970 Crow, Chloracne, 56 Trans St John's Hosp Dermatol 79 (1970).

Two-thirds of the British capacitor industry were reportedly surveyed. The author concluded that chloracne was associated with exposure to pentachlorobiphenyl but not trichlorobiphenyl. He noted that penta- and hexachloronaphthalene are both considerably more acneogenic than pentachlorobiphenyl.

1972 Hasegawa et al., Report on Survey of Work Area Environment Where PCB is Handled and of the Health of Workers Handling PCB, in Special Research Report on Prevention of Environmental Pollution by PCB-like Substances, Japan, Research Coordination Bureau, Science & Technology Agency, pp 141-149 (1972).

This study involved 99 exposed workers and 30 controls. Exposed workers had average PCB serum levels of 370 ppm. Skin conditions were characterized as brown chromodermatosis of the dorsal joints of the hands and fingers and of the nail bed, and acneform exanthema. The acne condition was present on the jaw, back, and thighs of several workers. Dermal conditions did not correlate with serum PCB levels, so dermal contact was thought to be the primary cause. (See discussion of above for alternative

explanation involving congenital or acquired predisposition.)

This study also appears to show that PCBs affect fat metabolism. Decreased blood levels of neutral fats, total glycerides, phospholipids, beta-lipoprotein, and total, free, and esterified cholesterol were noted.

Enzyme activity changes were noted, but were considered clinically insignificant. The following enzyme activity changes were noted: increased SGOT, SGPT, SAP and decreased serum cholinesterase.

1973 Bumgarner et al., Polychlorinated Biphenyl Residues in Refuse Workers. Research Triangle Park, NC, US Dept Health, Education, & Welfare, Public Health Service, National Institute of Environmental Health Sciences, June 1973, 10 pp.

The hair and blood of 37 refuse workers were tested for PCBs. Only moderate exposure was documented, and it appeared to be unrelated to hematocrit, blood cholesterol, and blood pressure levels.

1973 Kitamura et al., PCB in Blood of Workers Employed in an Electrical Parts Manufacturing Plant., 15 Jpn J Ind Health 539 (Japanese).

In this study, 13 capacitor manufacturing workers were examined when use of PCBs was discontinued in 1972, and at three months and six months thereafter. PCB half-life in the blood after exposure ended was about 90 days. It is not clear whether length of exposure was important in determining half-life (average length of exposure was two and a half years), or at what rate total body burden declined.

Skin problems were attributed to PCB exposure. It was reported that no abnormalities were apparent from blood, liver function and urine tests.

1973 Kaappanen and Kolho, The Concentration of PCB in Human Blood and Adipose Tissue in Three Different Research Groups, In PCB Conference II, Stockholm, 1972. Solna, Sweden, National Swedish Environmental Protection Board/Publications 1973:4E, pp 124-28.

A small group of workers with varying exposure were tested. Even those with high PCB levels in their adipose tissue and blood were said to be in good health. Investigators reported that there were no apparent biologic effects from the PCBs.

1974 Hara et al., Follow-up Study of Condenser Factory After Use of PCB Discontinued (Part I)., 16 Jpn J Ind Health 365 (Japanese).

This is a follow up study of the workers studied by Hasegawa et al. in 1972. Skin conditions had improved and no correlation was found between serum triglyceride and blood PCB levels.

1975 Hara et al., Follow-up Study of Condenser Factory After Use of PCB Discontinued (Part III), 17 Jpn J Ind Health 371 (Japanese).

118 workers were studied. 45% complained of skin conditions ranging from blackheads to chloracne. These conditions did not correlate with blood PCB levels, but they did greatly improve after exposure was reduced. Elevated triglyceride levels were found among the most heavily exposed workers.

The two Hara et al. reports, and Hara's observations up until 1979 (Hara, Health Status and PCBs in Blood of Workers Exposed to PCBs and of Their Children, 59 Environmental Health Perspect 85 [1985]) are interesting particularly because they document decreases in PCB levels after its use was discontinued. Certain trends were apparent, but exceptions were noted. For example, of two workers who initially had blood levels of about 180 ppb, one had less than 10 ppb and one had more than 100 ppb a year later. In contrast with the 1974 study, this study found a significant correlation between serum triglyceride and blood PCB levels.

Another interesting aspect is the lack of correlation between reports of skin problems and serum PCB levels. Since chloracne and related conditions are generally conceded to be caused by PCBs, the lack of correlation is not used to demonstrate the alleged harmlessness of PCBs. But when any other condition or disease is found at above average levels among PCB workers, lack of correlation with serum PCB levels is used to discount causality. The lack of correlation might be attributable to widely varying genetic or acquired predispositions to PCB induced toxicosis, or exposure to chemicals whose toxicity is increased by prior exposure to PCBs.

1975 Inoue et al., Discovery of PCB Pollution in a Textile Factory--I. PCB Level in Blood Serum of Laborers and Results of Physical Examination, 22 Jpn J Public 481 (Japanese).

Over twenty heavily exposed and thirty moderately exposed textile workers were examined. Skin and other abnormalities were described as "relatively mild" according to the 1977 NIOSH Criterea document.

1975 Michigan Department of Public Health (Lansing), Final

report on FDA contract 223-73-2209, Evaluation of Changes in the Level of Polychlorinated Biphenyls (PCBs) in Human Tissue.

PCB intake through fish consumption did not appear to be related to health problems. (A recent study did find that developmental problems in young children were related to Great Lakes fish consumption.)

1976 Inoue et al., Use and Health Effects of Arochlor 1242, a Polychlorinated Biphenyl, In an Electrical Industry, 31 Arch Environ Health 189.

34 Australian workers were studied. Some workers had elevated serum alkaline phosphatase and SGOT levels. Slight BSP retention was noted in four of seven workers judged to have had the heaviest exposures. Average liver function tests were within normal limits.

1976 Bahn et al, Melanoma After Exposure to PCBs, 295 New Engl J Med 450.

Statistically significant excess malignant melanoma and pancreatic cancer were reported among workers thought to have been exposed to PCBs in the Mobile Oil Plant in Paulsboro, New Jersey. PCBs may have acted as co-carcinogens with other, unidentified compounds.

1978 SCDH Study of Pickins, SC Plant of Sangamo Capacitor Division (News Report). South Carolina Department of Health and Environmental Control, Columbia, South Carolina.

1978 Alvares et al, Alterations in Drug Metabolism in Workers Exposed to Polychlorinated Biphenyls, 22 Clin Pharmacol Therap 140, (1978).

Increased rate of metabolism of antipyrine was observed among exposed workers. This is evidence of increased microsomal enzyme activity.

1979 Fischbein et al., Clinical Findings Among PCB Exposed Capacitor Manufacturing Workers, 320 Ann NY Acad Sci 703-714.

326 capacitor plant workers were studied. Skin problems and decreased vital capacity of the lungs were noted, as were a large number of complaints related to the nervous system. Little correlation between complaints and either duration of employment or plasma PCB levels was found. Of the numerous questions and tests administered to the participants, the only statistically significant correlations were between skin disease among male participants and higher chlorinated PCBs; between serum PCB levels and SGOT; and between PCB exposure and the rate of antipyrine metabolism. The last result suggests that liver

microsomal enzymes (drug metabolizing enzymes) were more active among the exposed workers.

1979 Warshaw et al., Decrease In Vital Capacity in PCB-Exposed Workers in a Capacitor Manufacturing Facility. 320 Ann NY Acad Sci 277.

14% of 243 workers exposed to PCBs for an average of greater than 15 years were found to have a reduced Forced Vital Capacity. Those with occupational exposure to asbestos, talc, or textile dust were not included. Smoking was discounted as a possible cause.

1980 Baker et al., Metabolic Consequences of Exposure to Polychlorinated Biphenyls (PCB) in Sewage Sludge, 112 Amer J Epidemiol 553.

No consequences were noted, except for a statistically insignificant elevation in PCB levels among Bloomington, Indiana, residents not exposed to the municipal sludge. Perhaps the most significant finding was the extraordinary background serum PCB level of 11.6 ug/L. Possible past contamination of the public water supply or widespread PCB dumping might account for this finding. PCBs were quantified as Aroclor 1254, a type of PCB not often found in local dumps, but which is in the lake sediment near the city's water intake pumps. But since lower chlorinated PCBs (the type which predominates in the dumps) are rapidly metabolized, higher chlorinated minor components of the predominantly lower chlorinated mixtures are what the body retains.

1981 Kreiss et al., Association of Blood Pressure and Polychlorinated Biphenyl Levels, 245 JAMA 2505.

This study of 458 people exposed to PCBs through eating fish showed that higher PCB levels were associated with increased age, increased fish consumption, higher serum cholesterol levels, higher serum GGTP levels, and higher diastolic blood pressure.

1981 Maroni et al., Occupational Exposure to Polychlorinated Biphenyls. II. Health Effects, 38 Br J Ind Med 55.

80 workers were studied. Chloracne was noted, but its occurrence did not correlate well with degree of exposure. Liver changes, possibly related to the induction of liver microsomal enzymes, did correlate significantly with blood trichlorobiphenyl levels, but not with tetrachlorobiphenyl levels.

Two fast-developing cavernous haemangiomas and one case of myelocytic chronic leukemia were observed. Eight workers complained of symptoms related to digestive

difficulties.

1981 Lawton, Studies of Employees Occupationally Exposed to PCBs, General Electric Research and Development Center, Schenectady, New York.

194 workers from the two plants studied in 1979 by Warshaw et al. and Fischbein et al. were evaluated in 1976 and again in 1979. (These plants were also studied by Brown and Jones in 1981.) Lawton reports that no evidence of skin disorders or any other health problems were found.

A correlation between the logarithms of serum PCB and cholesterol, triglycerides, and GGTP was found, but the correlation disappeared when adipose PCB concentrations were used. This suggests that the correlation is partly due to PCBs being more soluble in blood with higher triglyceride levels (triglycerides are lipids, so this is reasonable).

Bertazzi et al., Mortality Study of Male and Female Workers Exposed to PCBs. Presented at the International Symposium on Prevention of Occupational Cancer, April, 1981, Helsinki, Finland.

1310 capacitor production workers were studied. Cancer was elevated for both males and females, and was considered statistically significantly among the males. Both males and females had higher than expected frequencies of lymphatic and hematopoietic neoplasms. Three of the men died from neoplasms of the digestive organs and peritoneum, compared with 0.88 expected. The overall death rate among the female workers was significantly elevated.

1981 Brown and Jones, Mortality and Industrial Hygiene Study of Workers Exposed to Polychlorinated Biphenyls, 36 Arch Environ Health 120.

2567 capacitor production workers were studied. Cancer of the liver and rectum were elevated. No excess mortality from circulatory disease was observed.

1982 Zack and Musch, Mortality of PCB Workers at the Monsanto plant in Sauget, Illinois.

The vital status of 89 PCB production workers at Monsanto's Sauget, Illinois plant was evaluated. It was discovered that 4 of them had died of lung cancer; the expected number of deaths was 1.4. A statistically significant excess of deaths due to diseases of the circulatory system was discovered among the white, male workers. 7, as opposed to the expected 1.8, died of this cause.

1982 Chase et al., Clinical and Metabolic Abnormalities

Associated With Occupational Exposure to Polychlorinated Biphenyls (PCBs), 24 J Occup Med 109.

120 male railroad workers were studied. Fat PCB levels correlated with length of employment better than serum levels. A significant correlation between serum, but not fat, PCB levels and SGOT was noted. This is reminiscent of the 1981 Lawton study in which the correlation between PCBs and GGTP disappeared when fat, instead of blood, was considered. No correlation between fat or serum PCBs and GGTP was found in the Chase study.

1982 Smith et al., Metabolic and Health Consequences of Exposure to Polychlorinated Biphenyls, 39 Br J Ind Med 361-369.

The authors studied 321 workers at three sites. They found serum PCB concentrations to be positively and significantly correlated with mucous membrane and skin irritation, systemic malaise, altered peripheral sensation, SGOT, GGTP, and plasma triglycerides. They suggested that SGOT, GGTP, and plasma triglyceride induction might all be related to the induction of liver microsomal enzymes, or that they might reflect damage to the liver - or both.

The authors also discovered a negative correlation between PCB levels and high density lipoprotein-cholesterol (HDL-cholesterol). Since there is an inverse correlation between HDL-cholesterol and coronary artery disease, PCBs are implicated as a possible cause of coronary artery and heart disease.

1982 Kreiss et al., Serial PBB Levels, PCB Levels and Clinical Chemistries in Michigan PBB Cohort. 37 Arch Environ Health 141, (1982).

Primarily a PBB study.

1983 Emmett et al., Health Effects of PCB Exposure of GSA Switchgear Employees, NIOSH Technical Assistance Request #80-7, the Johns Hopkins University School of Hygiene and Public Health, Baltimore, Maryland, 1983. 55 electricians were studied. Abnormalities in the peripheral nervous system were documented. The logs (a mathematical term) of serum PCBs and SGOT correlated positively. serum log PCB and urinary 17-hydrocorticosteroid excretion were found.

The average blood level of the exposed electricians was reported to be 12ug/L, the same level Baker reported as background in Bloomington, Indiana.

1983 Humphrey, Population Studies of PCBs in Michigan Residents, page 299 of PCBs: Human and Environmental Hazards, 1983.

Humphrey reports significantly elevated frequency of cancer among farmers with PCB treated silos. Of 183 participants in the study, 0.3% were expected to have cancer based on data from the national Surveillance, Epidemiology, & End Results Program (SEER). The cancer rate observed was 3.3%.

The PCB silo cohort reported high frequencies of tiredness, headaches, rashes, numbness, and joint problems. This study shows that PCB victims had many symptoms in common with Michigan's PBB exposed farmers.

Humphrey also reviews data showing eating Great Lakes fish increases the bodies burden of PCBs. No adverse health consequences from this route of exposure are reported.

1984 Takamatsu et al., PCBs in Blood of Workers Exposed to PCBs and Their Health Status, 5 Amer J Ind Med 59. This study concludes that contaminants in PCBs, or exposure to chemicals along with PCBs, result in disease, but that PCBs alone do not appear to be very toxic.

1984 Amano et al., Statistical Observations About the Causes of the Death of Patients With Oil Poisoning, 39 Japan Hygiene (1984). Translated for the U.S. EPA by SCITRAN, Santa Barbara, California.

70 people out of a study cohort of 1086 Yusho victims had died by 1983. Statistically significant excesses of cancer of the liver among men, and ischemic heart disease among women were observed. Respiratory cancers were also higher than expected.

1985 Anonymous, Provisional Final Report of Exposure Assessment/Health Effects Study: Bloomington, Indiana - 1984, Indiana State Board of Health and the U.S. Public Health Service, Center for Disease Control, Atlanta.

114 residents of Bloomington, Indiana were divided into various categories and evaluated by means of blood tests and questionnaires. This report omits the data necessary for an adequate evaluation of possible consequences of PCB exposure, although it does provide tables assigning values to statistical variable in various categories. It is clear, however, that higher serum PCB levels were associated with elevated serum triglycerides, total bilirubin, cholesterol, and self reported, physician diagnosed high blood pressure and liver ailments. Higher serum PCB levels were associated with lower HDL-cholesterol levels. Average PCB levels for randomly selected residents were lower than the levels reported by Baker in 1980, but still significantly higher than expected based on similar populations.

1985. Lawton et al., Effects of PCB Exposure on Biochemical and Hematological Findings in Capacitor Workers, 60 Environ Health Perspect 165.

"No evidence for health impairment related to PCB;s was found, despite the high serum levels of PCBs in the study population", according to this General Electric Company reevaluation of the study published by Dawton et al., in 1981 (see above). Serum PCB levels were said to increase as a result of increased serum triglyceride and cholesterol levels. This is not a unique point of view, but it is the reverse of the view that PCBs cause serum triglyceride and cholesterol levels to rise. The authors suggest that obesity might account for high incidence of elevated serum triglyceride levels. Higher than expected levels of glucose and cholesterol were also reported, and likewise were thought to be related to factors other than PCB exposure. Other findings were elevated BUN and chloride, in monocytes and eosinophils, in urinary specific gravity and cell count, and in calculated serum osmolality. WBC's returned to near normal from higher levels observed in 1976.

The authors also found no evidence of liver damage. The tables indicate possible immune system disturbances, but these were not discussed.

This study is noteworthy not only because it may give an indication of Industry's current view of PCB toxicity - General Electric is unlikely to contest these findings, and the interpretations of the data were probably cleared at the highest levels - but because of the discussion of microsomal enzyme induction. The authors convincingly demonstrate, with their own results and by reference to the literature, that PCB exposure at levels once commonly encountered by workers induces the microsomal enzymes GGTP, glucuronyl transferase (detected indirectly by increased d-glucuronic acid elimination and decreased levels of bilirubin), and P-450 oxydase.

The authors conjecture that their findings of no liver damage might be related to their assertion that serious liver damage only occurs in rats after PCBs in their bodies reach levels over 1000 times the levels found in capacitor plant workers.

1985. Emmett, Polychlorinated Biphenyl Exposure and Effects In Transformer Repair Workers, 60 Environmental Health Perspect 185.

55 present and past transformer repair workers and 56 non-exposed workers were evaluated at Johns Hopkins Medical Institution in Baltimore. Average PCB serum and lipid burdens in the exposed group were only about three

times background. Statistically significant correlations between adipose PCBs and 24-hour urinary 17-hydrocorticosteroid excretion and with serum PCBs and serum γ -glutamyl transpeptidase. Correlations between serum triglycerides and cholesterol and serum PCBs, but not adipose PCBs were found. This seemed to rule out the hypothesis that PCBs were responsible for increased triglyceride and cholesterol. Abnormal serum albumin, serum LDH were found among the exposed workers, serum total protein, and serum alkaline phosphatase. These last four findings suggest PCB related abnormal liver function, but when adipose PCB levels are compared, the only correlation between PCBs and liver function parameters was a negative one with SGTP.

Possible evidence of microsomal enzyme induction was found, but the only direct test, antipyrine half-life, was negative. Excess urinary excretion of D-glucaric acid was not observed either.

Evaluation of the data was still in progress at the time of the study. This study is particularly noteworthy because efforts are being made to evaluate correlations between clinical findings and levels of specific congeners.

1985 Cordle et al., Determination of Human Risk in Regulating Polychlorinated Biphenyls (PCBs) - A Case Study

This article uses cancer and reproductive effects data from animal studies to assess the impact on human health of lowering or raising the amount of PCBs allowed in fish.

1985 Schechter and Tiernan, Occupational Exposure to Polychlorinated Dioxins, Polychlorinated Furans, Polychlorinated Biphenyls, and Biphenylenes After an Electrical Panel and Transformer Accident in an Office Building in Binghamton, NY, 60 Environ Health Perspect 305.

Voluntary medical surveillance of 50 patients who believed they had been exposed in the 1981 Binghamton, New York, office building fire showed transient skin rashes while in the contaminated building, skin cancers (three known cases), nervousness, irritability, difficulty sleeping, im-potence, fatigue, elevated serum cholesterol and triglyceride levels, hypertension, and "psychoneurotic" illness. A suicide may have resulted from anxiety over exposure.

1985 Hara, Health Status and PCBs in Blood of Workers Exposed to PCBs and of Their Children, 59 Environ Health Perspt 85.

Hara updates his earlier studies. He notes that Yusho-like symptoms among children whose mothers were

industrially exposed have not been diagnosed as PCB poisoning partly because they are not related to blood PCB levels.

1985 Sahl et al., Polychlorinated Biphenyls in the Blood of Personnel from an Electric Utility, 27(9) J Occup Med 639.

1986 Brown, Mortality of Workers Exposed to Polychlorinated Biphenyls - An Update, Centers for Disease Control, Department of Health and Human Services, Cincinnati, Ohio.

This is an update of Brown's 1981 retrospective mortality study. The number of deaths reported among the 2588 workers in the study cohort rose from 163 to 295. The study shows significantly elevated instances of cancer of the liver and biliary passages. Except for a slight increase in death due to diseases of the circulatory system, mortality from other causes was less than expected.

1987 Nicholson et al., Mortality Experience of Workers Exposed to Polychlorinated Biphenyls During Manufacture of Electrical Capacitors, Industrial Disease Standards Panel, Ontario Ministry of Labor.

Some increase in mortality from non-infectious respiratory disease and from accidents, violence, and suicide was observed. The authors conclude that the study shows no association between PCB exposure and death from any cause.

EFFECTS OF PCBs ON THE IMMUNE SYSTEM

Failure of the immune system can result in infections of greater duration and frequency, and greater susceptibility to contracting cancer. Stimulation of parts of the immune system can cause allergic reactions, joint inflammation, and possibly other medical problems. PCBs are known to suppress some aspects of the immune systems of some life forms, and stimulate some aspects of the immune systems of others (see bibliography). The persistent respiratory infections suffered by Yusho victims¹ may result from the suppression of aspects of their immune systems; the joint pains which afflict the victims of PCB exposure may result from the stimulation of some aspects of their immune systems.

The body's response to invasion by matter such as bacteria, viruses, cancer, foreign objects and chemicals involves two major, somewhat interrelated systems: the humoral immune system and the cell-mediated immune system. The humoral response is quick and is largely independent of the thymus. The cell-mediated response provides more long term and specific immunity. The functions of these systems are intricately interrelated, and as the articles in this report's bibliography indicate, PCBs can affect both of them.²

1. Chronic respiratory infection among yusho victims is documented in Shigematsu et al., Respiratory Involvement in Polychlorinated Biphenyls Poisoning, 16 Environ Res 92 (1978). These infections may also be related to the accumulation of PCB metabolites in the lungs.

2. The following summary of the effects of PCBs on the immune system appeared in a report issued by the State of California:*

"A number of reports implicate PCBs as immunosuppressants.* Lymphoid atrophy has been observed in rabbits, chickens and guinea pigs. Suppression of humoral immune responses to several antigens was observed in rabbits and guinea pigs, and decreased cell-mediated immune response followed PCB exposure in guinea pigs. A decreased tolerance to hepatitis virus was seen in ducklings without apparent intoxication. In monkeys exposed transplacentally and through contaminated milk, the lymph nodules of the spleen were extremely small and without germinal centers;† morphologic changes were indicative of reduced immunologic competence."

* Letz, Toxicity of PCBs, California Department of Health Services, p.18 (1981; updated, 1982) Citing: (a) Fishbein, Toxicity of Chlorinated Biphenyls, 14 Annu Rev Pharmacol 139 (1974) and (b) Allen and Barsotti, The Effects of Transplacental and Mammary Movement of PCBs on Infant Rhesus Monkeys, 6 Toxicol 331 (1976).

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Loose et al, Polychlorinated Biphenyl and Hexachlorobenzene Induced Humoral Immunosuppression, 22 J Retic Soc 253 (1977).

Thomas and Hinsdill, Effect of Polychlorinated Biphenyls (PPB) on Immune Response of Rhesus Monkeys and Mice, 44 Toxicol Appl Pharmacol 41 (1978).

Talcott et al., The Effect of Lead and Polychlorinated Biphenyl Exposure on Rat Natural Killer Cell Cytotoxicity, 7(2) Int J Immunopharmacol 255 (1985).

Vos and de Roij, Immunosuppressive Activity of a Polychlorinated Biphenyl Preparation on the Humoral Immune Response in Guinea Pigs, 21 Toxic Appl Pharmacol 549

(1972).

Vos and van Driel Grootenhuis, PCB-Induced Suppression of the Humoral and Cell-Mediated Immunity in Guinea Pigs, 1 Sci Total Environ 289 (1972).

Koller and Thigpen, Reduction of Antibody to Pseudorabies Virus in Polychlorinated Biphenyl-Exposed Rabbits, 34 Am J Bet Res 1605 (1973).

Loose et al, Polychlorinated biphenyl and Hexachlorobenzene Induced Humoral Immunosuppression, 22 J Retic Soc 253 (1977).

Street and Sharma, Alteration of Induced Cellular and Humoral Immune Responses by Pesticides and Chemicals of Environmental Concern, 32 Toxicol Appl Pharmacol 587 (1975).

Thomas and Hinsdill, Effect of Polychlorinated Biphenyls (PPB) on Immune Response of Rhesus Monkeys and Mice, 44 Toxicol Appl Pharmacol 41 (1978).

Vos and van Driel Grootenhuis, PCB-Induced Suppression of the Humoral and Cell-Mediated Immunity in Guinea Pigs, 1 Sci Total Environ 289 (1972).

Lu and Wu, Clinical Findings and Immunological Abnormalities in Yu-Cheng Patients, 59 Environ Health Perspect 17 (1985).

Faith et al., Effects on Immunocompetence by Chemicals of Environmental Concern, in Reviews in Biochemical Toxicology 2, p. 183 (1980).

McConnell, Acute and Chronic Toxicity, Carcinogenesis, Reproduction Teratogenesis and Mutagenesis in Animals, in Halogenated Biphenyls, Terphenyls, naphthalenes, Dibenzodioxins and Related Products, p.109 (1980).

Smith et al., Immunotoxicological Evaluation on Mice Exposed to Polychlorinated Biphenyls, 45 Toxicol Appl Pharm 330 (1978).

Silkworth and Loose, Cell-Mediated Immunity in Mice Fed Either Aroclor 1016 or Hexachlorobenzene, 45 Toxicol Appl Pharmacol 326 (1978).

Harris et al., Antibody Response and Reproduction in Bursa of Fabricus and Spleen Weights of Progeny of Chickens Fed

PCBs, 55 Poultry Sci 1933 (1976).

Animal Studies

The most direct method of measuring immune system changes is counting antibodies in the blood. Less direct means of monitoring immune system effects involve introducing foreign material into experimental animals to see how well their immune systems meet the challenge, or physically examining tissues (e.g., bone marrow), organs (e.g., the spleen) or glands (e.g., the thymus).

Introducing foreign substances into PCB exposed experimental animals has shown that PCBs usually suppress the immune response, but that under some circumstances, PCB exposure strengthens it. Evidence for suppression comes from a study showing ducklings fed PCBs have a reduced tolerance to hepatitis virus.¹ Another study reports reduced ability to resist malaria and endotoxin in PCB exposed mice.² Evidence for stimulation of the immune response comes from experiments involving skin grafts,³ foreign non-cancerous cells,⁴ and cancer cells⁵.

It has been shown that organs essential for normal immune response can be harmed by PCBs. Chickens fed PCBs had small spleens⁶ and atrophied lymphoid glands.⁷ Adverse effects on the lymph glands, thymus, and spleen were observed in exposed rabbits.⁸

1. Freind and Trainer, Polychlorinated Biphenyl: Interaction with Duck Hepatitis Virus, 170 Science 1314 (1970); but J.G. Vos, a leading researcher in the field of chemically induced immune system impairment, cautions that increased susceptibility to hepatitis could be due to the weakening of the liver by PCBs, and could have nothing to do with the immune system. See Vos et al, Immune alterations, in Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products, p.245 (1980).
2. Loose, Impaired Host Resistance to Endotoxin and Malaria in Polychlorinated Biphenyl- and Hexachlorobenzene-Treated Mice, 20 Infect Immun 30 (1978).
3. Flick et al, Studies of the Chick Disease: Similarity of Symptoms Produced by Feeding Chlorinated Biphenyl, 44 Poultr Sci 1460 (1965).
4. Vos and Koeman, Comparative Toxicologic Study with Polychlorinated Biphenyls in Chickens with Special Reference to Porphyria, Edema Formation, Liver Necrosis, and Tissue Residues, 17 Toxicol Appl Pharmacol 656 (1970).
5. Vos and Beems, Dermal Toxicity Studies of Technical Polychlorinated Biphenyls and Fractions Thereof in Rabbits, 19 Toxicol Appl Pharmacol 617 (1971).
6. Silkworth and Loose, Cell-Mediated Immunity in Mice Fed Either Aroclor 1016 or Hexachlorobenzene, 45 Toxicol Appl Pharmacol 326 (1978).
7. Bonnyns and Bastomsky, Polychlorinated Biphenyl Induced Modification of Lymphocyte Response to Plant Mitogens in Rats, 32 Experientia 522 (1976); Also: Silkworth and Loose, PCB and HCB Induced Alteration of Lymphocyte Blastogenesis, 49 Toxicol Appl Pharmacol 886 (1979).
8. Kerkvliet and Kimeldorf, Antitumor Activity of a Polychlorinated Biphenyl Mixture, Aroclor 1254, in Rats Innoculated with Walker 256 Carcinosarcoma Cells, 59 J Natl Cancer Inst 951 (1977).

Clinical Immune System Tests

1. Delayed-type hypersensitivity (DTH) tests. These are similar to the skin patch tests which are used to identify allergies. The DTH tests which were performed during the Quail Run Mobile Park in Gray Summit, Missouri,¹ were done with a device called a Multitest CMI, which tests skin responses to tetanus, diphtheria, Streptococcus, tuberculin, Candida, Proteus, and Trichophyton. A weak response to these antigens indicates that the immune system is suppressed, a condition called anergy.
2. T-Cell Tests These are blood tests for antibodies produced by the thymus. T-cells are given designations such as T3, T4, T8 and T11 according to their surface characteristics. Diminished total levels of T-cells, as well as abnormal ratios of the various types, are indications of impairment to the immune system.²
3. Lymphocyte Proliferative Response.³
4. Allogenic T Cell Cytotoxicity*

1. Hoffman et al., Health Effects of Long-term Exposure to 2,3,7,8-Tetrachlorodibenzo-p-Dioxin, 255 JAMA 2031 (1986).
2. Hoffman et al., Simple and Rapid Measurement of Human T Lymphocytes and Their Subclasses in Peripheral Blood, 77 Proc Natl Acad Sci USA 4914 (1981).
3. Schiff et al, Membrane Receptors and in Vitro Responsiveness of Lymphocytes in Human Immunodeficiency, 112 J. Immunol 376 (1974).
4. Namiuchi et al., Quinacrine Inhibits the Primary but not the Secondary Proliferative Response of Human Cytotoxic T Cells to Allogeneic Non-T Cell Antigens, 132 J Immunol 1456 (1984).

ANTIBODIES Antibodies are large proteins which engulf foreign substances which enter the body. They are the first line of defense against invasion by splinters, bacteria, viruses and toxic chemicals. Capture by antibodies can set off a chain of events leading to the production of more antibodies and various other natural substances which combine to destroy (change into a new chemical) the intruders. Since the body cannot efficiently store energy from the destruction of foreign chemicals by antibodies, the destruction process is not normally called metabolism and the byproducts are not called metabolites. Antibodies include the "T-cells" produced by the thymus and "B-cells" produced by the bone marrow. Together, the various types of antibodies make up what is called "immunoglobulin".

Testing for the various classes of immunoglobulin in the blood is routine, and is indicated whenever PCB exposure is suspected. Among victims of the Yu-Chen poisoning incident in Taiwan, two forms of immunoglobulin (IgA and IgM) decreased, but IgG remained normal. Although some aspects of the immune system were found to be suppressed, others were stimulated. Inappropriate stimulation can lead to numerous adverse consequences including joint inflammation and behavioral changes.

WHITE CELLS: White blood cell counts should be obtained in cases of possible exposure to PCBs and related compounds. Low levels of PCBs have been found to reduce levels of both leukocyte and lymphocyte white blood cells

in experimental animals.

Damage to the Nervous System

The nervous system conducts electrical impulses within the brain, and between the brain and other parts of the body. Impairment of the nervous system can result in subjective phenomena such as numbness, pain, and tingling sensations; in physical phenomena such as tremors and slowed reflexes; and in personality change and aberrant behavior.

PCBs and related compounds adversely affect the nervous system at dose levels typically encountered by victims of occupational or accidental exposure. This conclusion is inescapable in view of subjective complaints and physiological verification in both humans¹ and experimental animals².

The early literature on occupational exposure to chlorinated naphthalenes and PCBs (these were frequently encountered in combination) contains references to worker complaints of lethargy, impotence, headaches, insomnia, and disturbance in taste.³ Later surveys involving workers exposed to PCBs but not the chlorinated naphthalenes continued to produce accounts of problems related to the nervous system

It is impossible to say to what degree the workers' distress was due to systemic damage not directly related to the nervous system. For example, impotence can result from weakness due to general ill health, as well as from damage to nerves serving the penis. Recent research shows that to some degree the workers suffered an assault on their nervous systems.⁴

Victims of the Yu-cheng poisoning catastrophe in Taiwan reported hearing difficulties, dizziness, sluggish or absent deep tendon reflexes, pain over back and limbs, headaches, numbness of limbs, weakness and muscle spasms. Similar effects were documented after the Yusho disaster in Japan.

Damage to the peripheral motor and sensory nerves has been extensively documented through nerve conduction velocity tests. The Japanese and Taiwanese rice-oil poisoning victims' nervous systems were clearly damaged.

The initial complaints of those exposed to PCBs and PCB combustion products included headaches, irritability, anxiety, insomnia, sexual difficulties, and tingling in the extremities.⁵ One victim became obsessed with his exposure and attempted to kill himself.

1. (a) Chia and Chu, Neurological Studies on Polychlorinated Biphenyl (PCB)-Poisoned Patients, 5 American Journal of Industrial Medicine 117 (1984), citing numerous Japanese and Taiwanese studies; also (b) Okumura, Past and Current Medical States of Yusho Patients, 5 Amer J Ind Med 13 (1984). These Articles are Reprinted in Kuratsune and Shapiro (eds.), PCB Poisoning in Japan and Taiwan, Alan R. Liss, New York (1984); and (c) Seppalainen et al., Reversible Nerve Lesions After Accidental Polychlorinated Biphenyl Exposure, 11 (2) Scand J Work Environ Health 91 (1985).

2..(a) Fingerman and Short, Change in Neurotransmitter Levels in Channel Catfish After Exposure to Benzolalpyrene, Naphthalene and Aroclor 1254, 30 Bull Environ Contam 147 (1983);

(b) Rosin and Martin, Neurochemical and Behavioral Effects of Polychlorinated Biphenyls in Mice, 2 Neurotoxicology 749 (1981);

(c) Fingerman and Russell, Effects of the Polychlorinated Biphenyl Aroclor 1254 on Locomotor Activity and the Neurotransmitters Dopamine and Norepinephrine in the Brain of the Gulf Killfinch, 25 Fundulus Grandis Bull Environ Contam Toxicol 682 (1982);

(d) Karlsson et al, Locomotory and Dehydrogenase Activities of Red Starts, Phoenicurus phoenicurus L. (Aves) given PCB and DDT, 7 Environ Pollut 53 (1974);

(e) Dieter, Influence of Environmental Contaminants on Biochemical Adaptation to Stress in Birds, 29 Toxicol Appl Pharmacol 110 (1974);

(f) Kreitzer and Heinz, The Effect of Sublethal Dosages of Five Pesticides and a Polychlorinated Biphenyl on the Avoidance Response of Coturnix Quail Chicks, 6 Environ Pollut 21 (1974);

(g) Effects of PCBs on Pheasant Reproduction, Behavior and Survival, 35 J Wildl Manag 313 (1971); and

(h) Ulfstrand et al, Effect of PCB on Nocturnal Activity in Caged Robins, 231 Nature (London) 467 (1971);

(i) Tilson and Cabe, Studies on the Neurobehavioral Effects of Polybrominated Biphenyls in Rats, 320 Ann NY Acad Sci 325 (1979).

3. Good and Pensky, Halowax Acne ("Cable Rash"), 48 Archives of Dermatology and Syphilology, 43 Archives of Dermatology and Syphilology 251 (1943).

4. Seppäläinen and Vuojolahti, Reversible Nerve Lesions After Accidental Polychlorinated Biphenyl Exposure, 11 (2) Scan J Work Environ Health 91 (1985). This article indicates that the condition of the damaged nerves was greatly improved after six months.

5. Schecter, Contamination of an Office Building in Binghamton, New York by PCBs, Dioxins, Furans and Biphenylenes after an Electrical Panel and Electrical Transformer Incident, 12 (No.4/5) Chemosphere 669 (1983).

RELEVANT CASES

Probable Cause

Manko v. U.S., DC WMo, No. 79-1011-CV-W-9 (6/5/86)

Louis Manko sued the United States under the Swine Flu Act and the Federal Tort Claims Act, 28 USC 1346(b), after contracting Guillain-Barre Syndrome (GBS). Mr. Manko had received the swine flu vaccine, which, according to statistics developed by the Center for Disease Control, increased the risk of contracting GBS over three-fold during the period extending from the eleventh to the sixteenth weeks after vaccination.

Negligence was not an issue. All the plaintiff needed to show under Missouri law was that the preponderance of evidence indicated that the swine flu vaccine was a reasonable and probable cause of the onset of GBS. Although Mr. Manko's illness did not become acute until well after the statistically significant period of increased risk, the court found that his chances of contracting GBS had been more than doubled by the vaccine, and hence he was entitled to compensation. Witnesses for the plaintiff testified that a mild, "smoldering" form of the disease did set in during the period of highest risk.

It is interesting to note that the threshold for compensation based upon statistically increased risk was set at 100% increased probability; that is, at the point where risk doubles. The question concerning the degree of statistical certainty required for the 100% risk increase is, however, left open. If the study showed an 80% increased risk of contracting GBS with a 95% confidence level, and an increased risk of 110% with a 51% confidence level, the defendant would claim the 110% increased risk factor is statistically insignificant. But the plaintiff would say that there is a better than even chance that the risk of contracting the disease more than doubles with exposure to the vaccine, hence anyone who was vaccinated and contracted GBS probably became ill due to the vaccine.

Meloni v. Johns-Manville, DC NJ, No. 81-2542 (AET)

Plaintiff must show that he probably will contract cancer in order to avoid having his asbestos claim dismissed. The court ruled that a 43% probability is insufficient.

Ducre v. Mine Safety Appliances Co., DC La, No. 80-4338

The court ruled that "bodily injury" occurs with each inhalation of silica.

1 Toxics Law Reporter 564 (October 22, 1986)

PCB Cases

Unison PCB Separation Facility in Kentucky

Process Description: PCB Transformers at various locations are drained and then filled with a substance known as TF-1. The PCB contaminated transformer fluid which has been drained is drummed and shipped to an incinerator.

After several months, the fluid in the transformer, which by then will have dissolved the PCBs which were not initially removed, is itself drained. The fluid is called "TF-X" after it has been used to clean a transformer. TF-X is drummed and shipped to Henderson County, Kentucky for further processing.

In this process, transformers are repeatedly filled with TF-1, allowed to stand for months, and then drained again. Eventually they become clean enough to fill with a non PCB dielectric fluid and returned to service.

PCBs are separated from TF-X at the Unison Facility. The owners of the facility, Union Carbide, refuse to divulge the separation technique, although it almost certainly is distillation. Union Carbide also refused to reveal the identity of the solvent, TF-1. EPA has a complete description of the process, but it is being treated as a confidential business secret.

EPA Risk Assessment: EPA claims the Risk assessment considered potential releases due to fires, explosions, airplane crashes, earthquakes, flooding, tornadoes, and failure of the pollution control equipment. No details of the assessment are available, ostensibly to protect Union Carbide's trade secret.

Note: Page 2-6 of the risk assessment says that PCBs are not currently RCRA hazardous wastes, but they soon will be. The following appears on page 2-6: "PCBs are not as dangerous as other chemicals regulated under the Act and PCBs were already regulated under TSCA, PCBs were not named in the lists of hazardous chemicals."

The Bloomington Civil RICO Counts

Although federal judge Judge S. Hugh Dillin entered the consent decree resolving the conflict between Westinghouse and the plaintiffs in the Bloomington lawsuit, the City of Bloomington continued to pursue its case against Monsanto. In March of 1986, the City amended its complaint to include a count alleging violation of the

Racketeer Influenced and Corrupt Organizations Act,² and a count alleging conspiracy as defined by that act.

The City contends that for four years after the publication in 1966 of studies showing that PCBs had built up in the general environment, Monsanto attempted to obscure that fact by disseminating misleading information to its customers. They also imply that Monsanto concealed information concerning the toxicity of PCBs.

The City also contends that Monsanto used fraudulent test results which they obtained from a company called Industrial Bio-Test to convince the U.S. Food and Drug Administration, the U.S. EPA, its customers and the public that PCBs are not dangerous. They claim that by presenting these results, Monsanto delayed action against the manufacture, sale, and use of PCBs. A brief filed by the City states: "Although the EPA first considered regulation against PCBs in 1972 Nibsbati's lobbying efforts based on the false Industrial Bio-Test studies succeeded in delaying the imposition of a ban on PCBs until 1978. During the intervening six years Monsanto was able to sell an additional ten to twelve million pounds of its PCB products to Westinghouse, and many million pounds more to other customers."

Regarding the specifics of the Bloomington situation, the City contends that during the first 17 years of the operation of the Bloomington capacitor plant, they were unaware that PCBs were being used. The City claims that they were told only that such things as "capacitor insulating dielectric oils," or "Inerteen" were being used, and that they did not realize these materials were PCBs.

Bloomington, Indiana Personal Injury Suits

Bloomington, Indiana Property Damage Cases

People v. Todd Shipyards Corp., Calif SupCt (San Francisco), No.859923.

On June 19, 1986, Assistant San Francisco District Attorney Steven J. Castleman filed a complaint in San Francisco Superior Court against Todd Shipyards Corporation seeking an estimated \$11,000,000 in penalties for violations involving PCBs. The complaint alleges that Todd violated TSCA, the California Hazardous Waste Control Act, the state Health and Safety Code, and state right to know laws while improperly disposing of PCBs through an unlicensed hauler.

Brown v. SEPTA, DC EPA, No. 86-2229, 4/18/86 Burrell v. SEPTA, DC EPA, No. 86-2235, 4/18/86

Brown v. SEPTA is a personal injury and property damage class action suit against the Southeastern Pennsylvania Transportation Authority (SEPTA), National Railroad Passenger Corp. (AMTRAK), and Consolidated Rail Corp. (CON-RAIL). The plaintiffs charge the defendants with "reckless mishandling" of PCBs during the repair, servicing, storage, and operation of rail cars in the Paoli Rail Yard near Philadelphia.

U.S. v. SEPTA, DC EPA, No 86-1094, 2/25/86

EPA sued SEPTA, AMTRAK, and CON-RAIL under CERCLA to force a cleanup. EPA officials were prohibited from entering the railyard for testing in early 1986, but access was eventually granted. AMTRAK is trying to include the City of Philadelphia as defendant because the Philadelphia Transportation Co. had authority prior to the creation of SEPTA in 1968.

???? v. Robert E. Derecktor

Robert E. Dercktor was criminally indicted in May of 1986 for allegedly conspiring to conceal the existence of PCB materials from the EPA, and for releasing PCBs from a storage facility with notifying the proper authorities. Mr. Derecktor is the president of a Rhode Island shipyard. He was charged with 46 violation under the Clean Air Act, the Clean Water Act, CERCLA, and TSCA.

See May 21 U.S. EPA press release
See Toxics Law Reporter, 6-11-86

U.S. v. Sugarhouse Realty, DC EPA, No. 85-4829

On May 27, 1986, Judge Edward N. Cahn of the U.S. District Court for the Eastern District of Pennsylvania approved a consent decree between the U.S. EPA and Sugarhouse Realty providing for the cleanup PCBs originating from leaking electrical equipment. Sugarhouse Realty agreed to contract and pay for the removal of PCB contaminated equipment, buildings, water and soil at the site of the former Jack Frost Sugar House, a sugar refinery.

The settlement arose from a suit filed by EPA against Sugarhouse Realty and its president, William H. Thayer,

under TSCA and CERCLA. Under terms of the agreement, Sugarhouse must assume all liability related to the cleanup. It must post a \$250,000 performance bond, and can be fined \$750 per day in addition to other possible sanctions for non-compliance.

See Toxics Law Reporter, 6/18/86, page 48

Potomac Electric Power Co. v. Sachs, CA 4, Nos. 86-1572, 85 -1573

A Prince Georges County, Maryland grand jury is conducting a criminal investigation of parties allegedly responsible for PCB contamination in Beltsville, Maryland. The Potomac Electric Power Company is a major target of the investigation.

On April 16, the U.S. District Court for the District of Maryland held that TSCA does not pre-empt more stringent state laws regarding the disposal of PCBs. PEPCO asked the Court to rule in favor of pre-emption, and appealed the decision which was handed down. Maryland filed a cross appeal in U.S. Court of Appeals for the Fourth Circuit on June 16, 1986, agreeing with the lower court's ruling, but asking the Court of Appeals to abstain from ruling because, they claim, the federal courts lack jurisdiction to issue declaratory judgments.

The appeals court overturned the lower court's decision on October 17, 1986, ruling that the lower court should not have ruled regarding matters which were still before a local grand jury.

1 Toxics Law Reporter 66 (June 25, 1986).

Moreco Energy, Inc. v. Penberthy-Houdaille, Inc., DC Cill, No. 86020153.

Moreco Energy, Inc., a waste hauler and oil recycler sued Penberthy-Houdaille, Inc., a pump manufacturer, for allegedly delivering waste oil containing more than 50 ppm PCBs. Moreco filed suit under the citizen suit provision of TSCA, making this the first instance in which one citizen has attempted to sue another under that law. On June 16, 1986, the defendant moved to dismiss the case, alleging that citizen suits can only be initiated under TSCA to restrain ongoing violations.

1 Toxics Law Reporter 87 (July 2, 1986

Noble Oil Co., Inc v. EPA, US SupCt, No. 85-1650

On June 23, 1986, the Supreme Court declined to review a federal appeals court ruling upholding fines totaling \$40,000 which EPA assessed against Noble Oil Co, for improper storage and labeling of PCBs. Noble maintained that EPA failed to follow established procedures for taking samples.

1 Toxics Reporter 90 (July 2, 1986).

New York v. General Electric

The New York Department of Environmental Conservation (D.E.C.) and the General Electric Company signed a consent order in 1980 requiring General Electric to pay the entire cost of remedial action at three PCB contaminated sites, and contribute to the cost of cost of remedial action at four others.

On June 9, 1986, the D.E.C. announced its approval of the last phase of the cleanup, which involves clay capping, groundwater collection, and slurry walls. The sites, all in upstate New York, are in Nassau, Ft. Edward, Moreau, Stillwater and Kingsbury.

U.S. v. Ward, DC ENC, No. 83-63-CIV-5, 22 ERC 1235

Robert Earl Ward was sentenced to 30 months in prison and fined \$200,000 after being convicted in 1982 of violating the Toxic Substances Control Act by dumping PCBs along a North Carolina roadside in 1978. On June 12, 1986, Ward and the Ward Transformer Company agreed to pay \$2.95 million to the United States and North Carolina for the cost of the cleanup.

Brewer, et al. v. Monsanto Corp., et al., DC MTenn, No. 1:85-0071, 8/15/86.

Employees of an electronic component manufacturing plant in Waynesboro, Tennessee, along with their families, are pursuing a class action against two successive owners of the plant - Duracell and Emhart Industries - and the Monsanto Company. The suit does not seek compensation for injuries; it seeks to recover the amount Duracell and

Emhart would have had to spend on safety equipment and precautions, and the extra wages they would have had to pay, if the danger from PCBs had not been, so the suit contends, fraudulently concealed from the workers.

The court denied motions to dismiss based on the exclusivity provision of the Tennessee worker's compensation statute, ruling that the alleged fraud is an intentional tort and hence exempt from exclusivity.

1 Toxics Reporter 403 (September 17, 1986).

U.S. v. Commonwealth Edison, DC Nill, No. 84 C 1597

In 1984, the U.S. EPA filed suit against Commonwealth Edison under the Toxic Substances Control Act, alleging that capacitor discharges constitute improper disposal and that Commonwealth Edison was not properly cleaning up spill sites. Under a proposed consent agreement which the parties signed in September of 1986, Commonwealth Edison agreed to curb its use of PCB capacitors, perform routine inspections, take specified immediate response measures in case of accidental releases, and clean up more than 300 PCB contaminated sites.

Under the agreement, will use PCB capacitors only in generating plants and substations, and will physically remove such capacitors from poles by January 1, 1987. Cleanups in areas of high potential human contact will reduce contamination down to 5 ppm in soil, and 20 micrograms per square inch on hard surfaces. Contaminated playground equipment and outdoor furniture will be replaced.

1 Toxics Reporter 508 (October 8, 1986)

EPA Administrative Action Against Environmental Resources Management, Inc. and PCB Treatment, Inc.

Region VII of the U.S. EPA is seeking fines totaling almost 4 million dollars from two Kansas City, Missouri firms accused of improperly storing and disposing of PCB contaminated chemicals and equipment.

Geneva Industries

Along with Monsanto, Geneva Industries of Houston, Texas, is the only other company known to have manufactured PCBs on a commercial scale in the United States. It has

been estimated that Geneva Industries manufactured 1 million pounds of PCBs, considerably less than the estimated 1.3 billion pounds produced by Monsanto.

According to the EPA, Geneva began PCB production in 1972, one year after Monsanto stopped selling to customers who did not manufacture transformers and capacitors.¹

1. Superfund Program Project Update, Geneva Industries, Houston, Texas; September, 1986.

Kentucky vs. Rockwell International

The Kentucky Department for Environmental Protection cited Rockwell International on June 6, 1985, for discharging PCBs from their plant in Russellville into the waterways of Kentucky. The discharge moves five miles along a ditch and stream before it enters the Mud River. Rockwell has agreed to clean up part of its plant site, but litigation to force further cleanup is still pending. Private suits alleging property damage and personal injury have also been filed.

The ENSCO PCB Incinerator

The Ensco facility is a hazardous waste incinerator in El Dorado, Arkansas. Blood tests were arranged by the owners of the ENSCO incinerator, and performed upon incinerator workers. These tests showed elevated PCB levels.

NIOSH recently took blood samples from ENSCO workers in order to test for PCBs and possibly dibenzofurans and dioxins. The results are expected in late May. The official NIOSH report could take several more months for preparation and review.

The union representative at ENSCO is:

Scott Harper
Union Representative
Oil, Chemical, and Atomic Workers Union
El Dorado, Arkansas

The NIOSH health evaluation study number is HE 86519

The NIOSH contact person is:

Jay Bainbridge
Hazard Evaluation Division
NIOSH

The Arkansas Health Department contact is:

Rob Robinette
Arkansas Department of Health
4815 West Markham St.

CANCER

Until recently, it has been widely accepted that there is no conclusive evidence supporting the suspicion that exposure to PCBs increases human risk of contracting cancer.¹ Some studies, including one conducted by NIOSH,² indicated decreased overall cancer among exposed workers. There is even a strain of rat in which PCBs inhibit the growth of a certain type of cancer,³ and a type of fish for which PCBs provide protection against aflatoxin induced cancer.⁴

Studies suggesting that PCBs are not carcinogenic, however, are rare exceptions. The evidence linking PCBs to liver cancer in experimental animals is overwhelming,⁵ and the evidence linking PCBs to cancer in humans is growing. As the years pass, and increasing numbers of people exposed to PCBs die, it becomes increasingly likely that statistically significant evidence will link PCBs with some forms of cancer.

Re-examination of a cohort of previously studied electrical equipment production workers recently indicated a significant excess of deaths due to liver cancer.⁶ Statistically significant excessive liver cancer deaths have also been observed among male Yusho victims.⁷ Statistically significant elevations in skin cancer have been reported.⁸ Statistically significant excess cancer was found among farmers with PCB lined grain silos,⁹ and among a cohort of capacitor manufacturing production workers.¹⁰

The question of threshold dose for carcinogenesis is frequently raised by industry representatives. They assert that gross physical damage to the liver in the high dosage animal studies might play an important role in the onset of cancer. Industry spokesmen also assert that PCBs are not mutagens and are, therefore, not cancer initiators. They argue that PCBs are cancer promoters, and as such, have a threshold below which they have no effect. The industry position is that humans have not been exposed to PCBs above the cancer promoting threshold.¹⁰

1. Dr. Stephen Safe (Safe, Polychlorinated Biphenyls (PCBs) and Polybrominated Biphenyls (PBB's): Biochemistry, Toxicology, and Mechanism, 13 CRC Critical Reviews in Toxicology 319 [1985]) states: "The three major PCB-exposed populations, environmental, accidental, and industrial/occupational, have not yet shown any significant increases in cancer within these three groups."

Also, the following is from the pamphlet titled, "PCBs: Is the Cure Worth the Cost?" (obtained from The American Council on Science and Health 47 Maple Street Summit, NJ 07901):

"The most extensive occupational studies of long-term exposure to PCBs involve electrical equipment workers. Aside from occasional skin irritations that

disappeared quickly, no significant adverse health effects have been reported among workers in the electrical industry (page 13).

NIOSH (the National Institute for Occupational Safety and Health) studied maintenance workers exposed to PCBs at two utility companies. While their blood PCB levels were much higher than the national background levels (12 to 298 parts per billion [ppb] vs. 10 to 20 ppb), they did not exhibit any all health effects, not even chloracne. From

page 9, referring to: Smith, et al, "Cross Sectional Survey of Two Groups of Workers Occupationally Exposed to Polychlorinated Biphenyls (PCBs) in the Maintenance, Repair, and Overhaul of Electrical Transformers" (1981).

General Electric has studied the health of 194 workers heavily exposed to PCBs for an average of 15 years, some for as long as 35 years. The first report included studies done on the workers through 1976, and showed no ill effects. A 1979 re-evaluation again failed to produce evidence of ill health among these workers. From

page 9, referring to: R.W. Lawton, M.R. Ross and J. Feingold, G.E. Draft Report on PCBs (1981).

2. Industrial sources frequently cite the following NIOSH study which showed *decreased* cancer among 2,500 workers in two separate capacitor manufacturing plants: Brown and Jones, Mortality and Industrial Hygiene Study of Industrial Workers Exposed to Polychlorinated Biphenyls (PCBs), 36 Arch Environ Health 120 (1981). However, the same data have been interpreted to show PCB exposure put the workers at greater risk of contracting cancer (in Haley v. Monsanto Chemical Co., Mich, Huron County Circuit Court, No. 77-00-2593-NP, May 31, 1984).

3. Kirkuliet and Kimeldorf, Antitumor Activity of Polychlorinated Biphenyl Mixture, Aroclor 1254, in Rats Innoculated with Walker 256 Carcinosarcoma Cells, J. Natl Cancer Inst 951 (1977).

4. Bailey et al., The Sensitivity of Rainbow Trout and Other Fish to Carcinogens, 15(4) Drug Metab Rev 725, (1984).

5. The following studies link PCBs with cancer in animals:

Kimura and Baba, Neoplastic Changes in the Rat Liver Induced by Polychlorinated Biphenyls, 105 Gann 64 (1973).

Allen and Norback, Carcinogenic Potential of the Polychlorinated Biphenyls, in Origins of Human Cancer, p. 173 (1977).

Nagasake et al., Analysis of Various Factors on Liver Carcinogenesis in Mice Induced by Benzene Hexachloride (BHC) and Technical Polychlorinated Biphenyls (PCBs), 25 J Nara Med Assoc 635 (1974).

Kimbrough and Linder, Induction of Adenofibrosis and Hepatomas in the Liver of BALB/cJ Mice by Polychlorinated Biphenyls (Aroclor 1254), 53 J Natl Cancer Inst 547 (1974).

Ito et al, Histopathological Studies on Liver Tumorigenesis in Rats Treated with Polychlorinated Biphenyls, 65 Bann 545 (1974).

Kimbrough et al., Induction of Liver Tumors in Sherman Strain Female Rats by Polychlorinated Biphenyl Aroclor 1260, 55 J Natl Cancer Inst 1453 (1975).

Aishizumi, Enhancement of Diethylnitrosamine Hepatocarcinogenesis in Rats Exposed to Polychlorinated Biphenyls or Phenobarbital, 2 Cancer Lett 11 (1976).

Preson et al., Promoting Effects of Polychlorinated Biphenyls (Aroclor 1254) and Polychlorinated Dibenzofuran-free Aroclor 1254 on Diethylnitrosamine-Induced Tumorigenesis in the Rat, 66 J Natl Cancer Inst 509 (1981).

Pereira et al., Promotion by Polychlorinated Biphenyls of Enzyme Altered Foci in Rat liver, 15 Cancer Lett 185 (1982).

Deml and Oesterle, Sex-Dependent Promoting Effect of Polychlorinated Biphenyls on Enzyme-Altered Islands Induced by Diethylnitrosamine in Rat Liver, 3 Carcinogenesis 1449, (1982).

6. Brown, Mortality of Workers Exposed to Polychlorinated Biphenyls - An Update, Centers for Disease Control,

Department of Health and Human Services, Cincinnati, Ohio (1985).

7. Amano et al., Statistical Observations About the Causes of the Death of Patients With Oil Poisoning, 39 Japan Hygiene (1984). Translated for the U.S. EPA by SCITRAN, Santa Barbara, California (1984).

8. Schecter and Tiernan, Occupational Exposure to Polychlorinated Dioxins, Polychlorinated Furans, Polychlorinated Biphenyls, and Biphenylenes After an Electrical Panel and Transformer Accident in an Office Building in Binghamton, NY, 60 Environ Health Perspect 305 (1985). Also: Bahn et al, Melanoma After Exposure to PCBs, 295 New Engl J Med 450 (1976). The details of the Bahn report are unpublished.

9. Humphrey, Population Studies of PCBs in Michigan Residents, page 299 of PCBs: Human and Environmental Hazards, (1983).

10. Bertazzi et al., Mortality Study of Male and Female Workers Exposed to PCBs. Presented at the International Symposium on Prevention of Occupational Cancer, April, 1981, Helsinki, Finland.

Evaluation of the Cancer Risk

Monsanto's William R. Gaffey,¹ used the following requirements for acceptable epidemiological evidence, based on work by Doll,² to demonstrate his contention that PCBs have not been shown to be human carcinogens:

Positive associations in groups of individuals with known exposure (case-control or cohort studies) that

- 1) are not explained by bias in recording or detection,
- 2) are not explained by confounding,
- 3) are not explained by chance,
- 4) vary appropriately with dose,
- 5) vary appropriately with period of exposure,
- 6) are observed repeatedly in different circumstances.

Mr. Gaffey dismisses an unpublished study linking PCBs with melanoma because there could have been confounding from exposure to other, unknown compounds.³

He dismisses the Zack and Musch study which found 4 lung cancers among 89 Monsanto workers because the excess incidence is "statistically insignificant" and the workers were exposed to other chemicals.

Mr. Gaffey dismisses the excess deaths from liver cancer found by Brown and Jones⁴ because there is an inverse relationship between duration of latency of exposure. The excess rectal cancer deaths are said to be at least partly attributable to high background levels in the area.

The excess digestive, lymphatic, and liver cancer deaths reported by Bertazzi et al., are dismissed because only 27 deaths (for all causes) were involved and because of a "failure to agree on any particular pattern of mortality."

Mr. Gaffey appeared to be unaware of the Michigan Silo Farmers cohort study reported by Humphrey in which the rate of death from cancer was ten times the national average.⁵ He likewise seemed unaware of the excess cancer deaths among Yusho victims.⁶

In his critiques of the epidemiological studies, Mr. Gaffey seems to believe that exposure to other chemicals is sufficient to exonerate PCBs as a possible causal agent. But to the extent that PCBs are cancer promoters, and the experimental evidence indicates that they are,⁷ other chemicals would have to be present for there to be any effect. It is doubtful that any court would hold that cancer promoters cannot be the proximate cause of the onset of cancer. Industry spokesmen maintain that cancer promoters have thresholds below which they have no effect, and that humans have seldom or never been exposed to amounts large enough to have any effect.

Findings of diverse types of cancer at various locations would be consistent with PCBs being cancer promoters. The short latency times might likewise be explained. (Of fifty workers being monitored because they suspect they were exposed to PCB combustion byproducts during and after a fire in 1981, three were known to have skin cancer in 1984.■)

Many researchers seem to be unwilling to draw conclusions before more mortality data are available. All agree that PCB exposure and cancer might be linked. It would be interesting to survey the experts and ask, instead of: Is there compelling evidence linking PCBs to human cancer?; rather: Does the preponderance of the evidence implicate PCBs as a proximate cause of the onset of human cancer?

1. Gaffey, The Epidemiology of PCBs, page 297 of PCBs: Human and Environmental Hazards (1983).
2. Doll, Relevance of Epidemiology to Policies for the Prevention of Cancer, 23 J Occup Med 601 (1981).
3. Bahn et al., Melanoma After Exposure to PCBs, 295 N Engl J Med 450 (1976).
4. Brown and Jones, Mortality and Industrial Study of Workers Exposed to Polychlorinated Biphenyls, 36 Arch Environ Health 120 (1981).
5. Humphery (Population Studies of PCBs in Michigan Residents, page 299 of PCBs: Human and Environmental Hazards, 1983) reports significantly elevated PCB burdens and frequency of cancer among farmers with PCB treated silos. Of 183 participants in the study, 0.3% were expected to have cancer based on data from the national

Surveillance, Epidemiology, and End Results Program (SEER). The cancer rate observed was 3.3%.

6. Urabe et al, Present State of Yusho Patients, 320 Ann NY Acad Sci 273 (1979).

7. R.A. Neal, Mechanisms of the Biological Effects of PCBs, Polychlorinated Dibenzo-p-dioxins and Polychlorinated Dibenzofurans in Experimental Animals, 60 Environ Health Perspect 41 (1985): "There is evidence that PCBs may be causing increases in rodent liver tumors largely by mechanisms which do not involve permanent changes in the phenotypic expression of hepatocytes. In other words, they may be acting as promoters." (citing: (a) Preston et al., Promoting Effects of Polychlorinated Biphenyls (Aroclor 1254) and Polychlorinated Dibenzofuran-Free Aroclor 1254 on Diethylnitrosamine-Induced Tumorigenesis in the Rat, 66 J Natl Cancer Inst 509 (1981); (b) Oesterle and Deml, Promoting Effect of Polychlorinated Biphenyls on Development of Enzyme Altered Islands in Livers of Weanling and Adult Rats, 105 J Cancer Res. Clin Oncol 141 (1983); and (c) Kimura et al., Polychlorinated biphenyl(s) as a Promotor in Experimental Hepatocarcinogenesis in Rats, 87 Z Krebsforsch Klin Onkol 257 [1976]). Another article indicating PCBs might act as promoters is: Tsushimoto, Inhibition of Intercellular Communication by Various Congeners of Polybrominated Biphenyl and Polychlorinated Biphenyl, page 241 of PCBs, Human and Environmental Hazards, 1983. PCBs might promote cancer by inducing enzymes which change other chemicals into carcinogens, or by suppressing the body's immune response, as is suggested by Talcott et al., The effect of Lead and Polychlorinated Biphenyl Exposure on Rat Natural Killer Cell Cytotoxicity, 7(2) Int J Immunopharmacol 255 (1985).

8. Schechter and Tiernan, Occupational Exposure to Polychlorinated Dioxins, Polychlorinated Furans, Polychlorinated Biphenyls, and Biphenylenes After an Electrical Panel and Transformer Accident in an Office Building in Binghamton, NY., 60 Environ Health Perspect 305 (1985).

HEART DISEASE

There is some epidemiological data linking PCB exposure with high blood pressure.¹ There is a great deal of data linking PCB exposure with elevated serum triglyceride, elevated serum cholesterol, and diminished serum high density lipoprotein-cholesterol. All three of these blood constituent tendencies are associated with increased risk of developing coronary artery disease.

Hydropericardium (water buildup in the pericardial sac) and enlargement of the heart have been induced in experimental animals by PCBs.² The PCB contaminated rice oil responsible for the Yusho catastrophe reportedly caused hydrocardium in chicks.³

Two deceased Yusho victims were found by Kikuchi⁴ to have "severe myocardial lesions with small fibrotic and necrotic foci with basophilic myofibrillar degeneration, together with a large amount of PCBs depositing in the heart." The author speculates that hormonal disturbances might be involved. A statistically significant excess in mortality among female Yusho victims due to ischemic heart disease has been reported.⁵

1. Kreiss et al., Association of blood pressure and polychlorinated biphenyl levels, 245 JAMA 2505.
2. McCune et al., Hydropericardium and Ascites in Chicks Fed a Chlorinated Hydrocarbon, 41 Poult Sci 295 (1962); and Vos and Koeman, Comparative Toxicologic Study with Polychlorinated Biphenyls in Chickens with Special Reference to Porphyria, Edema Formation, Liver Necrosis and Tissue Residues, 17 Toxicol Appl Pharmacol 656 (1970).
3. Kikuchi, Autopsy of Patients With Yusho, 5 Am J Ind Med 19 (1984) citing: Goto and Higuchi, The Symptomology of Yusho (Chlorobiphenyls Poisoning) in Dermatology, 60 Fukuoka Igaku Zasshi 409 (Japanese, 1969).
4. Kikuchi, Autopsy of Patients With Yusho, 5 Am J Ind Med 19 (1984).
5. Amano et al., Statistical Observations About the Causes of the Death of Patients With Oil Poisoning, 39 Japan Hygiene (1984). Translated for the U.S. EPA by SCITRAN, Santa Barbara, California (1984).

VITAMIN A AND THYROID HORMONES

Introduction

Poisons and therapeutic drugs induce illness or provide relief by first interacting with the body's natural chemicals. These interactions typically set off a complex chain reaction which culminates in a change in the body's chemical balance, either in the bloodstream or in specific organs or types of cells. Overt effects of poisons and drugs are a consequence of these often little understood chemical events.

Numerous adverse health consequences of PCB exposure have been identified. Interactions between PCBs and natural molecules inside the body have also been identified. In addition to this information at the two extremes - chemical interaction and the onset of symptoms - there is also information about intermediate biochemical abnormalities and imbalances which can be induced by exposure to PCBs. Some of these abnormalities and imbalances are part of the chain linking molecular interactions between PCBs and the body's chemicals with the appearance of the symptoms of disease.

As experimental scientists continue to generate information, medical practitioners will be increasingly able to link the biochemical changes induced by PCBs with specific disorders and diseases. This increased knowledge could help in determining the cause of disease in specific patients, and in the treatment of people whose maladies are induced or exacerbated by exposure to PCBs and related compounds.

PCB Interference With Normal Thyroid Function

The thyroid gland, located in the neck, produces the thyroid hormones triiodothyronine (T_3) and thyroxine (T_4). T_4 molecules have 4 iodine atoms, T_3 molecules have 3 iodine molecules. Iodine deficiency leads to visible swelling of the thyroid gland - a condition called "goiter."

Triiodothyronine and thyroxine are interconvertible inside the body. The prevalent form in the bloodstream is thyroxine; the prevalent form inside cells is triiodothyronine. Together, the thyroid hormones are involved in a complex array of life sustaining processes. They play a critical role in regulating cell growth and metabolism, and in the production of protein. Thyroid hormones are produced only in the thyroid gland, but they are necessary for the normal functioning of all the organs of the body.

Water insoluble compounds can be transported through the bloodstream and through cell cytosol² by attaching to soluble proteins. Thyroxine is not soluble in water; yet it

must be transported from the thyroid gland, through the bloodstream, across cell membranes, and through the cytosol into cell nuclei. It does this by attaching to water soluble proteins in the blood, crossing the cell membrane in the free (unbound) form, and attaching to water soluble protein inside cells. At any give instant, about 0.02% of thyroxin and 0.3% of triiodo-thyronine are in the free form. Only the free form is biologically active, so drastic effects can be felt when the amounts of the free forms vary - even when total thyroid hormone levels remain constant.

Thyroxin binding prealbumin is a blood plasma protein which transports thyroxin from the thyroid gland, through the bloodstream, and to the body's cells. The modern name for thyroxin binding prealbumin is "transthyretin (TTR)." Other proteins which transport thyroxin are called "thyroxin binding globulin" and "albumin."

The protein which carries vitamin A through the blood is called retinol-binding protein (RBP). Prealbumin and RBP bind together in blood plasma to form the transport system for thyroxin and vitamin A.² By means of TTR, thyroxin and vitamin A travel together through the bloodstream.

TTR is over 100 times the size of thyroxin. Thyroxin binds to only two specific sites on this molecule. These sites are called thyroxin binding sites.

PCB molecules are approximately the same size and shape as thyroxin. Many PCB congeners - particularly those with chlorine in all four meta positions - have 3 dimensional contours which are similar to thyroxin, and these congeners readily bind to the thyroxin binding sites of TTR.³ 3,3',4,4'-tetrachloro-biphenyl has chlorine atoms in only two meta postitions, but it binds to the thyroxin binding sites of TTR within hours of being introduced into the body, after hydroxyl (O-H) groups attach to the meta positions through enzyme induced metabolism.⁴

By binding to TTR, PCBs and related compounds can interfere with the transport of thyroxin and vitamin A. In addition, PCBs may interfere with normal body function by attaching to thyroxin binding sites in the body's tissues.

Some PCBs and related compounds cause levels of thyroid hormones to decrease. Perhaps because they act as substitutes for these hormones, PCBs trick the body into thinking it already has enough, and therefore shutting down

thyroxin production. By limiting the amount and availability of thyroxin, PCBs induce symptoms of hypothyroidism. The production of abnormal skin, hair, and nail proteins which is observed in cases of exposure to PCBs and related compounds is also observed in hypothyroidism.⁵

To the extent that some PCB congeners act as thyroxin substitutes, they can cause the body to react as though it had too much thyroxin. PCBs could do this directly, by acting as thyroxin substitutes inside the body's cells, or indirectly by displacing thyroxin from blood serum transport proteins. It has been suggested that the PCB induced wasting syndrome - the syndrome in which experimental animals waste away despite normal eating - may result from excessive free thyroxin inside the cells.⁶

The disease associated with excessive thyroid hormone production is called hyperthyroidism. Dioxin causes hyperthyroidism in hamsters,⁷ so it is reasonable to assume that some of the PCB congeners also have this effect. It has been suggested that PCB poisoning induces symptoms of both hypo- and hyperthyroidism.⁸

No information has been made available concerning the extent to which physicians are using the research discussed in this report to treat the long term debilitation which apparently afflicts many of those who have experienced past massive exposure to PCBs and related compounds.

1. The small components of the body called "cells" have an outer membrane which contains the cells' fluid, its nucleus, and other components. The fluid inside of cells is called "cytosol" or "cytosolic fluid."

2. Brouwer and van der Berg, Binding of a Metabolite of 3,4,3'4'-tetrachlorobiphenyl to transthyretin Reduces Serum vitamin A Transport by Inhibiting the Formation of the Protein Complex Carrying Both Retinol and Thyroxin, 85 Toxicol Appl Pharm 301 (1986).

3. Rickenbacher et al., Structurally Specific Binding of Halogenated Biphenyls to Thyroxine Transport Protein, 29 Med Chem 641 (1986).

4. Brouwer and van der Berg, Binding of a Metabolite of 3,4,3'4'-Tetrachlorobiphenyl to Transthyretin Reduces Serum vitamin A Transport by Inhibiting the Formation of the Protein Complex Carrying Both Retinol and Thyroxin, 85 Toxicol Appl Pharm 301 (1986).

5. McKinney et al., 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) as a Potent and Persistent Thyroxine Agonist: A Mechanistic Model for Toxicity Based on Molecular Reactivity, 61 Environ Health Perspec 49 (1985), citing: Dratman, The Mechanism of Thyroxine Action, In: Li (ed), Thyroid Hormones, p. 442 (1978). Also citing: Kimbrough, Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Compounds, p. 406 (1980).
6. Brouwer and van der Berg, Binding of a Metabolite of 3,4,3'4'-Tetrachlorobiphenyl to Transthyretin Reduces Serum vitamin A Transport by Inhibiting the Formation of the Protein Complex Carrying Both Retinol and Thyroxine, 85 Toxicol Appl Pharm 301 (1986).
7. Lu et al., Toxicity and Evidence for Metabolic Alterations in 2,3,7,8-Tetrachlorodibenzo-p-dioxin-treated Guinea Pigs Fed by Total Parenteral Nutrition, unpublished paper based on a presentation at the 24th Annual Meeting of the Society of Toxicology. Citing Gasiewicz et al., Temporal and Dose-related Characteristics of Biochemical and Morphological Alterations in the Hamster, in the proceedings of the 5th International Symposium on Chlorinated Dioxins and Related Compounds, Bayreuth, Federal Republic of Germany (1985).
8. Rickenbacher et al., Structurally Specific Binding of Halogenated Biphenyls to Thyroxine Transport Protein, 29 Med Chem 641 (1986). On page 647, the authors state, regarding PCBs and related compounds, "... a complex overlay of hyper- and hypothyroid effects could be produced that may result in toxicity."

Immune System Impairment

Aside from transporting thyroxin, transthyretin (TTR) is apparently also involved with the function of the thymus.¹ The thymus is essential for normal immune system functioning. It produces molecules called "T cells" which attack foreign material in the body.

PCBs are known to cause thymus atrophy and suppression of T-cell activity.² A connection between immunological alterations and PCB interference with TTR has been suggested.³

1. Burton et al., Thymic hormone-like restoration by Human Preambulin of Azathioprine Sensitivity of Spleen Cells from Thymectomized Mice, 75 Proc Natl Acad Sci USA 823 (1978).
2. See the report on the immune system.
3. Brouwer and van der Berg, Binding of a Metabolite of 3,4,3',4'-Tetrachlorobiphenyl to Transthyretin Reduces Serum vitamin A Transport by Inhibiting the Formation of the Protein Complex Carrying Both Retinol and Thyroxin, 85 Toxicol Appl Pharm 301 (1986).

PCB Induced Hypovitaminosis A

Chloracne and hyperkeratosis are symptoms of PCB poisoning which resemble symptoms of vitamin A deficiency.¹ Other symptoms common to PCB poisoning and vitamin A deficiency include immunosuppression, neurological symptoms, and reproductive abnormalities.² It has been shown that PCBs and related compounds can reduce vitamin A levels in the blood and livers of monkeys and rodents.³

One research group has shown that vitamin A depletion in the liver is not well correlated with PCB induced enzyme induction.⁴ This group felt that vitamin A was being destroyed in the liver by chemical reactions which do not involve enzymes.

Another group's research suggests that the depletion of vitamin A in the liver is due to interference with the vitamin A transport system by PCBs.⁵ Retinol binding protein (RBP) was found to be greatly reduced in rats treated with 3,3',4,4'-tetrachlorobiphenyl. This appeared to be related to the fact that the tetrachlorobiphenyl became attached to either the thyroxin binding sites or the

RBP binding sites of TTR, preventing the formation of the RBP-TTR complex. RBP molecules are small enough to pass into the urine, so the inability to form the RBP-TTR complex could lead to RBP depletion.

1. Brouwer et al., Transthyretin (Prealbumin) Binding of PCBs, a Model for the Mechanism of Interference with Vitamin A and Thyroid Hormone Metabolism, 15 Chemosphere 1699 (1986); citing: Kimbrough, 2 Crit Rev Toxicol 445 (1974), and Van Putten et al, page 155 in: Infections and immunosuppression in subhuman primates, Mundsgaard, Copenhagen (1970).

2. Hakansson et al, The Effect of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) on the Distribution and Excretion of the Endogenous Pool of Vitamin A in Rats with Low Liver Vitamin A Stores, 15 Chemosphere 1715 (1986).

3. Brouwer et al., Transthyretin (Prealbumin) Binding of PCBs, a Model for the Mechanism of Interference with Vitamin A and Thyroid Hormone Metabolism, 15 Chemosphere 1699 (1986); citing: McConnell et al, 29 Lab Sci 666 (1979); Thunberg et al, 45 M Arch Toxicol 273 (1980); Innami et al, 20 J Nutr Sci Vitaminol 363 (1974); Darjono et al, 71 Toxicol Appl Pharmacol 184 (1983); Brouwer and van der Berg, Toxicol Appl Pharmacol 204 (1984); and Brouwer et al., 78 Toxicol Appl Pharmacol 180 (1985).

4. Azais and Pascal, Effects of Congeneric Polychlorinated Biphenyls on Liver and Kidney Retinoid Levels, 15 Chemosphere 1905 (1986).

5. Brouwer et al., Transthyretin (Prealbumin) Binding of PCBs, a Model for the Mechanism of Interference with Vitamin A and Thyroid Hormone Metabolism, 15 Chemosphere 1699 (1986).

BIOCHEMICAL OVERVIEW

PCB and related compounds cause or contribute to bad health by first interacting with the body on the molecular level. Molecular activity can then lead to gross changes such as liver enlargement, joint inflammation, nerve damage, or cancer. Research continues to generate information concerning the distribution of PCBs among various organs, tissues, and cell components; on the varying tendencies of PCB congeners to bind to proteins involved in the regulation of the immune and endocrine (hormone) systems; and on the addition of oxygen or sulfur to PCBs, transforming them into potentially toxic compounds known as PCB "metabolites."

Invasion of Cells

Just as fat accumulates on the surface of soup, or oil separates from water, so fat soluble chemicals in the human body avoid the water of the bloodstream. Like paints, all natural compounds, drugs, and pollutants can be classified according to whether they dissolve in water or organic solvents. With rare exceptions, chemicals which are water soluble have strong aversions to organic solvents, and chemicals which dissolve in organic solvents have strong aversions to water. Compounds which dissolve in water are called "hydrophilic;" those which dissolve in organic solvents are called "hydrophobic."

Lipids are relatively small hydrophobic molecules. They include fats, oils, and waxes. Glycerides and cholesterol are examples of lipids. PCBs are hydrophobic, so PCB molecules which enter the body quickly find their way to fatty portions of the so called "lipo-proteins" which circulate in the blood and transport hydrophobic compounds across cell membranes and into the cell cytoplasm, into fat deposits (adipose tissue), and onto the inside surfaces of cell membranes.

On the molecular level, the body functions largely inside the individual cells. There are many different kinds of cells serving many different functions. One thing they have in common is the ability to convert nutrients into energy. Cells have been called the body's "little factories." The form and structural integrity of cells is maintained primarily by compounds called "glycerides."

Glycerides are long molecules which, while hydrophobic overall, are hydrophilic at one end. Glycerides form the membranes which enclose the fluid (cytoplasm) inside the body's cells, and form a barrier keeping unwanted compounds in the blood stream from entering cells and interfering with life processes. Proteins which extend from outside the cell to the inside (cytoplasm) are interspersed among the glycerides. These proteins allow chemicals outside the cell to communicate with chemicals inside the cell by attaching to either end of the proteins, or the assist

chemicals in entering or leaving the cell. The system which transports vitamin A and thyroid hormone from the bloodstream to the inside of cells may be involved in transporting PCBs into cells. It is known that PCBs have a strong affinity for thyroxin binding prealbumin (TBP) in the blood.¹

The inside surface of cell membranes consists of the hydrophobic chains of glycerides; steroids such as sex hormones, vitamin A, and cholesterol; and many other hydrophobic compounds. Hydrophobic compounds move easily around the inner surface of the cell membrane, but rarely cross it to enter the bloodstream or leave unassisted to enter the cytoplasm.

Many cell membranes have a network of smaller membranes called the "endoplasmic reticulum." Fragments of this network constitute much of the so called "microsomal fraction" of cells: the fraction of homogenized cells which goes to the bottom of a test tube in a high-speed centrifuge. Under an electron microscope, the reticulum can be seen to be either smooth or rough. One of the first manifestations of PCB exposure is an increase in the amount of (a "proliferation" of) the smooth endoplasmic reticulum of the liver. This proliferation is believed to be associated with increased production of certain enzymes, called "microsomal mixed function oxydases (MFO's)," which reside in the endoplasmic reticulum.

Some of the 209 individual PCB congeners are very potent inducers of MFO's. The most toxic PCBs are the most powerful inducers. The abilities of PCBs to induce MFO's is directly related to their abilities to bind to mobile proteins in the cytoplasm called "Ah-receptor proteins." Ah-receptor proteins apparently transport PCBs from the cell membrane into the cell nucleus where the Ah-receptor protein/PCB complex stimulates the production of MFO's. The Ah-receptor mobilized PCBs may do more than merely stimulate MFO production. Evidence that they play a larger role includes the fact that some compounds which are less toxic than PCBs are also potent MFO inducers.

Transformation of Liver Cells

During the days and weeks following exposure to PCBs, liver cells increase in size and change in appearance. The endoplasmic reticulum proliferates as the number of enzyme molecules in the cell membrane increases. As is explained in the report dealing with enzymes, steroid hormones, and Vitamin A, these transformations cause fundamental changes

in the ways liver cells function.

Also see the reports on enzymes and metabolites

LUNG DISEASE

PCBs¹ and PCB metabolites² accumulate in the lungs of mice, and presumably also in the lungs of humans. They have been found in the sputum of exposure victims.

Capacitor manufacturing workers were shown to suffer from reduced vital capacity to a degree comparable to that of asbestos insulation workers,³ and Yusho victims were plagued with chronic lung infections. Autopsies of the victims showed a distinct pattern of pathological change.⁴

1. Brandt, Tissue Localization of Polychlorinated Biphenyls Chemical Structure Related to Pattern of Distribution, 40 Acta Pharmacol Toxicol (Suppl 2) 62 (1977).

2. Jansson, Transformation of Some Organochlorine Compounds in the Environment Studied by Gas Chromatography and Mass Spectrometry, Report from the National Swedish Environment Protection Board, Special Analytical Laboratory, Wallenberg Laboratory, University of Stockholm (1978).

3. Warshaw et al., Decrease in Vital Capacity in PCB-exposed Workers in a Capacitor Manufacturing Facility, 320 Ann NY Acad Sci 277 (1979)

4. Shigematsu et al., Respiratory Involvement in Polychlorinated Biphenyls Poisoning, 16 Environ Res 92 (1978)

SKIN DISEASE

Skin reactions are characteristic symptoms of exposure to PCBs and related compounds. Even Industry spokesmen and apologists concede the existence of "reversible" skin conditions.¹

These conditions range from the temporary rashes reported by at least one person involved in cleaning up the aftermath of the 1981 Binghamton, New York, office building fire, to the horrible chloracne which broke out among the victims of Yusho and Yu-cheng.

Dermal reactions have also been reported by experimental research teams. Rabbit ears develop lesions² from exposure as do hairless mice.³ Some experimenters have poisoned monkeys and photographed their faces.⁴

Malignant melanomas were reported in a letter to the New England Journal of Medicine dated August 19, 1976, and signed by six researchers affiliated with the University of Pennsylvania. Among 31 heavily exposed workers, two malignant melanomas appeared. There is only 1 chance in 25 that a single malignant melanoma would be found in a group that size. The same letter indicates that among another group of 41 less exposed workers, one malignant melanoma was diagnosed. The authors suggest that there may be connection between this rare form of cancer and other skin problems known to be caused by PCB,s. If PCBs were involved in this outbreak of cancer, they probably acted in combination with another or other chemicals.

By 1984, there were three confirmed cases of malignant melanoma among a group of 50 people who were being monitored after the 1981 Binghamton, New York, office building fire. This group of 50 joined a voluntary monitoring program because they believed they had been exposed to PCBs and PCB combustion products.

1. The Following is from the American Council of Health and Science pamphlet titled, "PCBs: Is the cure worth the cost?":

The most extensive occupational studies of long-term exposure to PCBs involve electrical equipment workers. Aside from occasional skin irritations that disappeared quickly, no significant adverse health effects have been reported among workers in the electrical industry (page 13).

2. Vos and Beems, Dermal Toxicity Studies of Technical Polychlorinated Biphenyls and Fractions Thereof in Rabbits, 19 Toxic Appl Pharmacol 617 (1971).

3. Puhvel et al., Hairless Mice as Models for Chloracne, 64 Toxic Appl Pharmacol 492 (1982).

4. (a) Allen et al., Residual Effects of Short-term, Low-

Level Exposure on Nonhuman Primates to Polychlorinated Biphenyls, 30 Toxicol Appl Pharmacol 440, (1974); (b) Altman et al, A Spontaneous Outbreak of Polychlorinated Biphenyl (PCB) Toxicity in Rhesus Monkeys (Macaca Mulatta): Toxicopathology, 29 Lab Anim Sci 661 (1979); and concerning the experimental poisoning of fetal and infant monkeys: (c) Allen et al., Comparative Toxicology of Chlorinated Compounds on Mammalian Species, 7 Pharmacol Ther 513 (1979); and (d) Allen and Barsotti, The Effects of Transplacental and Mammary Movement of PCBs on Infant Rhesus Monkeys, 6 Toxicology 331 (1976).

EYE PROBLEMS

Hypersecretion of the Meibomian glands, swelling of the upper eyelids, and hyperpigmentation of the conjunctivae were prevalent among Yusho and Yu-cheng victims. Burning of the eyes has been reported by factory workers and emergency response personnel involved in PCB fires; and about 10% of the workers in two capacitor manufacturing facilities exhibited Yusho-like eye conditions when they were examined.

Blurred vision, "weak" vision, and spots before the eyes are associated with exposure to PCBs and related products. Sensitivity to light is also commonly reported.

REPRODUCTIVE EFFECTS

Adverse reproductive effects of PCB exposure can form the basis of various kinds of litigation, including personal injury claims, demands for remedial clean-up to prevent loss of wildlife, and claims for damage to livestock¹

PCBs diminish the abilities of experimental and domesticated animals to reproduce;² adversely affect prenatal nervous system development in mice,³ rats,⁴ monkeys,⁵ and possibly man,⁶ and are known to cause deformities in mice⁷ and pigs.⁸ PCBs diminish reproductive capability in ways which are not thoroughly understood, but it is known that they decrease sperm production in mice,⁹ monkeys,¹⁰ and possibly man;¹¹ and interact with female sex hormones in animals¹² and humans.¹³

PCBs are known to cross the placenta, thereby contaminating children from the earliest stages of their development.¹⁴ PCBs are also transferred to children through their mothers milk.¹⁵

1. \$1.02 million was awarded to a mink breeder because PCB contaminated fish meal destroyed his stock; see: Bethlehem Mink Farm, Inc v. South Shore Packers, Inc., et al., Civ No.72-148 (D. N.H. Nov. 13. 1974). For a discussion of the adverse reproductive effects of PCBs on domestic animals, see Kaomar et al., 30 Vet Med 323 (1985); and for a discussion of the effects on mink, see: Bleavins et al., Polychlorinated Biphenyls (Aroclors 1016 and 1242): Effect on Survival and Reproduction in Mink and Ferrets, 9 Arch Environ Contam Toxicol 627 (1980). Effects on swine are reported in: Hansen et al., Effect of Polychlorinated Biphenyl Mixture on Swine Reproduction and Tissue Residue, 36 Am J Vet Res 23 (1975).
2. An review of the extensive literature linking PCBs with decreased reproductive ability appears in: Fuller and Hobson, Effect of PCBs on Reproduction in Mamals, page 101, in: Waid (ed), PCBs and the Environment (1987).
3. Chow et al., Neuropathology of "spinning syndrome" induced by prenatal intoxication with a PCB in mice, 31 Ann NY Acad Sci 373 (1979).
4. Shiota, Postnatal Behavioral Effects of Prenatal Treatment with PCBs (Polychlorinated Biphenyls) in rats, 53 Okajamas Fol Anat Jpn 105 (1976).
5. Bowman et al, Correlation of PCB Body Burden With Behavioral Toxicology in Monkeys, 9 Pharmacol Biochem Behav 49 (1978); also; Bowman and Heironimus, Hypoactivity in Adolescent Monkeys Perinatally Exposed to PCBs and Hyperactive as Juveniles, 3 Neurobehav Toxicol Teratol 15 (1981).
6. Jacobson et al., The Effect of Intrauterine PCB Exposure on Visual Recognition Memory, 56 Child Dev 853 (1985).
7. Watanaba and Sugahara, Experimental Formation of Cleft Palate in Mice with Polychlorinated Biphenyls (PCB), 19 Toxicology 49 (1981).
8. Earl et al., The Reproductive Effects of PCB 1254 in Beagle Dogs and Miniature Swine, 29 Toxicol Appl Pharmacol 104 (1979).
9. Sanders et al, Polychlorinated Biphenyls and Nutritional Restriction: Their Effects and Interactions on Endocrine and Reproductive Characteristics of Male White

Mice, 40 Toxicol Appl Pharmacol 91 (1977).

10. Allen et al., Reproductive Effects of Halogenated Aromatic Hydrocarbons in Nonhuman Primates, 230 Ann NY Acad Sci 419 (1979).

11. Dougherty et al., Sperm Density and Toxic Substances: A Potential Key to Environmental Health Hazards, page 263 in: McKinney (ed), Environmental Health Chemistry, Ann Arbor Press (1981). Dougherty states: "Multiple linear regression was used in an attempt to correlate the presence of specific toxic substances uncovered in the NCI screening with sperm densities. PCB uniformly gave negative slope correlations with sperm densities."

12. Gellart, Uterotropic Activity of Polychlorinated Biphenyls (PCB) and Induction of Precocious Reproductive Aging in Neonatally Treated Female Rats, 16 Environ Res 123 (1978).

13. Human effects were observed in the aftermath of the Yusho disaster. Effects included erratic menstrual cycles and possibly other manifestations as reported in: Yamashita and Hayashi, Fetal PCB Syndrome: Clinical Features, Intrauterine Growth Retardation and Possible Alteration in Calcium Metabolism (1985), referring to Kusuda, Yusho and Females, 38 Sanko to Fujinka 1063 (1971).

14. Yamashita and Hayashi, Fetal PCB Syndrome: Clinical Features, Intrauterine Growth Retardation and Possible Alteration in Calcium Metabolism, 59 Environ Health Perspec 41 (1985). See also: Allen and Barsotti, The Effects of Transplacental and Mammary Movement of PCBs on Infant Rhesus Monkeys, 6 Toxicology 331 (1976); also: Curley et al., Polychlorinated Biphenyls: Evidence of Transplacental Passage in the Sherman Rat, 11 Food Cosmet Toxicol 471 (1973).

15. Vodick and Lech, The Transfer of 2,4,5,2',3',4'-hexachlorobiphenyl to fetuses and nursing offspring, 54 Toxicol Appl Pharmacol 293 (1980). Human milk has been extensively studied and found to contain pesticide and PCB residues; see, for example: Musial et al., Presence of PCB, DDE, and DDT in Human Milk in the Provinces of New Brunswick and Nova Scotia, Canada, 12 Bull Environ Contam Toxicol 258 (1974).

Notes: I. PCBs cross the placenta. I. Allen and Barsotti, The Effects of Transplacental and Mammary Movement of PCBs

on Infant Rhesus Monkeys, 6 Toxicology 331 (1976); also, Polychlorinated Biphenyls: Evidence of Transplacental Passage in the Sherman Rat, 11 Food Cosmet Toxicol 471 (1987).

Direct evidence that PCBs and related compounds adversely affect human reproduction is documented in descriptions of the "cola-colored" offspring of Yusho victims.¹ Behavioral abnormalities were observed among the children of women exposed to PCBs and other contaminants through consumption of fish from the Great Lakes. It was suggested that these abnormalities were delayed central nervous system effects caused by PCBs.² The hypothesis that the PCBs in the fish are a significant toxic component is supported by a study showing that PCBs play a significant role in inducing cytochrome P-450 in young trout.³

Experimental evidence overwhelmingly shows that PCB exposure causes adverse reproductive effects,⁴ although one recent study indicates that PCB exposure reduces the number of terata observed in cyclophosphamide exposed mice. PCBs have been shown to affect sex hormone metabolism in animals⁵ and disrupt the menstrual cycles of women.

There has been at least one court case involving the adverse reproductive effects of PCB exposure. Bethlehem Mink Farm recovered a little over one million dollars after PCBs caused the mink to stop reproducing.⁶

1. Higuchi (ed), PCB Poisoning and Pollution, Academic Press, pages 19 and 20 (1976).
2. Jacobson et al., The effect of intrauterine PCB exposure on visual recognition memory, 56 Child Dev 853 (1985).
3. Xenobiotics in gametes of Lake Michigan lake trout (*Salvelinus namaycush*) induce hepatic monooxygenase activity in their offspring, 4 Fundam Appl Toxicol 1042 (1984).
4. Letz, Toxicity of PCBs, An Overview with Emphasis on Human Health and Occupational Exposure, prepared by the California Department of Human Health Services: page 15: "Adverse reproductive effects of PCBs have been noted in many mammalian and avian species. The pattern of reproductive effects include alterations in estrus cycles, failure of implantation, increased frequency of spontaneous abortions, low birth weight offspring, and decreased post-natal survival." page 16: "No specific teratogenic effects of PCBs have been observed in a variety of avian species." page 17: "PCBs show significant effects on reproductive competence in a variety of species. These effects increase

in intensity with increasing dosage and decrease with increasing chlorination of the PCB isomers. PCBs do not appear to be mammalian teratogens. A reasonable explanation for most of the reproductive effects of PCBs could be based on their estrogenic activity." Also: Barsotti et al., Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls, 14 Food Cosmet Toxicol 99 (1976); Kihlstrom, J.E. et al., Sexual function of mice neonatally exposed to DDT or PCB, 5 Environ Phys Biochem 54 (1975); and Linder, R. et al., The effect of PCBs on rat reproduction. 12 Food Cosmet Toxicol 63.

5. Kalcmar et al., [Dynamics of exogenous progesterone in the blood of pigs stressed with polychlorobiphenyls], 30 Vet Med 323 (1985); also, Bitman and Cecil, 18 J Agri Food Chem 1108, (1970).

6. Bethlehem Mink Farm, Inc v. South Shore Packers, Inc., et al., Civ No.72-148 (D. N.H. Nov. 13. 1974) \$1.02 million jury verdict. For a discussion of the adverse reproductive effects of PCBs on domestic animals, see Kaomar et al., 30 Vet Med 323 (1985).

DIAGNOSIS AND SURVEILLANCE

This report deals with two separate, but overlapping issues: medical examination (including laboratory tests) of possible victims of past exposure, for the purpose of assessing any damage done, and medical surveillance of recently exposed persons for the purpose of monitoring their progress. The schedule of examinations and tests, along with the methods to be used, is often called a "medical protocol", and we will adopt that usage here.

Need For a Testing Protocol

Both sides in any current or likely litigation will want to make sure that pertinent medical information is gathered at an early stage. Persons who have suffered recent accidental exposure can probably obtain funds from the potentially liable parties to pay for extensive medical surveillance. Refusal to pay would cast the potentially liable parties in a bad light, and leave open the possibility that they would end up paying for alleged damage that had not actually occurred. It would also mean that they would have no say about how and where the surveillance would be conducted.

Medical protocols for persons who suffered past exposure are essentially the same as those for recent victims, but they differ in two important ways; namely, those who have suffered recent exposure should be examined as soon as possible to establish a baseline profile against which any changes can be compared, and those who have suffered recent exposure need frequent follow up examinations to document possible consequences of and recovery from exposure.

Devising a Laboratory Protocol

Attorneys responsible for approving medical protocols will want to make sure the physician in charge is a PCB expert, or else they will want to insist that every test mentioned in the following sections be performed. Retaining a PCB expert is ideal because portions of this report can rapidly become obsolete. Protocols developed by experts can be tailored to specific circumstances such as the recentness severity of the suspected exposure, and the presence of specific suspected synergists.

Laboratory Tests

Laboratory tests should include:

LIVER FINE-STRUCTURE

1. electron microscopic examination of a liver biopsy specimen

COMMENT: Proliferation of the endoplasmic reticulum

was reported in autopsies on Yusho victims. It was observed more recently among persons exposed to smoke and soot from the Binghamton office building fire. Even in the absence of gross or microscopic morphological changes, electron microscopic examination can reveal pronounced changes in the ultra-fine structure of the liver; particularly, proliferation of the endoplasmic reticulum, a condition which has been attributed to elevated enzyme activity.

LIVER FUCTION - PORPHYRIA

2. porphyrin balance and levels
 COMMENT: The purpose is to detect the possible presence of chemically induced hepatic porphyria. This condition would indicate extreme exposure to PCBs, or else exposure to other chemicals.

LIVER FUNCTION - ENZYMES

3. urinary 6-b-hydroxycortisol
 4. urinary glucuric acid
 5. aminoantipyrine half-life
 6. Aryl hydrocarbon hydroxylase (AHH) and cytochromes P-448 and P450 should be measured directly if that is possible. (Urinary d-glucyric acid, urinary 6-b-hydroxycortisol, and aminopyrene half-life are indirect measures.)
 7. serum alkaline phosphatase
 8. serum pseudocholinesterase
 9. serum alanine aminotransferase (ALT)
 10. serum aspartrate aminotransferase (AST)
 11. serum gamma-glutamyltranspeptidase (GGT)
 12. serum ornithincarbamoyl transferase (OCT)
 13. glutamic oxalacetic transaminase (SGOT)
 14. other

URINALYSIS

15. complete routine morning urinalysis
 16. glucuric acid (also listed above)

BLOOD

TESTS

17. plasma triglyceride
 18. serum triglycerides
 19. serum total cholesterol
 20. serum high-density lipoprotein-cholesterol
 21. serum very high density lipoprotein-cholesterol
 COMMENT: Numerous studies have linked increases in 17, 18 and 19. and decreases in 20 and 21 to PCB exposure. These changes possibly indicate alterations in lipid metabolism which are indicative of serious underlying pathology and increased risk

of cardio-vascular disease.

22.	serum	albumin
23.		osmolality
24.	sperm	analysis
25.	peripheral lymphocyte	chromosome studies
26.	serum bilirubin	(total and indirect)
27.	serum protein	electrophoretic fractions
28.	hemoglobin and	packed cell volume
29.		hematocrit
30.	red cell count	

IMMUNE

SYSTEM

31.	serum globulin		
	Immunoglobulin A		
	Immunoglobulin B		
	Immunoglobulin		C
32.	lymphocyte		count
33.	leucocyte		count
34.	Total	T	cell
35.	T	helper	count
36.	T	cell	suppressor
37.	All other tests and measurements which would be performed in any thorough examination.		

ATTEMPTED DOCUMENTATION OF EXPOSURE

38. GC/MS analysis of blood and liver and adipose tissue biopsy specimens
 The best facility available should contract to :
 a) Quantify each specific PCB congener (testing for total levels would be of minimal value.)
 b) Quantify each PCDF congener.
 c) Quantify each PCDD congener.
 d) Attempt to quantify each chlorinated naphthalene congener.
 e) Attempt to quantify each chlorinated biphenylene congener.
 f) Attempt to quantify each chlorinated chrysene congener.
 g) Attempt to quantify all other chlorinated organic compounds

Item 38 is crucial. The analytical laboratory must be first rate. Analytical standards won't be available for many of the compounds determined, but educated estimates and raw data must be provided. The analysts must be alert to the presence of oxygen and phosphorous-containing metabolites.

Skin Examination

Patients who have been exposed to PCBs and related compounds should be examined for chloracne, erythema, and general appearance of the skin. Those who have been recently exposed to large amounts of PCBs and/or PCB combustion

byproducts would not be expected to develop disorders during the initial days and weeks after exposure, but documentation of the condition of the skin before the possible onset of symptoms is very important.

Chloracne occurs most frequently on the face, neck, shoulders, chest, and back. Color photographs of these areas taken before changes occur can become very valuable. Photography of the entire body is recommended. Close-up photography of the face will document swelling of the eyelids and pigmentation around the nose and lips should these Yusho-like symptoms occur. Karatotic plaques of the palms and soles were observed in about one-third of the Yu-Cheng victims.

Any acne which develops after exposure can be diagnosed as "chloracne", but many physicians require a certain degree of severity before they call it that. Some even insist on confirming hyperkeratosis and atrophy of the sebaceous glands by microscopic examination of lesion tissue. There is no general agreement on what is encompassed by the word "chloracne".

Itching, skin irritation, cysts, or boils may also be caused by exposure to PCBs and related compounds.

4:45. -- Eye Examination

"Cheese-like" discharges from the eyes and conjunctivitis were common among the Yusho victims. Exposed workers often complained of burning eyes, but that might have been primarily topical irritation. Other eye problems encountered among persons with histories of PCB exposure are blurred vision and sensitivity to light. It is not known if this is related to porphyria.

4:46. -- Lung Examination

Serial pulmonary function tests are recommended once every two years. Wheezing or signs of edema should be noted following heavy exposure. Decreased vital capacity and pulmonary secretions have been implicated as symptoms of PCB toxicosis. Initial chest X-ray would help establish a baseline.

4:47. -- Heart Examination

Studies of the general population have demonstrated a correlation between PCB levels and hypertension. Other studies have linked PCBs and elevated levels of serum triglycerides and cholesterol so monitoring of the major blood vessels is indicated. Fibrosis and cell death were observed in the heart of a dead Yusho victim.

4:48. -- Nerve Examination

Nerve conduction velocity can be significantly slowed by exposure to PCBs and its byproducts. The latency period for this effect is not known, so a determination immediately after accidental exposure is indicated. Numbness, tingling, insomnia, headache, fatigue, impotence, irritability, anxiety, and paranoia may all result from exposure to PCB related compounds. Even in the absence of physical trauma, fear of cancer and other diseases and suicidal impulses can result from perceived exposure to toxic substances.

The examinations for peripheral neuropathy and central nervous system damage might be the most important aspects of the protocol. It would be wise to consult with an expert in this area.

4:49. -- Liver Examination

In cases of sudden, massive exposure, especially where PCBs have been burned, an immediate needle biopsy for ultrastructural examination by electron microscopy is indicated. Months, or even years later, another biopsy is liable to reveal dramatic changes in the fine structure, even in the absence of gross or microscopic changes. The specimen should be handled carefully and stored in specially cleaned glass for future analysis by GC/MS; detectable quantities of PCBs and related compounds can be found even in a sample that small.

It is still not common to examine liver biopsy specimens when past, long term exposure is the issue, but that is likely to change. Evidence of past massive exposure remained in the livers of Yusho victims years after PCB levels in their bodies had returned nearly to normal. Autopsies revealed the evidence in the ultra-fine structure and the levels of some extraordinarily tenacious PCDF congeners. Persons with histories of occupational exposure to PCBs also suffered exposure to PCDFs, the amount depending on the type of job they had. Serious thought should be given to the feasibility of determining PCB and PCDF levels in biopsy specimens. It is only a matter of time before someone tests the liver of a deceased electrical equipment production or salvage worker. After that, a better assessment of the usefulness of subjecting previously exposed persons to the biopsy procedure can be made.

The examining physician should check for the presence of tenderness or enlargement of the liver. Examination should include CAT scans.

4:50. -- Genital Examination

Deformation of the penis¹ and atrophy of the testicles could result from exposure. Women are advised to schedule regular pelvic examinations and Pap smears after exposure to carcinogens such as PCBs and PCDFs.

1. This information, along with other unique items of interest, is reportedly contained in the deposition of B. Carnow and R.G. Loew, taken between November 1981 and April 1982 for the Circuit Court, Third Judicial Circuit of Illinois, Madison County; No. 79-L-810.

4:51. -- Miscellaneous Examinations
Gums Presence or absence of discoloration should be noted.

Nails Presence or absence of discoloration, hardness, brittleness, or deformation should be noted.

Stomach X-ray using contrast medium might be useful.

Spleen The spleens of experimental animals are harmed by PCB exposure. CAT scans are indicated.

Kidneys Persons recently exposed to fumes from burning PCBs should be checked for tenderness, since renal infections and hemorrhage have occurred after dioxin exposure.

Temperature Fever directly after exposure and long term changes in baseline temperature are possible.

Feet Stiffening and peeling of the soles have been frequently reported among persons exposed to PCBs. Thickening, hardening, and deformation of the toenails are also possible consequences of exposure.

Hair follicles Distinctive hair follicles were noted among approximately half of the Yusho and Yu-Ching victims.

Hands Hardening of the palms, discoloration near the joints, and discoloration near the cuticles all appear to be related to PCB exposure.

Joints Joint pain, sometimes diagnosed as bursitis, is liable to develop in persons exposed to PCBs and related compounds.

Other The examination should be quite thorough. Not enough is known about the effects of PCBs to rule out any outcome of exposure. Some symptoms not listed above which are often reported by exposure victims are:

1. sweating for no apparent reason
2. back pain
3. decreased nerve conduction velocity
4. increased sensitivity to the smell of chemicals
5. tumors
6. onset of allergies
7. weakening of grip
8. swollen fingers
9. shooting pains in the knees and elbows.

4:52.

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Medical

Questionnaire

The physician in charge should supervise the recording of:

- A) Each subject's complete medical history. This includes review of existing medical records and, wherever it might help, consultation with the subject's personal physician.
- B) Family history of cancer, allergy, heart disease, etc.
- C) History of medication, alcohol consumption, smoking, and all exposure to chemicals at work or elsewhere.
- D) Menstrual frequency, duration, and amount of discharge.
- D) All subjective symptoms including but not limited to
 - 1. itching skin
 - 2. loss of grip
 - 3. blurred vision
 - 4. spots before eyes
 - 5. sensitivity to light
 - 6. dizziness
 - 7. disorientation
 - 8. personality change
 - 9. irritability
 - 10. agriphobia
 - 11. claustrophobia
 - 12. nervousness
 - 13. fatigue
 - 14. memory loss,
 - 15. loss of libido
 - 16. sweating without apparent cause
 - 17. hot and cold flashes
 - 18. tingling or numbness in the extremities.
 - 19. nightmares
 - 20. sleeping paralysis
 - 21. hearing loss
 - 22. pain in bones
 - 23. pain in joints
 - a) including sore knuckles
 - b) including shooting pain in elbows
 - c) including shooting pain in knees
 - 24. pain in abdomen
 - 25. pain in back
 - 26. skin irritation
 - 27. eye irritation
 - 28. nose irritation
 - 29. nausea
 - 30. increased ability to smell xenobiotic chemicals
 - 31. tendency to drop things
 - 32. digestive problems
 - 33. colon problems

OCCUPATIONAL EXPOSURE TO PCBs

Introduction

Commercial PCB production began in the United States in 1929 and ended in 1977. Approximately 1.3 billion pounds were produced. Little of this was sold directly to consumers; nearly all went to the work place. Tens of thousands of workers were exposed.

Commercial PCB production was initiated by the Swann Chemical Company in 1929. The Monsanto Chemical Company purchased Swann in 1935, and dominated the domestic market until production ceased in 1977. Monsanto manufactured approximately 1.3 billion pounds of PCBs.

It is reported that after 1971, the year Monsanto severely restricted PCB sales, a now defunct Houston, Texas firm called Geneva Industries, produced approximately 1 million pounds of PCB based heat transfer fluid.² Another million pounds were imported into the United States. PCBs continued to be available on the open market from importers, reprocessors, and others until the Toxic Substances Control Act of 1976 made PCB sales illegal. According to information provided to the U.S. EPA in 1975 by Monsanto, a firm called Pyramid Chemical Sales Company of Fort Washington, Pennsylvania, was currently selling Aroclor 1221, Aroclor 1232, and Aroclor 5460. It is unclear whether these materials were authentic Monsanto products. If they were unused Aroclors, they must have been warehoused before Monsanto restricted sales, or else diverted to the open market by Monsanto's ostensibly electrical industry customers.

Prior to 1972, the major uses of PCBs in the U.S., listed in descending order, were: capacitor fluids, plasticizers, transformer fluids, hydraulic fluids and lubricants, and heat transfer fluids. In 1972, Monsanto, the sole domestic manufacturer of PCBs, restricted sales to customers using PCBs in "closed" electrical applications. This effectively limited sales to companies which manufactured capacitors and transformers, although it did not restrict their use of the product once they bought it. Monsanto stopped producing PCBs in late 1977.³

During the 1930's and 1940's, many capacitors and transformers were filled with "halowax", a product composed of chlorinated naphthalenes blended with larger amounts of PCBs.⁴ Outbreaks of occupational disease from exposure to these products is described in Chapter IV. Death from yellow atrophy of the liver was one consequence of exposure.⁵ The elimination of chlorinated naphthalenes from most electrical equipment applications may have resulted in fewer cases of a sometimes disfiguring disease called chloracne, and in fewer reported cases of rapid

liver degeneration.

1. Bourne, Polychlorinated Biphenyls (PCBs): An Environmental Health Hazard, Congressional Research Service, Library of Congress, page 1 (1976).

2. Brinkman and De Kok, Production, Properties, and Uses, page 3. In: Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products (1980).

3. MacLeod, Polychlorinated Biphenyls in Indoor Air, 15 Environ Sci Technol 926 (1981).

4. Brinkman and De Kok, Production, Properties, and Uses, page 23. In: Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products (1980).

5. Kimbrough, Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products, page 385 (1980).

The Production of PCBs

Monsanto is the only American firm known to have produced PCBs in amounts over 1 million pounds per year. Production occurred at two major plants, one in Anniston, Alabama and one in Sauget, Illinois.¹ These facilities stopped producing PCBs in 1971 and 1977 respectively.

To manufacture PCBs, biphenyl was heated slightly past its melting point (1xx degrees Centigrade) while chlorine gas was introduced for periods of time varying between 12 and 36 hours. The longer chlorination times resulted in mixtures with higher chlorine contents. Small amounts of ferric chloride were added to the reaction mixture to promote the reaction. Hydrogen chloride gas (HCl) was the major reaction byproduct.

After chlorination, the crude mixture was held at elevated temperature while air was blown over it for several hours. The ferric chloride and any hydrogen chloride which still remained were destroyed by the addition of a small amount of alkali to the reaction mixture. Most PCB products, including Aroclor 1242 and Aroclor 1254, were then distilled at reduced pressure.

Workers could have been exposed to PCBs primarily through respiration and skin absorption. An outbreak of chloracne among PCB production workers was reported in 1936,² but little subsequent information has surfaced. An unpublished Monsanto study of the Sauget facility shows above average occurrences of many categories of disease among the 86 PCB production workers studied.

1. Monsanto Company
Box 249
Anniston, Alabama 36201

Monsanto Company
Route 3
Sauget, Illinois 62201

Monsanto's primary mailing address is:

Monsanto Industrial Chemicals Co.
800 N. Lindbergh Boulevard
St. Louis, Missouri 63155

2. Jones and Allen, An Acneform Dermatargosis, 33 Arch
Dermat & Syph 1022 (1938).

For a detailed account of the environmental impact of PCB
production, see: Versar, Inc., PCBs in the United States
Industrial Use and Environmental Distribution, EPA 560/6-
76-005, (1976).

The Electrical Industry

PCBs have been used primarily by the electrical equipment manufacturing industry. Such equipment consists primarily of capacitors and transformers, items which will be discussed in greater detail shortly. They range in size from electrical substation transformers weighing many tons, down to small circuit board components, although the smaller items are not commonly referred to as "equipment." The metal boxes and cans commonly seen on power transmission line poles are capacitors and transformers.

Monsanto developed markets for PCBs outside the electrical equipment manufacturing industry, especially during the late 1950's and 60's. Sales volume for these other applications approached that of the electrical industry in 1970; but these applications were abruptly curtailed in 1972 after Monsanto's restricted sales to firms involved in so called "closed applications". Between that time and the termination of PCB production in 1979, sales were essentially restricted to the electrical equipment manufacturing industry. Only a small amount of PCBs were imported for other uses.

The electrical power transmission industry is unique in that it still uses large quantities of PCB's. Regulations banning PCBs from most uses do not apply to certain categories of electrical equipment currently in use or in storage, particularly when such equipment is not being used near food or feed, or near commercial buildings.

The Capacitor Manufacturing Industry

Capacitors improve the efficiency of power distribution through interstate grids, as well as the efficiencies of fluorescent lights, air conditioners, and most other electrically powered equipment. Aside from heaters, almost anything which requires electricity runs more economically when an appropriate capacitor is placed between it and its power source.

More PCBs have been used to manufacture capacitors than for any other use. Over 20 million pounds of PCBs were used for this purpose each year during the late 1960s. In 1976, there were 17 companies in the United States which manufactured PCB containing capacitors at 19 plants (see table 3-1).¹

Typically, PCBs arrived at the factories via tank car or tank truck, where they were transferred to large storage tanks. They were then purified by filtration through Fuller's earth, and stored prior to being introduced into capacitors.

Two major components of PCB capacitors are a metal can and a roll of paper or polypropylene film (in some cases, both) and aluminum foil. The paper or film is covered with the metal foil prior to rolling, so the final product has alternating layers of paper or film and foil. The paper or film and foil are wound a sufficient number of times to produce a roll which fills most of the capacitor can. The amount of PCBs used in capacitors ranges from less than a pound to 77 pounds. Fluorescent light ballasts and air conditioner capacitors typically contain about 1 pound of PCBs; the most common power line capacitors contain 36 pounds.

1. PCB regulations are in 40 CFR Part 761. The most recent regulations pertaining to electrical equipment were promulgated in Fed Reg, 17 July 1985.

Capacitor Manufacturing Operation

Capacitor cans are fabricated from sheet metal either on site or elsewhere. The internal foil rolls are assembled, placed inside the pre-cleaned cans, and the appropriate electrical connections are made. Leak tests are performed prior to filling with PCBs.

Both the liquid PCBs and the assembled, unfilled capacitors are thoroughly dried to prevent even minute traces of water from being enclosed inside. To accomplish this, the PCBs are filtered through Fuller's earth and the unfilled capacitors are heated inside vacuum chambers. After drying, the vacuum is released and PCBs are allowed to flood the chamber. The capacitors, submerged in PCBs, fill through special valves or orifices. After a suitable length of time, the capacitors are removed, washed, and tested. Those which pass the test are processed for distribution.

PCB exposure inside capacitor manufacturing plants may have been greatest in areas where capacitors were filled with PCBs. The heat used to dry unfilled capacitors vaporized PCBs which were present from previous fill operations, leading to exposure through the air. Capacitor explosions in testing areas also led to a certain amount of exposure. To some extent, PCBs permeated entire capacitor manufacturing plants, and all workers received above average exposure.

Other exposures in capacitor manufacturing plants involved electroplating and de-greasing operations. The de-greasing often involved cleaning with trichloroethylene. There is evidence that exposure to PCBs increases susceptibility to the ill effects of TCE.

The Transformer Manufacturing Industry

Transformers consist of copper coils inside of a metal casing which is usually flooded with an oily fluid. Most transformer fluid consists of mineral oil, but between 5 and 10 percent contain PCBs mixed with 30 to 40 percent chlorobenzenes.

The large boxes in electrical substations are transformers, as are cans on utility poles and controls for toy electric trains. Most PCB transformers contain between 350 and 15,000 pounds of the fluid, about 2500 gallons being average.

Transformers are used to regulate voltages and to change alternating current supplied by utilities companies to direct current for use in electric motors. Some are used to reduce the high voltage in power distribution lines to more usable levels, such as the familiar 110 and 220 Volts used in the home and in light industry. Others raise voltages for specific applications. Some, called reactors, do not normally change voltages at all; but during power surges they reduce voltages in order to maintain the normal level.

Pure PCBs were seldom used to fill transformers. The usual industry practice was to use mixtures containing 60 to 70 percent PCBs and 30 to 40 percent chlorobenzenes. The chlorobenzenes were usually trichlorobenzene. These mixtures were called "askarels." "Askarel" is also used as a generic term meaning PCBs, or flame resistant transformer fluid. For the purposed of this monograph, we will restrict the use of "askarel" to mean PCBs mixed with chlorobenzenes. Askarels were blended and distributed by Monsanto.

Transformers last for over thirty years. Current regulations require the replacement or conversion of only a small percentage of those which contain askarels; primarily those involved in food or feed production, near commercial buildings, and on railroad locomotives and cars. 5 percent of the transformers in service contain askarels; most others contain mineral oil.

Askarels in transformers provide insulation, protection from corrosion, and in some units, cooling. Mineral oil performs about as well as askarels in most respects, but it has the disadvantage of being flammable.

Askarel-insulated transformers were manufactured by 13 U.S. companies at 18 plants (see table 3.2.1-1). 12 million pounds of PCBs were used for this purpose in 1974.²

1. Most of the information in this chapter can be found in: Varsar Inc., PCBs in the United States Industrial Use and Environmental Distributions, EPA 560/6-76-005, NTIS PB-252 012 (1976). See page 88 for figures on U.S. production.

The Transformer Manufacturing Process

As was the case with capacitors, it was necessary to exclude air and moisture from the inside of askarel transformers. This was done by filtering the askarels through Fullers earth, and by the use of heat and vacuum on the metal transformer components. Particular processes depended on the size of the transformers being manufactured.

Some transformers were pressure tested before filling. All transformers were electrically tested after filling. Large or medium sized transformers which failed the electrical tests were drained and reprocessed. This is in contrast with the procedure for capacitors, which was to discard most rejects.

Farmers

Farmers and their families were exposed to PCBs though the use of PCB containing silo coatings. Exposure was possible through various routes, including consumption of milk and meat from livestock which consumed contaminated silo stored grain.

Waste Disposal Workers

Emergency Response Personnel

Railroad Workers

Other Current Occupational Exposure

Metal salvagers transformer repair workers can become exposed to PCBs and related compounds. Salvagers who burn insulation away from electrical equipment are particularly threatened by chlorinated phenols, chlorinated dibenzofurans; and other toxic products of PCB combustion.

Office workers become exposed to high levels of PCBs when fluorescent light ballasts overheat and leak. Maintenance and repair workers can experience PCB exposure through contact with old hydraulic fluids, such as brake fluid or hydraulic lift fluid. Old heat exchange fluids are also potential sources of exposure.

Fishermen are also in potential danger since some of them, because of the availability of shellfish and fish, eat more of it than most people. Freshwater catches and shellfish from contaminated bays estuaries contain far more PCBs than other currently available foods.

Fires and Explosions

PCB fires and explosions may now represent the most common route of occupational PCB and PCB byproduct exposure. National attention was focused on the hazards of PCB fires in early 1981 as information became available that PCB transformer fire had spread dioxins and furans throughout an 18 story office building in Binghamton, New York. \$1 billion dollars in liability claims have been filed, and the cleanup cost is estimated at about \$24 million.¹

Another widely publicized incident occurred on May 15, 1983, in San Francisco, California. Smoke from a fire in an underground transformer vault escaped into a building and out through a sidewalk grate. \$10 million was spent cleaning up five floors of the contaminated building. California Pacific Power and Light is now in the process of retiring all PCBs from service.

Numerous similar incidents involving PCB fires have been reported,² and while these incidents are alarming in themselves, their significance for those not directly affected might be primarily to warn of what happens in thousands of fires each day which are not characterized as PCB fires. Transformers, light ballasts, and thousands of other PCB containing products threaten victims, firemen and bystanders whenever fire breaks out.

Fires involving PCBs are important not only because heat disperses PCBs into the air, but also because the products of incomplete combustion include chlorinated polycyclic compounds such as the PCDFs.³ Accidental fires involving PCBs present a grave threat to anyone exposed to the smoke and soot.

Capacitor explosions are a common occurrence. In the absence of fire, they do not appear to generate the extraordinarily toxic byproducts which are associated with PCB combustion, but they do create dangerous situations requiring prompt and careful attention

1. Lee, Assessment of PCDDs and PCDFs from PCB Transformer and Capacitor Fires, page 2, U.S. EPA, Cincinnati (1984).
2. Lee, Assessment of PCDDs and PCDFs from PCB Transformer and Capacitor Fires, U.S. EPA, Cincinnati (1984).
3. Erickson, PCDF Formation from Fire Conditions, Midwest

Research Institute Draft Interem Report No. 1, for U.S. EPA
(1984).

Incinerator Workers

The intentional incineration of PCBs (still advocated by some as a safe alternative to landfilling) may be having serious adverse consequences on the health of incinerator workers, nearby residents and on the environment in general. The extent to which incoming PCBs, leaking vapors, toxic ash, and other sources of contamination have harmed workers is not currently known, but it can be assumed that such workers experience higher than average exposure.

Workers at the ENSCO incinerator in El Dorado Arkansas were found to have large amounts of PCBs in their blood (See the attached correspondence from NIOSH to the Arkansas Health authorities).

See Jensen, The PCB story, 1(4) Ambio 123 (1972); also Rita Gray Beatty, The DDT Myth (1973).

UPCOMING REGULATORY DEADLINES

Upcoming deadlines for removing or protecting electrical equipment in service were published in the Federal Register on July 17, 1987 (50 Fed. Reg. 29170), and are found in the Code of Federal Regulations at 40 CFR 761. Proposed revisions to the deadlines were published in the Federal Register on August 21, 1987, at 52 Fed. Reg. 31738.

Definitions

PCB Transformer means any transformer containing fluid with more than 500 ppm PCBs.

PCB Contaminated equipment is equipment containing fluid with between 50 and 500 ppm PCBs.

Higher Secondary Voltage is used to refer to transformers with secondary voltages equal to or greater than 480 volts.

Lower Secondary Voltage is used in reference to transformers with secondary voltages under 480 volts.

Commercial Building means a non-industrial building which is typically accessible to both members of the general public and employees. These include public assembly properties, educational properties, institutional properties, residential properties, stores, office buildings, and transportation centers.

Deadlines in Effect Now

After October 1, 1985

The use or storage of PCB Transformers that pose an exposure risk to food or feed was prohibited.

The installation of PCB Transformers was prohibited.

Registration with fire service personnel of all PCB Transformers, both in and out of service, was required.

Registration with building owners of all PCB Transformers in or near commercial buildings was required.

October 1, 1988

The use and storage for reuse of PCB Large High Voltage Capacitors and PCB Large Low Voltage Capacitors which pose an exposure risk to food or feed will be prohibited.

The use of PCB Large High Voltage Capacitors and PCB Large Low Voltage Capacitors used outside of restricted access electrical substation and restricted-access indoor locations will be prohibited.

October 1, 1990

The use of network PCB Transformers with higher secondary voltages in or near commercial buildings will be prohibited.

All radial transformers and lower secondary voltage network transformers must be protected from high voltage faults.

All radial transformers with higher secondary voltages must be protected from high and low voltage faults.

PCBs may be used in circuit breakers, reclosers, and cable for the rest of the equipments' useful lives.

PROPOSED REVISED DEADLINES

Discovery If a previously untested mineral oil transformer is found to contain greater than 500 ppm PCBs, the owner must take measures including registering it, and removing, retrofilling, or protecting it by the following deadlines or within 18 months, whichever is later.

October 1, 1990

Deadline for retrofilling "askarel" PCB Transformers while they are in service, or for installing retrofilled PCB Transformers for reclassification purposes.

October 1, 1993

Deadline for removing from service low secondary voltage network PCB Transformers as an alternative to installing enhanced electrical protection before October 1, 1990.

Deadline for removing from service lower secondary voltage network PCB Transformers located in sidewalk vaults.

REGULATION OF PCBs IN ELECTRICAL EQUIPMENT

Development of Regulations

On September 1, 1971, representatives of several agencies of the Federal Government formed an interdepartmental task force to study PCBs. The major sponsoring agencies were the Environmental Protection Agency and the Departments of Agriculture; Health, Education, and Welfare; Interior; and Commerce. Industry representatives played a crucial role in helping the task force develop its recommendations, particularly those relating to the continued use of PCBs in electrical equipment.

On March 20, 1972, the Task Force issued the following findings, conclusions, and recommendations:

1. PCBs should be restricted to essential or non-replaceable uses which involve minimal direct human exposure since they can have adverse affects on human health.

2. PCBs have been used so widely over such a long period that they are ubiquitous.

3. PCBs were first identified as potential food contaminants in 1966. The three principal dietary sources are fresh water fish, food packaging material, and accidental PCB leaks into food or feed.

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 task force went on to state that: "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 task force recommended passage of the Toxic Substances Control Act.

5. Housekeeping is particularly important in the manufacture, use, and disposal of PCBs.

6. The use of PCBs should not be banned entirely. The task force elaborated as follows: "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.

8. PCBs are manufactured in countries other than the United States.

9. More scientific information about PCBs is needed, and several Government agencies are seeking it through research.

Bureau of Standards Report

The report of the Task Force included a three page review of the benefits and utility of PCBs prepared by the National Bureau of Standards and based partly upon information supplied by the National Industrial Pollution Control Council and other unspecified associations. The National Bureau of Standards report stated that fluorocarbons are the other class of non-flammable fluids which are comparable to PCBs. They pointed out that the fluorocarbons are more volatile than PCBs. They concluded that "If codes did allow flammable materials in (buildings in which flammable fluids in capacitors are prohibited by insurance companies and building codes), 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."

The Bureau concluded that adequate substitutes for PCBs were available for use as hydraulic fluids, lubricants, heat transfer fluids, and plasticizers.

PCB Regulation Pursuant to TSCA

The Toxic Substances Control Act of 1976 (TSCA)¹ requires the EPA to gather information about toxic substances from manufacturers, regulate chemical substances and mixtures which "present an unreasonable risk of injury to health or the environment", and to take action with respect to "imminent hazards". In addition to these general purposes, TSCA specifically requires that EPA take action regarding PCBs.² PCBs are the only chemicals which are specifically mentioned in the Act. Section 6(e) of TSCA bans the manufacture, processing, and use of all PCBs which are not "totally enclosed" after January 1, 1979, bans the distribution of PCBs in commerce after July 1, 1979, and requires proper disposal of PCBs. The EPA is authorized to make renewable one-year exceptions upon petition.³

1. 15 U.S.C.A. Sections 2601 et seq.
2. 15 U.S.C.A. Sections 2601 et seq.
3. 15 U.S.C. 2605(e)(2)

The PCB Ban Rule

Pursuant to the requirements of TSCA, EPA promulgated regulations in 1979 which prohibited the manufacture, import, processing, distribution in commerce, or export of PCBs unless an exemption was granted. 44 Fed. Reg., 31514 (May 31, 1979). Eleven specific exemptions were granted for uses which did not seem to pose a significant threat, and where adequate substitutes were unavailable. These included the use of PCBs in servicing transformers, the use of small quantities for research, and as a microscopic mounting medium.

In the 1979 promulgation, EPA also exempted from regulation all material containing less than 50 ppm PCBs; all intact, nonleaking capacitors and electromagnets; and all intact, nonleaking transformers which were not being used on railroads.

The Environmental Defense Fund petitioned the U.S. Court of Appeals for the District of Columbia Circuit to review these decisions, ¹ and on October 30, 1980, the court ruled that there was insufficient evidence in the record to support the EPA's classification of transformers, capacitors, and electromagnets as "totally closed", and remanded the matter to EPA for further action. On January 21, 1981, EPA, EDF, and certain industry interveners in EDF v. EPA filed a joint motion asking the court to stay its mandate setting aside the classification of transformers, capacitors, and electromagnets as totally enclosed. EDF agreed with the other parties' contention that immediately taking all of the PCB containing equipment out of service would be impractical.

On February 12, 1981, the court issued an order staying its mandate regarding capacitors, transformers, and electromagnets pending further rulemaking. 46 Fed. Reg. 16090 (March 10, 1981). On April 13, 1981, the court stayed its mandate with respect to activities involving PCBs in concentrations of less than 50 ppm pending further rulemaking. 46 Fed. Reg. 27615 (May 20, 1981). The court orders contained several provisions, including the following:

- 1) The EPA would publish an Advance Notice of Proposed Rulemaking, and follow up by developing a rule.
- 2) The Edison Electric Institute (EEI) and the Utility Solid Waste Activities Group (USWAG) would develop some of the factual material necessary for the rulemaking.
- 3) The parties would abide by a specified Interim

Measures Program.

4) The EPA would promulgate a final rule within six months of receipt of the study from EEI and USWAG.

1. The Environmental Defense Fund, Inc. (Petitioner) v. Environmental Protection Agency (Respondent), 636 F.2d 1267 (1980). Interveners were: Ad Hoc Committee on Liquid Dielectrics of the Electronic Industries Association et al., Joy Manufacturing Company, Edison Electric Institute et al., and Aluminum Company of America. The Color Manufacturers Association filed an amicus curiae brief.

Electrical Equipment Regulations Under TSCA

Following the decision in EDF vs. U.S. EPA, EPA published interim regulations regarding the inspection and maintenance of transformers. 46 Fed. Reg. 16090, March 10, 1981. On August 25, 1982, EPA promulgated "final rules" (they have since been changed) concerning PCBs. 47 Fed. Reg. 37342, codified at 40 CFR Part 761. The 1982 rules banned the use of PCB Transformers (electrical transformers containing more than 500 ppm PCBs) in facilities involved in the handling of food or feed effective October 1, 1985, and allowed the use of all other non-railroad electrical transformers for the remainder of their useful lives.

Stricter rules were promulgated on July 17, 1985, because of danger posed by PCDFs and other products of incomplete PCB combustion. Highlights of these rules are as follows:

1) Prohibition of the continued use of some types of high voltage PCB transformers near commercial buildings beyond October 1, 1985.

2) Prohibition of future installation of some types of high voltage PCB Transformers near commercial buildings.

3) Installation by 1990 of protective equipment on certain high voltage PCB transformers located in or near commercial buildings.

4) Required registration of all PCB Transformers with fire departments and building owners by December 1, 1985.

5) Required marking of all PCB Transformer locations (except grates and manholes) by December 1, 1985.

6) Required removal of combustible material from the vicinity of PCB Transformers by December 1, 1985.

7) Required precautions in the event of a PCB Transformer fire.

8) Required notification of the National Response Center in the event of a PCB Transformer fire.

Responsibility for compliance with TSCA regulations rests primarily with owners of transformers. Users of transformers posing a risk to food are responsible for required inspection, maintenance, and record keeping until they notify the owner of the risk. EPA holds users at least partly responsible when the

user has agreed to perform the owner's duty, the user has traditionally taken responsibility, the owner's access to the equipment has been limited, or there is an emergency. TSCA Compliance Program Policy No. 6-PCB-1, available from EPA.

PCB Disposal Under TSCA

When PCBs and PCB items are removed from service, they must be disposed of in accordance with regulations promulgated by the U.S. EPA pursuant to TSCA. PCBs, PCB items, and PCB contaminated dirt and debris which are in disposal sites are considered "in service" for the purposes of the regulations. PCBs which were landfilled prior to February 17, 1978, are not regulated under the TSCA regulations unless they are removed from the site.

PCBs which have been removed from service may be stored for no more than one year prior to disposal. 40 CFR Section 761.65. This requirement is not literally interpreted when PCBs are placed into bulk storage tanks. In those cases, levels in the tanks are not permitted to rise during any 12 month period.¹

EPA has established detailed requirements concerning the disposal of various types of materials. 40 CFR 761.60. Diluting contaminated material does not affect its regulatory category. Material containing over 500 ppm PCBs may not be landfilled; it must be disposed of in a way which EPA deems to be at least as efficient as incineration.² Performance standards for incinerators which burn PCBs are found at 40 CFR 761.70.

1. TSCA Compliance Program Policy No. 6-PCB-10, August 13, 1985.

2. 40 CFR 761.60(a)(1) requires incineration, 40 CFR 761.60(e) provides for alternatives which are at least as efficient.

PCB Spills

The U.S. EPA Published its policy regarding PCB spills on April 2, 1987. 52 Fed. Reg. 10688. The Federal Register notice includes the following background information:

- On an annual basis, about 3.3 percent of PCB transformers in use will leak or spill PCBs.
- On an annual basis, EPA expects that about 264,000 pounds of PCBs are leaked or spilled into the environment from PCB transformers.
- Of the 1.6 million PCB capacitors in use in electrical substations, EPA expects that over 12,000 leak each year, releasing about 200,000 pounds of PCBs.
- Of the 1.2 million PCB capacitors in use inside buildings and on utility poles, EPA expects that over 9,000 leak each year, releasing about 154,000 pounds of PCBs.
- The majority of spilled PCBs are spilled from capacitors, and capacitor spills typically result from violent ruptures.
- The cost of cleaning up a typical capacitor spill is on the order of \$4,000, \$10,000, or \$60,000 to \$140,000 depending on whether the cleanup of the contaminated area is to 25 to 50 ppm, 10 ppm, or background levels of PCBs.

The EPA requires that PCBs be cleaned up to a level of 25 or 50 ppm in fenced-off electrical substations and other restricted access areas; to a level of 10 ppm in most other areas; and to lower levels when water is threatened, vegetable gardens are affected, or grazing land is contaminated.

Regulations Pursuant to RCRA and CERCLA

The Resource Conservation and Recovery Act of 1976 (as amended in 1984) gives the EPA the authority to regulate waste disposal facilities, and to initiate judicial action where waste disposal presents an "imminent hazard". Because Section 6(e) of the Toxic Substances Control Act specifically requires the regulation of PCBs, they were not listed as hazardous substances in the regulations which were promulgated pursuant to the enactment of RCRA.¹ Because of this, wastes which contain no hazardous constituent other than PCBs are not regulated under RCRA. The EPA has proposed classifying PCBs as a RCRA hazardous wastes, but the proposal has come under opposition from a coalition of industry and environmental groups. The RCRA regulations already include them as hazardous constituents of waste and subject them to special handling, reporting and record-keeping requirements. 40 CFR 261.11, promulgated 5/19/80.

The Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA) as extended and amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA),² commonly known as the "Superfund" act, does not specifically mention PCBs, but it contains provisions which are relevant to environmental contamination by PCBs. Section 103(b) provides for penalties of up to \$10,000 in fines and one year in prison for failure to notify the Emergency Response Center of a spill involving the release of more than 10 pounds of PCBs within a period of 24 hours. The reportable amount would have been 1 pound under CERCLA alone, but the 10 pound limit set pursuant to Section 311(b)(4) of the Clean Water Act takes precedence. Under EPA's spill policy (52 Fed. Reg. 10688) all PCB spills must be cleaned up, regardless of whether or not the spill must be reported.

Section 103(c) of CERCLA provides for fines and imprisonment of up to \$10,000 and 1 year for failure to report stored or discarded PCBs within 180 days of December 11, 1980. Section 103(d) provides for fines and imprisonment up to \$20,000 and one year for knowingly destroying, mutilating, erasing, disposing of, concealing, or otherwise rendering unavailable or unreadable or falsify any records concerning stored or discarded PCBs.

Sections 106 et seq. give the president of the United States the authority to require the Attorney General of the United States to protect the public from any actual or threatened release of hazardous substances, including PCBs. This authority allows the federal government to clean up PCB

contaminated sites, and force the liable parties to pay punitive damages of three times the government's expense. This has the effect of encouraging potentially liable parties to cooperate in clean-up efforts.

1. The original proposal (43 FR 243, p.58946 [12/18/78]) did specify PCBs.

2. CERCLA is Pub. L. 96-510, 42 U.S.C. 9601 et seq. SARA is Public Law 99-499.