PCBs In Perspective

by Harry A. Onishi
and W. Corey Trench

Once again PCBs have surfaced to become a major topic for discussion. For that reason it seems worthwhile to develop a brief history of the life of this group of organic chemicals that has drawn such extensive attention from scientists, mass media and the general public. This article endeavors to bring PCBs into perspective and to express a reasonable hope for a sound solution to the PCB problem in the long range interest of the nation.

PCBs are Polychlorinated Biphenyls—a broad range of organic chemicals also commonly known as chlorinated hydrocarbons. PCBs are a complex mixture of compounds. Theoretically, some 209 different molecule structures may be formed. In general though, a much smaller number arise in the manufacturing process or occur

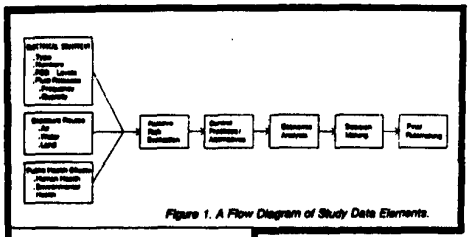

Figure 1. A Flow Diagram of Study Data Elements.

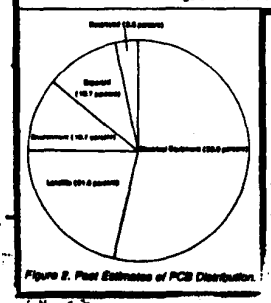

Figure 2. Past Estimates of PCB Distribution.

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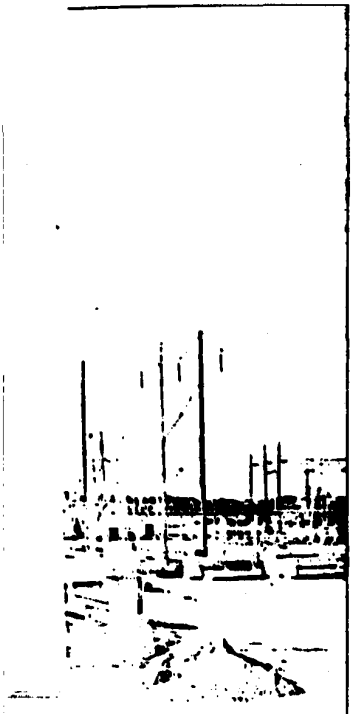
in the environment.

PCBs are very stable, possess excellent dielectric and cooling properties and are fire resistant. Initially, these properties made PCBs a superb fluid for use in transformers and capacitors to prevent or reduce fire risk in cases where the location of equipment was of particular importance. Later, PCB uses expanded to include: hydraulic lubricants, paints, varnishes, sealants, plasticizers, carbonless paper and other coatings.

The generic name for the fire resistant dielectric fluids is Askarel. Transformers that employ PCB fluids are commonly referred to as Askarel transformers. Companies manufacturing electrical transformers and capacitors with PCB may use a different trade name: Prydaual, Pyranol, Inerteen, SAF-T-Kuhl exemplify just a few. Askarel fluids consist of a mixture of PCB and solvents, generally chlorinated benzenes.



Typical transformer use in a low-pressure substation.

Monsanto Company was the principal manufacturer of PCBs, trademarked Aroclor, in the United States. The company entered the business in 1935 upon the acquisition of Swann Chemical Company. From 1935 through the early 1970s, production steadily increased to meet the demand. In the early '70s, however, Monsanto voluntarily restricted the sale of chlorinated biphenyls. Production of open-ended uses—hydraulic fluids, lubricants and the like—was curbed owing to a greater potential of PCB dispersal into the environment. Enclosed electrical systems uses of PCBs continued until 1977. By 1977, all manufacture of PCBs ceased, nearly two years in advance of a ban on their manufacture imposed by the Toxic Substance Control Act.

In 1976, Versar Inc., under contract to EPA, assembled an inventory of PCB sources. The quantities could only be estimated yet they represent an attempt to account for the mass balance of PCBs in use and in the environment. Commercial manufacture of PCBs is probably not the entire source of the compounds. Other industrial and municipal processes have the potential for producing different PCBs in varying quantities. Nature's processes have never been identified as PCB contributors, but the mechanisms have not been proven implausible. Of the documented estimates, an approximate 1.4 billion lb of PCB was produced in the United States. Fifty million lb have been destroyed, 300 million lb reside in landfills, 150 million lb occur in the environment, 150 million lb were exported and 750 million lb exist in use. Seven hundred and fifty million lb represent about 53.6 percent of the total PCB production and the bulk of these PCBs occur in enclosed electric equipment systems. The remaining material is used in "open-ended" applications such as the hydraulic fluids, lubrication fluids, etc. (Fig. 2).

For electrical systems, an estimated 300 million lb occur in transformers, 370 million lb in large capacitors and 180 million lb are in small capacitors and other devices (Table 1).

Environmental and Health Significance

PCBs became the center of national and international concern in the late 1960s. Two events are credited with the evolution of concern: The discovery and identification of PCBs in environmental samples by Soresen Jensen, and the Yusho incident in Japan.

In the course of environmental studies to detect chlorinated hydrocarbons and DDT in a variety of samples, Jensen discovered PCBs. Their presence, though an unexpected find, distinguished PCBs as an environmental contaminant. Subsequent studies detected PCBs in remote as well as industrial environments and established the widespread distribution phenonmenon of PCBs. Also, some of the desirable aspects of PCBs were singled out as undesirable from an environmental perspective, such as the chemical's stability and non-biodegradability. PCBs were found in varying concentrations in ecological food chains.

Meanwhile, an incident occurred in Yusho, Japan that involved the contamination of rice oil by heat exchange fluids and ingestion of the oil by an isolated human population. An outbreak of adverse human health effects was related back to the contamination. The fluid was tested and found to contain concentrations of PCBs, polychlorinated dibenzofurans (PCDFs) and polychlorinated quaterphenyls. The latter two compounds were impurites of the PCB fluid. The PCBs and the impurities were linked to the registered health effects. In retrospect, it seems more likely that PCDFs or other impurities may have played the more significant role in the outbreak as opposed to PCBs. PCDFs are known to be more toxic than PCBs on several orders of scale.

Nevertheless, the results of Yusho began the establishment of PCB as toxic to man.

Actions Taken to Control PCBs

Following the Yusho event, Japan prohibited the use of PCBs in new replacement and replaced PCB equipment in high exposure areas such as electric trains. However, existing PCB-filled transformers and capacitors were to remain in use until termination of the service lives of the equipment. Other European countries have also prohibited further use of PCBs; however, some countries have not believing PCBs can be controlled by proper handling and disposal practices. As a matter of interest, PCBs are still manufactured in Germany, France, Spain, Italy and certain Eastern European countries. They continue to be used throughout the world.

As for practices in the United States, the Toxic Substances Control Act (TSCA) was passed in 1976 authorizing the U.S. Environmental Protection Agency (EPA) to promulgate PCB regulations. Initial regulations were issued in February, 1978 setting forth requirements for marking, recordkeeping, storage for disposal and

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regulations banned the manufacture, processing, distribution in commerce and use of PCBs, except in totally enclosed systems such as transformers and capacitors or other uses specifically authorized by the Administrator of EPA.

Regulations were developed to control PCBs from all sources. The extent of these regulations has been primarily applied to the utility industry and owners/operators of electrical equipment. The voluntary termination of production of PCBs relieved a major source of potential environmental contamination. Regulations have also effected some chemical manufacturers incidently producing low level PCBs in certain chemical processes.

The full brunt of regulations has been borne by those with PCBs in closed systems, i.e., electrical transformers and capacitors. Utilities have significant numbers of PCB (Askarel) transformers and power capacitors on their systems. In addition, utilities possess transformers and other electrical devices that may contain incidental concentrations of PCBs in the oils of these pieces of equipment. The May 31, 1979 federal regulations define PCBs as any mixture containing 50 ppm (parts per million) or greater. The definition covers mineral oil-filled transformers which may be contaminated by previous manufacturing practices or in the servicing of transformers. Virtually all transformers on utility systems are classified as PCB contaminated under the current regulations and special care must be taken on ultimate disposal of these oils.

Actions Taken by Utilities

The government control of PCBs from the Congressional ban to regulation by state and federal agencies has been extensive. Utilities and enterprises have, in many cases, gone well beyond regulatory requirements. A summary of these activities includes

- Rapid response/comprehensive spill prevention and cleanup protocol.
- Use of sensor and relay devices to detect incipient equipment failure.
- Phase-out of PCB-filled equipment on accelerated bases or use of retrofilling options.

One should keep in mind that for self-imposed control activities, the possibilities may be limited. Limiting variables abound in:

- Excessive capital and operating costs balanced against limited benefits.
- Unavailabilty of replacement equipment or fluids.
- Pending tests of new technologies, and resulting unresolved problems or economic infeasibility.
- Unavailability of adequate or realizable disposal capacity.

EDF vs. EDA: Starting over Again

In late 1979, the Environmental Defense Fund (EDF), a Washington, D.C based environmental group, challenged certain provisions of the EPA regulations. The results of the case temporarily stunned the regulated community and resulted in further government actions. Challenged issues included: The distinction between a non-PCB item and a PCB-item by a 50 ppm cut-off; classification of transformers, capacitors, and electromagnets as totally enclosed systems; and authorized use of 11 activities, including the servicing and maintenance of transformers.

The U.S. Court of Appeals rendered a decision on October 30, 1981, the Court ruled in favor of EDF on the first two issues and in favor of EPA on the use authorizations. On the former issues, the Court found the rulemaking record deficient in information justifying EPA's selection of a 50 ppm cut-off level and classification of electrical equipment as totally enclosed systems. The deficiency in recorded information precipitated the Court's decision, not the inappropriateness of EPA's intent. The Court remanded the two issues back to EPA and ordered the Agency to initiate a new rulemaking in a timely fashion, replete with the appropriate support information.

The consequences of the decision were potentially significant. No cut-off would exist and formerly totally enclosed systems could be construed to be open uses. The possibly far-reaching effects led representatives of industry, Edison Electric Institute (EEI) and the Utility Solid Wastes Activities Group (USWAG) among other parties to achieve a negotiated agreement with EPA and EDF and avert the disruption of electrical service. A joint petition was filed on January 21, 1981. Shortly, thereafter, on February 12, 1981, the Court acted to stay its mandate for 18 months granting time for EPA to promulgate new regulations. As conditions of the stay, the electric utilities agreed to carry out a temporary, Interim Measures Program and to initiate an information gathering study to assist the Agency in the new rulemaking.

The Interim Measures Program requires the inspection and maintenance, if necessary, of certain transformers. Instituted May 11, 1981, the program monitors the possibility of exposure to fluids that may contain PCBs. Specifically, *all* transformers at federally inspected meat, poultry products and egg product establishments as well as other establishments engaged in the manufacture, processing, packaging or holding of human food or animal feed for wholesale purposes—exclusive of retail locations such as grocery stores, restaurants, bakeries, etc., that pose a risk of potential contamination—must be inspected on a weekly basis. In addition, *all* other PCB (Askarel) transformers must be inspected on a quarterly basis. The Interim Measures Program aims to further reduce a minimal risk of widespread PCB contamination

Table 1
Quantities of PCBs in Use

Use	Pounds Used	Pounds Regulated	Pounds Prohibited
PCB Transformers	286,000,000	286,000,000	0
PCB Contaminated Transformers	400,000	400,000	0
Railroad Transformers	6,000,000	6,000,000	6,000,000
Mining Motors	34,000	34,000	34,000
Hydraulic Systems	780,360	780,360	780,360
Carbonless Copy Paper	?	?	?
Pigments	6,700 /yr.	6,700 /yr.	6,700 /yr
Electromagnets	20,000 -30,000	20,000 -30,000	0
Compressors	Small	All	All
Small quantity R&D	Very Small	All	Authorized Use
PCB Capacitors/PCB Equipment	480,000,000	286,500,000	Authorized Use
Process Contamination	100,000 -600,000/yr.	100,000 -600,000/yr.	100,000
Dust Control, Sealants, Coatings from Waste Oil	32,000 /yr.	32,000 /yr.	32,000
	780,880,000	583,300,000	543,000

Reference: Federal Register/Volume 44, No. 108, Thursday, May 13, 1979, Rules and Regulations, P. 31515

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during the 18 month period of new rulemaking.

Above and beyond the inspection program, the utility industry will furnish EPA with data on utilities to add to new rulemaking. EEI/USWAG is undertaking the wide scale study. Data under study cover:

- Numbers and types of equipment that may contain PCBs.
- Typical distribution of PCB levels in that equipment.
- Frequency of fluids releases from equipment.
- Volumes of material lost.
- Feasibility of various mitigative programs: inspection/maintenance, secondary containment, and equipment phase-out.
- Health effects of PCBs vs. replacement fluid.
- Analysis of environmental pathways taken by PCBs from equipment.

Analysis of the Problem

In the next year, EEI/USWAG will be preparing a data base of information and analysis of PCB uses, practices, and economics, forming the bases for new regulations. The study will be performed over 9-12 months. The data requirements are extensive and the electrical utility industry sizable. Yet, the design of the study must be comprehensive enough to yield reasonable data for the benefit of the decision-makers.

In the conceptualization of a study, definition of the problem is a first step. One must comprehend and combine the source, forms and distribution of PCBs; the release mechanisms, exposure routes, health effects from PCB exposure, then evaluate the magnitude and significance of this compilation as the best summary that can reasonably be expected. More specific knowledge must be developed on the quantites of PCBs occurring where they reside, and the harm they present – short- or long-term. This information along with other considerations should form the building blocks for strategies and types of control to maintain a certain warranted level of protection.

Upon reaching a definition we must proceed to design an approach to solving the problem. Data requirements have previously been identified. They fit together in a flow of (Fig. 1) data inputs leading to interpretation and decisions.

On physical effects, a wealth of information, both published and unpublished, exists on human and environmental health, and environmental chemistry of PCBs. In general, it appears that PCBs, though linked to certain health effects in animals, wildlife and man, are only slightly toxic in the acutely toxic sense. PCBs persist in the environment and have a tendency to bioconcentrate in fatty tissues, but the chronic, long term significance of these facts appear uncertain.

If one examines the more recent literature on the trends of PCB levels in the environment, with the past control of bulk manufacture and open uses, a gradual, but marked decline has been observed. Studies performed on the Great Lakes and in other major surface waters, employing concentrations in fish as an indicator of PCB, document the decline. One must examine the period of PCB existence since 1929 and its consequences to begin to develop an understanding of its significance. Extensive studies of workers involved in equipment manufacturing have shown no adverse health effects. The exposure to levels experienced by these workers has far exceeded that of the general public.

Another emphasis of the health effects effort is to provide a common base from which to compare substitute fluids with PCBs and their respective and acceptable applications.

Limited information is available on substitutes for PCBs. Since PCBs were employed in equipment primarily to provide fire protection, few, if any, substitutes have been marketed that offer the same characteristics. If they do exist, certain environmental and health criteria must be met.

On PCB sources and distribution, the study plan includes refining and upgrading information on typical quantities of PCBs in present electrical equipment as summarized earlier. This effort will supply a more accurate insight into electrical equipment, making use of PCBs compared to that equipment which contains a fluid with low to non-detectable PCB levels.

The frequency and likelihood of the escape of fluid from electrical equipment will tend to identify the exposure of the environment to PCBs. Data on the types, frequency, corrective/preventative action and environmental setting of releases will be collected.

Later, release data will be evaluated opposite the health evidence and PCB distribution to help determine the magnitude and significance of episodic releases of fluids containing PCBs. Interest in this part of the study lies with the sources and concentration of material, pathways of PCB movement, the ultimate receptors of PCB exposure and the likely consequences.

Availability of approved PCB disposal equipment exerts a major effect on the ability to remove or retire electric equipment before its usefulness ends. In the case of disposal, the utility industry is deeply concerned about their ability to handle capacitor disposal. Only one incinerator is available to dispose of the nation's capacitors. Two incinerators, including the one already mentioned have been approved to handle PCB liquids. The options appear severely limited. Logistically, the transportation of PCBs seems the less desirable approach. Available technologies, new processes, possible mobile equipment and expanded use of high efficiency boilers on site will be evaluated in the interest of attempting to be helpful in speeding up the disposal. Similarly, the ability of manufacturers to supply new and replacement equipment and/or fluids will be addressed.

Various control strategies are under consideration. They have been limited to: (1) inspection, maintenance and secondary containment, and (2) phase-out or accelerated removal options. An effort has been made to outline, in general, the type of report which will be prepared for the EPA. Following an analysis of the data, EPA will initiate a rulemaking proceeding that will culminate in final regulations.

Final Observations

PCBs are one of many organic and inorganic chemicals to which man may be exposed. Each is essential or unwanted depending on the specific circumstances of its use and residency. Though specifically cited in TSCA for a ban, PCBs occur in the environment, limiting the provision of a PCB-free society. Similarly, there are limits to providing a risk-free way of life. Therefore, society must evaluate the devotion of resources to deal with an accumulative exposure to situations or materials based on: Can the effort necessary be justified and can the nation afford the required costs?

PCBs are not "acutely toxic," "deadly poison," "lethal" or a proven "cancer-causer." Evidence clearly demonstrates this case and accordingly the preservation of a right to question and seek improvement in regulations that may lead to the betterment of all.

Careful evaluation of the current protection under TSCA and existing regulations, review of the trends in PCB levels in the general environment must precede the setting of a future course for regulatory control. Finally, in a responsible way, assurance that workers and the general public are given all the necessary and current facts to see PCBs in a proper perspective is essential. ■

About the Authors

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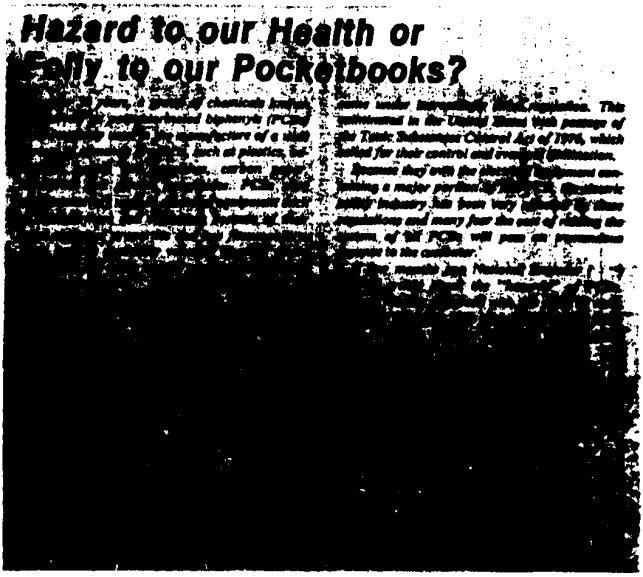

PCB White Paper

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PURPOSE

What we have done in this report is to take the arguments, and the facts behind those arguments, of our opponents (those who think PCBs are dangerous) from their own literature and we have presented both their arguments and conclusions to show that their conclusions are not justified by the facts available to them. By this method of reasoning we hope to show that the conclusion of the environmentalists that PCBs must be banned, or even more tightly regulated in transformers and capacitators than they are now, is based on *facts that do not support their conclusions.*

BACKGROUND INFORMATION

Production of Polychlorinated Biphenyls began in the U.S.A. in 1929 by Swann Chemical Company. In 1935, Monsanto bought out Swann Chemical and became the sole producer of PCBs for transformers and other uses. The reasons for this monopoly lay in the fact that the standards for purity as set down by General Electric Company — the first ones to apply PCBs to electrical equipment — were so high that no one else wanted to enter into the business. As a result of these standards, the PCBs produced in America under Monsanto Company's trade name of Aroclor® were the purest of any PCBs produced in the world.

PCBs were manufactured primarily because of their excellent dielectric and heat transfer qualities, not to mention their non-flammability. Not only did their non-flammability capabilities make them suited for use in capacitors, but also made them ideal for use in areas where public safety was concerned, such as, in transformers inside public buildings.

This set of uses, coupled with the unusual stability of the compound, have led to the calling of PCBs as the "wonder chemical of the century." They greatly aided America's industrial expansion since the early 1930's. It has been said that Russia's industrial successes could not have oc-

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Material Category	Purpose	Conditions	Life Expectancy	Limitations
Asphalt Emulsion with Fiberglass or Polyester Membrane	Water repellancy and reinforcement of aged asphalt BUR	Fracturing of top felt plys; underlying felts still sound	3 to 10 years depending on product quality, installation techniques and a reflective coating	Severely affected by trapped roof moisture; requires smooth/prepared surface; should not be used if deck movement is causing roof splits
Reflective White and Aluminum Coatings	Protect roof against U.V. exposure; reduce interior temperatures; extend serviceable coating/roof performance	New BUR or new coating/reinforcement systems requiring additional protection; energy conservation	1 to 5 years depending upon product quality and coverage rate	Strictly preventive design should not be utilized to solve roof deficiencies
Proprietary Coatings	Usually as preventive maintenance protection; some products developed as weathering seal and/or remedial capability	Wide variety of conditions depending upon product formulation	Varies widely; manufacturer's guarantee suggested (including labor to replace coating if defective)	Careful and thorough scrutiny of manufacturer's reputation, integrity, and proficiency advised prior to purchase
Cold Process BUR (Felts and Factory Formulated Adhesive)	Overlay existing roof that has aged beyond other corrective techniques	Sound deck and roof substrate; not more than one roof system on deck; existing roof cannot be correctively maintained	5 to 10 years depending on application techniques and specification quality	Severely affected by trapped moisture; should have insulation underlayment; preferably mechanically fastened to substrate; deck weight load capacity must be considered
Urethane Foam Systems	Lightweight resurfacing of existing aged roofing systems; provide or enhance insulation capability and benefits	Adequately sloped building requiring additional roof insulation capability; lightweight roof weatherproofing system is necessary	2 to 10 years depending upon insulation density, coating quality, and method of installation (manufacturer's guarantee advised including labor to replace system if defective)	Requires expert installation expertise; severely affected by moisture conditions during application; system is totally dependent on surface coating product quality
Single-Ply Systems	Overlay existing roof that has aged beyond other corrective techniques; provide lightweight system without roof removal	Sound deck and roof substrate; minimal roof projections; existing roof mat cannot be correctively maintained	1 to 10 years (manufacturer's guarantee advised including labor to replace system if defective)	New roof technology; first generation problems anticipated; usually requires expert installation knowledge; careful scrutiny of manufacturer's reputation, integrity, and proficiency advised

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Material Category	Purpose	Conditions	Life Expectancy	Limitations
Asphalt Emulsion with Fiberglass or Polyester Membrane	Water repellancy and reinforcement of aged asphalt BUR	Fracturing of top felt plys; underlying felts still sound	3 to 10 years depending on product quality, installation techniques and a reflective coating	Severely affected by trapped roof moisture; requires smooth/prepared surface; should not be used if deck movement is causing roof splits
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curred had it not been for PCBs. What then, went wrong?

In 1966, a Swedish scientist was trying to determine the level of DDT in fish. He found some unidentified chemical in his studies. This unknown chemical turned out to be PCBs. That PCBs had been found in the environment should have come as no surprise since they are so stable and they were used in such a multitude of products (e.g. paints, adhesives, caulking compounds, plasticizers, inks, lubricants, carbonless copy paper, sealants, coatings, dust control agents, heat transfer systems, hydraulic systems, etc.).

THE PRIMARY EVIDENCE

The big blow to PCBs, however, came two years later in Japan. It was reported that PCBs, as a contaminant, got into some rice oil, which was then sold to the surrounding population. This rice oil was used for cooking purposes for a period of approximately 100 days. Up to 1300 people were adversely affected from the ingestion of this cooking oil containing PCB. Those affected ate 720 ml. to 4.4 liters of oil containing 0.5g of the contaminant, with the average consumption being approximately 2g.[1] These effects were erroneously reported as being caused by the PCB.

The symptoms of this "oil disease" ("Yusho") have been described as follows: dermal disorders, including swelling of the eyelids, acneform eruptions and pigmentation of the nails, gums and lips and an increased discharge from the eyes was observed. The people also complained of things like headaches, indefinite stomach ache, soreness or pain in the extremities, coughing, and bronchitis-like symptoms. Gains in the height and weight of boys "decreased significantly" after ingesting this contaminated oil.[2] Babies born to women with Yusho had abnormal skin pigmentation and some anatomical abnormalities. However, "these were not permanent, and postnatal body and mental development appeared normal in these and other Yusho children."[3] (NOTE: All of these symptoms were alleged to be caused by PCBs. The allegations have proven to be false. But, on the basis of "Yusho," the Congress of the U.S.A. passed legislation to Regulate and Ban PCBs — TOSCA).

These, then were the illnesses associated with Yusho — illnesses that would make the world want to rid itself of the "rice oil contaminant" that caused all these problems. (This is especially true, since the results of the contaminants on humans, were graphically portrayed in a picture book). That contaminant that was first thought to cause the Yusho disease was first diagnosed to be Kanechlor 400, a Japanese PCB in the amount of up to 3000 ppm. Thus began the movement that eventually saw PCBs banned in Japan, Sweden, Canada and the United States.

THE DEFENSE

As is often the case with initial findings and the accompanying announcements, not all the facts in this case were in at first. Kanechlor 400 was present but it was not the most toxic (if it even is toxic at all) chemical involved. Polychlorinated Dibenzofurans (PCDFs) a partial oxidation by-product of PCBs, were found to be in concentrations of up to 500 times that normally found in Japanese PCB.[4] Not only this, but the tetrachloro- and pentachloro- fractions, which have through animal toxicity studies, been determined to contain the more toxic isomers, were found in high concentrations.[5] PCDFs affect the thymus, skin (acne), liver and hematopoietic system to a much greater degree than PCBs, and are 1000 times more potent as enzyme inducers.[6]

In addition to this, PCDFs were chronically retained in the livers of Yusho victims. Three Yusho patients eventually died (two in 1969, one in 1972); these deaths were not attributed to the ingestion of the contaminated PCBs. In the tissues obtained at autopsy, PCDFs were found in high concentrations. No PCDFs were found in the tissues of two controls, "although PCB concentrations of about 1-1.5 ppm were found in the adipose tissue and liver fat (of the controls)."[7] The PCB/PCDF ratio in the rice oil was about 200, while the ratio of PCB/PCDF found in the adipose tissues at autopsy were 2, 5 and 12, a considerable difference.[8]

By the end of 1977, 31 classified deaths of Yusho patients had been recorded. Eleven deaths were from neoplasm, which is 4.5 deaths more than would normally be expected.[9] However, this 4.5 could easily be zero or not attributed to Yusho, since the data were not age-adjusted.[10] In fact, a booklet prepared by the Hazard Evaluation System of the State of California, one of the strictest states with respect to environmental issues, cites a 1979 study (Urabe, et al.) "the evidence is not conclusive that PCBs caused these deaths."[11]

PCB LAW IS BASED ON "YUSHO"

Now is the time to see what is presently being said about the relevance of Yusho to the study of the toxicity of PCBs and their possible human effects.

We will begin this exercise with a quote from John F. Brown, Jr., PhD, Manager of the Life Science Branch of G.E.'s Corporate Research and Development as he explains the irrelevance of linking PCBs and Yusho together. ". . . it was the PCDF's that were chronically retained in the livers of the chronically-ill victims. In these victims, the average dose of the PCB/PCQ (Polychlorinated Quatraphenyls)/PCDF mixture ingested was 2.0g., which is less than the PCB body burden being carried by many seemingly normal (U.S.) capacitor workers today. Thus, it would appear that the toxic agent responsible for Yusho disease was PCDF, not PCB."[12]

Dr. Renate Kimbrough, M.D. a Toxicologist at the Center for Disease Control in Atlanta, Georgia, who has been one of the government's star witnesses against PCBs was involved in the following dialogue in a North Carolina court case in 1979:[13]

Question: But, Dr. Kimbrough, did you participate in the preparations of a report by a sub-

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committee of the Department of Health, Education and Welfare, July, 1976?

Dr. Kimbrough: Yes.

Question: And let me ask you if I'll read from that study and ask you if this is a fair statement. "The complexities and uncertainties associated with the most recent reports of the Yusho incident in Japan make it extremely difficult to quantify possible human health effects resulting from exposure to PCBs alone." In other words, Doctor, isn't it true that there is a strong suspicion now that the other chemicals that got into that rice oil other than PCBs are the things that caused the problems?

Dr. Kimbrough: They certainly caused part of the problem. We don't know whether they caused all of the problem, but they are very toxic.

Question: And by the same token we don't know whether PCBs caused any of the problems because of the presence of those other chemicals. I'm talking about scientific.

Dr. Kimbrough: In the Yusho incident, no."

A 1980 report issued by the State of California Research Division contains many such statements. One of them says that "it is therefore considered highly probable that some of the cause of Yusho toxicity is due to the actions of PCDF."[6] (This same report finally concludes this: "a review of the available literature on PCB, PCDDs (Polychlorinated Dibenzo Dioxins), and PCDFs indicates that exposure to these substances, even in microgram quantities, can lead to extremely serious human health effects."[7] What is the source of this convoluted reasoning? Their bibliography of 51 sources is constituted of five articles on PCB, all five sources deal specifically with the Yusho incident, and 46 articles deal with PCDDs and PCDFs. They admit that PCBs have no proven record of human health problems, but because they "look alike," PCDFs and PCDDs, they group all three together and call them bad!!! (NOTE: it is this type of reasoning that has led to the unreasoning and unreasonable pressure of the radical environmentalist that will eventually cost our country $4,000,000,000,000.00 — or $50,000.00 for every man, woman and child in the country today!!!)

A 1981 report prepared by the Hazard Evaluation System of the State of California states that "It is not clear how much the health effects observed in Yusho victims can be extrapolated to occupational exposures for the following reasons:

1. The average amount of PCB (Kanechlor 400) ingested was estimated to be 2 grams and the minimum, 0.5 grams (Kuratsune et al., 1971). This is a higher dose than has been reported in most occupational exposures. In addition, the PCBs were ingested as opposed to inhaled or skin-absorbed as is the case with occupational exposures.

2. The contaminated oil contained "used" (oxidized) Kanechlor 400, the exact chemical composition of which is unknown.

3. Frying of foods with the rice oil could have produced further new compounds which may have altered the toxicity of the PCBs or the toxicity of possible contaminants.

4. Yusho oil was shown to contain high concentrations (2500 ppm) of dibenzofurans.

5. Reported concentration of PCBs in the oil may not have been accurate enough to permit a rigorous quantitative analysis since the methods for estimating PCBs in foods were not fully developed at the time."

The National Institute for Occupational Safety & Health (NIOSH) published a criteria document on PCBs in 1977. They say that:

The relevance of the Yusho episode to occupational PCB exposure is compromised because: (1) the oil was ingested; and (2) it contained large concentrations of dibenzofurans compared with those in the PCBs to which workers generally have been exposed in their occupations. Its relevance is further compromised because the effects observed from daily ingestion of 1-15 mg of PCBs (165, 167) were peculiar and excessive compared to those observed in workers exposed by inhalation of PCBs 1-5 mg/M³ (120, 125), notwithstanding that the amounts absorbed may have been similar.

Nevertheless, information obtained from the Yusho episode is relevant to the study of PCB toxicology and occupational exposure. The information established that PCBs can be transmitted from mother to fetus, and, in the milk, from mother to child. It also establishes that some PCB compounds are eliminated from the body relatively rapidly, and that others may require years for elimination."

After NIOSH came to this conclusion that Yusho could not be used to determine PCB toxicity, they turned around, and in the same document, state that the "toxic effects from ingestion of PCBs have been well documented in humans."[8]

The authority for this statement? Forty footnotes — every single footnote referring to the early Japanese studies of the Yusho incident, when all of the effects were erroneously attributed to PCBs.

EPA — THE AGENCY TASKED BY CONGRESS TO CONTROL PCBs

Finally, what has EPA said about Yusho? Please note the dates as you read the conclusions reached.

EPA 1975 — Subsequent investigations (have shown that the "Yusho Oil") had been more than half pyrolyzed to PCQs, and that this PCB-PCQ mixture contained 0.25% (2500 ppm) of PCDFs. It was the PCDFs that were chronically retained in the livers of chronically-ill victims.

The same oils were reanalyzed in 1976.

EPA 1977 — Because of the relatively large exposure of the Yusho victims to PCDFs it is inappropriate to use this data to make quantitative estimates to the TOXIC hazard posed by PCBs to humans.

Compare these first two statements with EPA's 1979 statement.

EPA 1979 — Knowledge of PCB toxicity is

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based primarily on the Yusho Japan incident in 1969. (NOTE: Why has the EPA contradicted itself here, and take a position contrary to all recognized scientific evaluations of Yusho?)

Does not this last statement (when compared with EPA's earlier statements and the statement by all the "authorities" that a determination of PCB toxicity from Yusho is scientifically inappropriate) tell us that the case against PCBs, is "based primarily on no evidence at all?" This is what EPA has said, whether wittingly or not. The evidence is here. Our rules regulating PCBs are based on this fallacy! This is what the Congress must know; this is what the public should know: that PCBs are not a health problem to humankind!

In 1976-77, Congress passed the Toxic Substances Control Act (TOSCA). The only specific chemical listed in this law was PCBs — based upon one senator's amendment to the original TSCA law because of what happened in Japan in 1968 and to political pressure brought about by publicity of the event. This law prohibited the sale, distribution or manufacture of PCBs after 1979. Monsanto, the only U.S. manufacturer, ended its production of the chemical in 1977. After that time, PCBs were to be controlled. The EPA ended up with this regulation job, and set down rules which, in part, said that transformers with less than 50 ppm PCB could be considered a non-PCB object. The radical environmentalists, working through the Environmental Defense Fund (EDF) sued EPA for allowing any ppm of PCB. (EDF vs. EPA DC Cir No. 79-1580 Act 501980, October 30, 1980). The court ruled in favor of the EDF and said that Congress mandated a ban (0 ppm) on PCBs in transformers and capacitors. EPA has said that transformers and capacitors were "closed systems" but the court ruled in favor of EDF, recognizing that transformers and capacitors do sometimes leak or fail. EEI (Edison Electric Institute), as a friend of the court, told the court that the ruling left the electrical industry of America two choices; they could either operate illegally (which they would not do) or they would have to shut down all operations immediately. The court decided that this might not be such a good idea so it gave EPA 18 months, till October, 1982 to address these two questions:

1. PPM level of PCB to be regulated; and
2. Rules of operation of "totally enclosed" devices containing any PCBs.

Since Congress passed TOSCA, identifying PCBs as a priority pollutant to be regulated, the EPA must act on the mandate of Congress. Therefore, the solution to this problem must go back to the source of the problem: Congress. Congress must be made aware of the growing body of literature that completely exonerates electrical grade PCB from the health and environmental problems it has allegedly caused.

WHY ANIMAL STUDIES ARE USED

The consensus of those who study PCBs are in agreement that the Yusho event cannot, scientifically, be the basis for condemnation of PCBs. What, then, is the basis for the ban on PCBs? A review of the literature alludes to a lack of epidemiologic evidence to support their position. (Note: They are willingly ignorant of over 15 epidemiologic studies!!! And the reason why they are willingly ignorant of these 15 studies? Because they can not find any evidence — anywhere to support the conclusion they have predetermined to make! That's right. If they can't find evidence to support their conclusions — let's "create our own evidence." Hence — animal studies!) Therefore, the people fighting PCB turn to animal studies to build their case.[1] Due to the superabundance of animal studies, it would be repetitious and hence boring to cite all those studies here. We will concern ourselves, therefore, with looking at the results of the more representative and widely cited studies.

ANIMAL DOSAGES

In order to understand the results of these animal tests, a comparative knowledge of dosages is needed. The NIOSH criteria document cites a 1975 paper presented before the EPA in a national hearing on PCBs as estimating that the "general U.S. population has a dietary intake of PCBs of between 10-20 μg/day."[2] Inhalation of the air, NIOSH says, may add up to another 7/μg/day (100μg/m3 x 50 liters/min. . . .). Taking maximum figures, an average person in the U.S. will take in 27/μg/day, or for a 154 pound person, or approximately .39/μg/kg of body weight/day. Whichever figures are used, it still works out to be about 0.01 grams intake/year. Now compare this with the amounts fed these animals.

Dr. Renate Kimbrough, the toxicologist widely quoted by environmentalists concerned about PCBs, has done extensive testing on rats and mice. In one experiment in 1975, Dr. Kimbrough and Dr. Linder experimented on BALB/cJ male mice. The mice were fed Aroclor 1254 over eleven months. The average PCB intake was 49.8 mg/kg/day; the equivalent of over 2.5 pounds for an average person, or 129,111 times what one, at the maximum, would be expected to eat. Adenofibrosis was identified in the livers of these mice. "These lesions, according to the authors, may or may not be precursors of malignant lesions."[3]

In the following year, Dr. Kimbrough fed Aroclor 1260 (60% Chlorine, by weight; Aroclor 1242 would be 42% chlorine, by weight) to 200 rats in their diet for 21 months. The amount consumed was from 5-10 mg/kg/21 months.[4] This is 20-40 times what an average person (154 pounds) would consume in that same period. On 170 rats, elevated nodules were found on the livers. Twenty-six of these were hepatocarcinomic (compared to one in the control group) and the other 144 had "hepatocellular nodules that were described as characteristic of neoplastic nodules or synonymously, hyperplastic nodules. The authors said that 'neoplastic nodules are part of the spectrum of response to hepatocarcinogens and must

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be included in the evaluation of tumorigenesis.' "[5] Hence came the cry that PCBs might cause cancer (Note: that is, cancer of the liver). (see page 10). Monsanto had previously conducted tests with Aroclor 1260, as well as with Aroclors 1242 and 1254 in which liver injury was produced, but no cancers were seen. They, therefore, did a reexamination "of the original liver slides from the Monsanto studies as well as additional liver sections from all rats in the Monsanto 2-year feeding studies."[6] The slides were independently examined by Prof. W. Richter of the University of Chicago, by Dr. D. Gordon of Industrial Bio-Test Labs, and by Prof. Parvis Pour of the Eppley Institute for Research in Cancer. Dr. Pour also examined sections of liver from the rats in Dr. Kimbrough's study. No carcinomas were seen in Monsanto's rats, and Dr. Pour did not agree with Dr. Kimbrough's diagnosis of liver cancer in her rats." So, at the very least, this study of Dr. Kimbrough's that has been cited as proof of the potential carcinogenicity of PCBs has to be viewed with serious reservations.

Another 1974 study done by Linder, Gaines and Kimbrough involved feeding rats Aroclor 1254 at 5 ppm or Aroclor 1260 at 100 ppm level. The study covered a "two-generation period" and no effects on reproduction were observed."

Another of the "big guns" of the environmental groups opposing PCB use is Dr. J. R. Allen. Although he has also done studies on rats, he and his co-workers are cited most often with respect to their experiments on rhesus monkeys. In their 1973 experiments, Aroclor 1248 or Aroclor 5460 was fed to the monkeys for 12 weeks. Each set of monkeys had access to 400g daily food supply laced with 300 ppm and 5000 ppm of PCB, respectively. This means that in a 23 week period, these monkeys received 10g and 168g, respectively. It would take a U.S. resident 1,014 and 17,047 years, respectively to consume this much PCB. The results for both groups will be put together for this one part. The monkeys lost, on an average, between 19 and 26% of their body weight, they lost hair, had puffy faces, edematous lips, and swollen eyelids with purulent exudates around the eyes. At necropsy, extensive alopecia, acneform lesions of the skin, subcutaneous edema and liver hypertrophy with fatty infiltration were evident."

Another study by Allen on 12 female rhesus monkeys fed 26 ppm Aroclor 1248 in an unspecified diet turned up some reproductive problems. The average weight of the monkeys was 5.6g. If these monkeys ate 300g of food a day, two-thirds of the amount used in the experiment on 1973 monkeys, then after the two month feeding time, they consumed about 457.5 mg, or 1.34 mg/kg/day. This is equivalent to 3472 times what a 154 pound person would normally eat."

Allen's 1976 study, done in conjunction with Deborah Barsotti, has been cited as an example of what will happen with low doses over a long time period. At first glance, it would seem to fit that category, for they fed the rhesus monkeys 2.5 and 5.0 ppm Aroclor 1248. But then it turns out that the total amount of PCBs fed the female monkeys was 180 mg and 364 mg respectively. This was over a six month period. Taking 6 kg as the average weight (in the 1974 experiment, the average weight was 5.6 kg — 6 kg will make the numbers more conservative). This works out to 30 mg/kg/6 mos, some 426 to 862 times the amount an average U.S. resident would consume. In fact, it would take a 154 pound woman 213 years to consume that much (adjusted for weight) given a normal diet — well past a woman's normal childbearing days. And that was what this study tested for: Reproductive problems. Conceptions did occur, some abortions, a still birth and other various problems also occurred. The babies that did survive showed signs of PCB toxicity after 2 months. (Acne, swollen eyelids, increased skin pigmentation)."

The following animal studies have been included in this paper not because their results or test methods show much deviation from the previously cited studies, but because NIOSH includes them in their criteria document and we wish to give our opponents' arguments enough exposure so that they cannot criticize our position by saying we blew down a straw man.

Allen, 1975, rhesus monkeys. Gave them a single 18 mg/kg dose of the above mixture. It would take an average U.S. resident 128 years to consume this much PCB. "No overt clinical effects were seen in the treated monkeys . . . Only the adipose tissue and adrenals had high PCB assays, and microscopically, except for a moderate proliferation of hepatocytic SER, all tissues were normal."[8]

Bruckner, 1973, rats. Gave them a single dose by oral intubation of either 2.5 g/kg or 6.0 g/kg Aroclor 1242. A 2.5 g/kg dose for a 70 kg man (154 pounds) is 4.6 ounces, or 175 g. Both groups got sick and the 6 g/kg group eventually died. "The conditions of the rats receiving the lower dosage gradually improved after the first 24 hours and was normal at the end of 72 hours."[9]

Barczy, Cobb and Cherry, 1974, rats. This was an inhalation study in which the rats inhaled air that contained 2.54 mg/liter PCB (2500 times the allowable amount for humans), for six hours. "During exposure there were repeated episodes of blinking and sneezing. Signs of irritation disappeared after cessation of the exposure. During the subsequent 14-day observation period, food and water consumption and growth of the rats were considered by the authors to have been similar to those of the controls. No gross pathological changes were seen when the rats were killed 14 days after removal from exposure."[10]

These next three studies can be found in a booklet published by the State of California. No mg/kg amounts were given in the reports, and no standard for determining that from PPM seems to work adequately in those cases where both are known, so only PPM figures will be given. Anyway it is the results of these tests that are important.

. . .

Conclusion to appear in September issue.

MONS 003899

PCB White Paper — Pt II

By: Dana S. Myers
Stanley D. Myers
J.J. Kelly
PCB Information Center
S.D. Myers, Inc.

The National Cancer Institute, 1978, Fisher 344 rats. Groups of 24 eight-week-old rats of either sex were fed Aroclor 1254 at dietary levels of 0, 25, 50 or 100 PPM for 105 weeks. Their report stated that Aroclor 1254 was not carcinogenic under test conditions."

Norback and Weltman, 1980, rats. They fed Aroclor 1260 to Sprague-Dawley rats at dietary levels of 0 and 100 PPM for 105 weeks. The study concluded that this mixture was "carcinogenic in female rats, causing significant increases in liver hepatocellular carcinomas."[6] (Note that it was the liver that had the carcinomas — see page 10).

Treon, 1956, 10 mice, 6 guinea pigs, 4 rabbits and a cat. This was an inhalation test. Each set of animals was exposed for 7 hours/day, 5 days a week at differing concentrations and different mixtures. The group inhaling Aroclor 1242 did this either at 8.6 mg/liter or 3 weeks, or 1.9 g/liter for 31 weeks or 6.83 mg/liter for 17 weeks. The groups inhaling Aroclor 1254 did this at either 1.5 μg/liter for 31 weeks or 5.4 μg/liter for 17 weeks. The standard for workers is 1 mg/m³ which is the mathematical equivalent of 1 μg/liter.

FROM NIOSH STUDIES

Torok, 1976, Pregnancy in NMRI mice. Group one was a control group, group two received 375 mg/kg/day for three days and group three received 750 mg/kg/day for three days. In other words, these mice received in three days, from 7,990 to 15,900 times what a 154 pound woman would ingest in one year. The result was "longer intervals from breeding to parturition (birth) . . . (and) reductions in the number of dams with litters and in the mean litter size."[6]

No consistent changes in mortality, growth, pathology, organ size, liver function, or hemotologic parameters were found in animals exposed to Aroclor 1242. The animals exposed to Aroclor 1254 vapors showed no changes in growth or mortality but microscopic evidence of apparently reversible hepatic cellular injury was found in all species but the cat. At both exposure levels, enlarged livers were found in the animals exposed at 5.40 μg/liter."

These, are a representative sampling of the animal studies conducted which involve PCBs. Animal studies are one of only two arguments (excluding Yusho) that have been put forward in defense of the position that PCBs should be banned. It is therefore of the utmost importance to determine what relevance these animal studies have to the human population. This is especially so with regards to the four inducing potentially carcinogenic label that gets pinned on PCBs. (Note: Of the scientific reports we have read none say PCBs are carcinogenic to humans, only the media has pronounced them so — see addendum).

	EPA MSDHP	HMS$_A$	HMS$_B$	50 kg	70 kg	100 kg
	Percent of Test Subjects					
NONE	— —	24.5	9.1	0	0	0
1 PPM	69 56	40.2	50.8	.06	.07	.1
1-2 PPM	31 36	29.8	35.4	.1	.14	.2
2 PPM	— 9	5.5	4.9	.5	.7	1

ANIMAL STUDIES AND RELATION TO HUMAN BEINGS

A 1980 study by the California Research Division of PCBs had this to say:

The biggest difficulty in using animal studies to determine potential health effects is in extrapolating toxicological results from animal species to humans. It is often seen that two types (species) of animals do not react the same way to the same chemical."

One good example of this was seen in a study with TCDF (Tetrachloro Dibenzo Furan), the contaminant in the Yusho incident. The single oral LD_{50} (lethal dose for 50% of test subjects) in guinea pigs was estimated at between 5 and 10/μg/kg body weight while single oral doses of 6000 μg/TCDF/kg body weight did not produce any toxic effects in mice."

The 1981 California study states that "the effect of PCB exposure on immune and endocrine system function has not been carefully studied in humans. So the relevance of these animal observations to human health remains unknown."[6]

The National Academy of Sciences Division of the National Research Council has also called into question the relevance of animal studies to human health. The following quotation was with respect to TOSCA's testing results.

It should be noted that most of the chronic tests proposed to assess human health hazards require use of species (rodent-plus one other mammal) that would not adequately reflect the human toxic response to PCB exposure. The common laboratory test animals (rats and dogs) do not always respond to PCB exposure in the same manner as humans, and Table 3.6 suggests that no one species exhibits "typical" human symptoms of PCB poisoning except in the case of reproductive tests."

Note: In regards to this statement, consider the fact that penicillin kills guinea pigs." Should penicillin be banned because of this? Aspirin administered to rats causes birth defects in rats." Should aspirin be banned because of the rats?

Dr. Leonard Goldwater, M.D., an expert in the field of toxicology gave a courtroom his opinion of animal studies. "Animal studies tell you what happens when you give chemicals to animals."[7]

MONS 003900

• Oil Assistance and Treated Coal

To prevent dusting in transportation, coal is frequently sprayed in the cars. We have found that up to 2 gallons per ton of #2 oil has no effect on the baghouse. Residual oil or used crankcase oil should definitely *not* be allowed. Coal treated with this is difficult to burn and will effect the baghouse. In winter ethylene glycol is frequently used, and this does not seem to have any effect.

Many times it is necessary to burn oil along with the coal, for instance during flameout. This is acceptable. However, many customers ask "How much oil can I burn continuously?" There has not been much experience with this on baghouses. We would recommend oil not to exceed 5% of the thermal capacity of the system until enough operating experience has been accumulated to verify the percentage.

• Bag Cleaning Problems

Probably the most aggravating problems with baghouses is high pressure drop. When this occurs it could limit boiler capacity because the I.D. fan may not have enough capacity to overcome the increase in pressure. The problem can be caused by a number of things.

We have previously discussed the verification of flue gas volume under "other variables." Undersized equipment due to this problem will require additional cloth or a larger fan to solve the problem. However, during normal operation occasionally this problem can occur. In a reverse air baghouse a malfunction of the reverse air fan may trigger the high pressure drop. If for some reason the fan has stopped, cleaning will not be accomplished and just putting the fan back on line may not rectify the situation. The baghouse pressure may be so high that the fan can not overcome it. In this case the baghouse must be placed on bypass and cleaned.

Improper bag tension can cause cleaning problems. This should be checked periodically. Smoking boiler conditions can generate very small particles that imbed themselves in the cloth. Off line cleaning will often remedy this. On rare occasion the wrong type material to "seed" the bags prior to start-up can cause high pressure drop. Lime should never be used. We recommend fly ash, but even then the calcium content must be low.

• High Temperatures

Many installations are adding economizers to conserve energy. These are also necessary to prevent temperature entering the baghouse from exceeding 500°F. The finish on the fiberglass bags deteriorates rapidly above this temperature. The finish protects the bags from acid attack and also is a lubricant for long bag life.

Failure to have feed water or other economizer problems can allow high temperature flue gas to enter the baghouse. An automatic bypass is very strongly recommended to prevent bag failure.

High temperature is not a problem for electrostatic precipitators. Temperatures up to 700°F. may be handled with standard design units.

Conclusion

This article has concentrated on experience and problems. We hope you don't assume that every project has problems. This is not the case. This article will hopefully present ways to learn from the past and incorporate what we learn to future projects. The best way to avoid problems is to have a great deal of past experience. We would recommend you investigate your supplier and verify that he has a number of successfully operating projects. □

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Acknowledgement

The author thanks Jack Riley, Product Manager of E.P. Systems with American Air Filter Company, for his review and comments on this paper.

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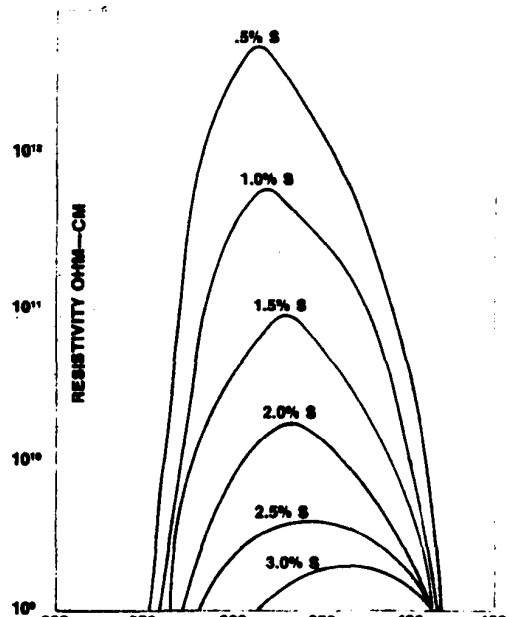
This article was presented at the Twenty-First Annual Kentucky Industrial Coal Conference, University of Kentucky, April 1982.



FLYASH RESISTIVITY
FIGURE 3

MONS 003901

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Conclusion

This article has concentr
problems. We hope you
project has problems. Th
ticle will hopefully preser
past and incorporate wha
ects. The best way to avo
great deal of past experi
mend you investigate you
he has a number of successfully operating projects. □

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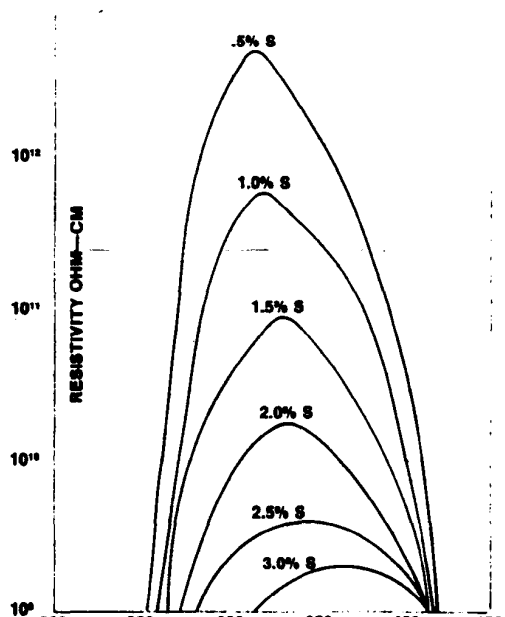

FLYASH RESISTIVITY
FIGURE 3

MONS 003902

STUDIES ON HUMAN BEINGS

It is with this recommendation that studies of the human population be included in any decision that we begin our next section.

Before examining specific cases of human contact with PCBs, however, it would be good to look at the risk to the population as a whole. This can be done by first determining the amount of PCBs that are in the population as a whole. The following three paragraphs describe studies done that determine the PCBs content of fat. The findings are summarized in a table following the paragraphs. It shows the percentages of people falling within each grouping.

The EPA's Human Monitoring Survey analyzed human adipose tissues, a major deposit area, from 1968 to 1972."

Price and Welch, 1972, describe the findings of a study done by the Michigan State Department of Health Pesticides (MSDHP) on over 4000 human adipose tissues. They also mentioned finding 100-250 ppm in the adipose tissues in an autopsy of a 77 year old man. The highest PCB concentration was in the liver."

The Human Monitoring Survey (HMS) examined 1277 (A) adipose tissues in one year and 1047 (B) in another. The years ran between July, 1972 through June, 1974."

The right side of the above table gives the figures for how much the PPM translates into for a 50, 70 and 100 kg person. The numbers are in grams and were calculated using the high figure, i.e., 1 and 2. The important things to note from this table are: One, the low amount of PCBs present in the body; and Two, that over 90% of the U.S. population has less than 2 ppm PCB.

The National Academy of Sciences has estimated that the mean concentration of PCBs in human adipose tissue of U.S. residents is 1.2 mg/kg. For a 154 pound man, that works out to 0.084 g. Compare this amount to what a 154 pound West German carries, which is over six times that amount: 0.55 g.

As can be seen, the U.S. has one of the lowest mean concentrations of PCBs in the industrialized world.

These figures also provide a check for the NIOSH figures for daily PCB intake that have been used for comparative purposes in the section on animal studies. NIOSH said the dietary intake was on the order of 10-20/µg/day and intake from inhalation up to .1 µg/m³. We used 27 µg/day as the high figure. Fifty years of intake at this level would yield a body burden of 0.49275 g. For a 154 pound person that works out to 7 mg/kg, or 7 ppm — almost six times higher than the average. Which means that the estimated daily intake of 27/µg/day is either too high or else the PCBs are leaving the body. So, if we have misrepresented the comparative amounts in the animal studies, we have certainly done so on the conservative side.

Polychlorinated Biphenyls have also been found in mother's milk, a fact that some try to exploit for sensational purposes. The most comprehensive study we have thus far encountered was done by E. P. Savage, who reported his findings to NIOSH in 1977. One hundred-forty-one

TABLE 1.24 PCBS IN HUMAN ADIPOSE TISSUE

Country	Mean Concentration (mg/kg)
Denmark	5.0
Japan	4.7
West Germany	7.9
Austria	4.6
U.K.	1.3
Norway	0.9
Canada	1.2
East Germany	6.4
Israel	3.6
New Zealand	0.9
U.S.A.	1.2

human milk samples from 40 states were tested for PCBs. One hundred-twelve samples contained over 50 ppb, (parts per billion) with the highest being 350 ppb. (Caution should be exercised when looking at milk figures because many times they list the findings on a *fat* basis, which is less than 3% of the milk. This way they can put down a bigger number to evoke more fear. For example, on a *fat* basis, the 350 ppb in the milk translates into 12.6 ppm.)" After discussing the PCB level in human milk and adipose tissue, NIOSH concludes with the following statement, giving 12 sources from which it made this decision.

"While no adverse effects have been associated with PCBs at the concentrations found in adipose tissue, blood, or milk of individuals whose only known exposures were from environmental concentration, knowledge of these concentrations is important to the evaluation of reports on occupational exposure."" Further at a Toxic Substance Control Institute Seminar October 15-19, 1979 Raymond D. Harbison, Director of the Toxic Substances Control Laboratory at Vanderbilt University — said PCBs are totally immobile and are not carcinogenic to humans."

INCIDENCE FOR HUMAN EXPOSURE

Since PCBs in the environmental levels present have caused no adverse effects on human life, what segment of the population might have cause for concern? Those workers who are occupationally exposed. NIOSH cites a 1978 study, when PCBs were still being manufactured. In its report it states "that 12,000 workers have potential occupational exposure as a result of current uses of PCBs in their working environment."" (Note: Production of PCBs was terminated in 1977). To better understand the potential risks involved let us compare this figure with some other risk numbers. The American Cancer Society estimates that 78,750 persons will die of lung cancer caused by smoking in 1981. These are real deaths and are six times more numerous than a "potential occupational exposures."" For some other examples, let's translate the 12,000 into a per 100,000 basis. It works out to 8.3. FBI statistics from 1976 state that a person has 66% more of a chance of being murdered (and by non-negligent manslaughter) than he does of being "potentially-occupationally exposed" to PCBs." By 1976, the deaths of

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Whenever policy is being determined, the risk factor must be taken into account. Life is filled with risks and nothing is ever risk-free. Even if PCBs killed on contact, the associated risks are less than those when compared with deaths that occur from such social activities as drinking and smoking. But that argument does not really matter, for PCBs have never caused one death in their fifty-plus years of existence. In fact, American PCBs have never caused one illness,* except when they were heated. And even then there were only reported cases of chloracne — a skin disorder not unlike teenage acne. And this was attributed to contaminants in the compounds used to make the PCBs — not the PCBs themselves.

EPIDEMIOLOGIC STUDIES (15)

Our opponents in this matter have said that not enough epidemiologic evidence exists on which to make a decision of PCBs.[70-71] So they utilized animal tests. Below are over 15 epidemiologic studies that show why animal studies were needed by the radical environmentalists: not one serious illness could be attributed to PCBs.

The first epidemiologic study we would like to cite was conducted by Dr. Leonard Goldwater, a toxicologist working at Duke University and the University of North Carolina, with the EPA and with NIOSH. His observations of people who worked with PCBs came out in sworn testimony before a court of Law. The observations relate to his studies done for the Navy in 1943. The studies were conducted in four major cable factories (two in New York City, one in Rome, New York, and one in the San Francisco Bay area).[72]

In one of these plants in particular they were very, very sloppy in their work, and they were under great pressure to turn out as much cable as possible, they were working a lot of overtime and working around the clock three shifts a day, and the air was thick with the fumes from these things because they were heating them to impregnate the cable, and the workers were practically bathing in this stuff. They were not provided with any adequate shower facilities or work uniforms and they literally had their skins caked with the condensed insulating material which contained PCBs. Now, these are the ones who had the main difficulties. In one of the other plants — another extreme — they were careful, they had adequate ventilation, they had showers, they had work uniforms. There they had practically no problems at all.[73]

The "main difficulties" the people had was chloracne.

He also examined 3000 electricians in the Brooklyn Navy Yard who put this cable on the ships.

And they were examined for evidence of acne, chloracne — which were mentioned here the other day — and for evidence of any liver injury, which is the main organ that one would expect to be affected if there was any effect; and, just by examining these people and assuring them that nothing was happening and then re-examining any who were nervous about themselves, all of this fear and almost hysteria that had been whipped up by the news media very quickly subsided and the shipbuilding effort resumed with the results we all know.[74]

Capacitor workers probably received more exposure to PCBs than workers at PCB manufacturing plants did. A 1980 retrospective cohort mortality study of 2349 workers in two capacitor manufacturing plants was recently completed by NIOSH. The total exposure of these men was 38,890 "man-years." Some of the workers had 38 years on the job, some as little as three months.[76-80]

The major organ checked for carcinogenicity and other such problems in the animal studies was the liver. Therefore, one would expect that if any serious health problems were connected with PCBs, they would show up in the liver. This, however, is not the case. A twenty-five year study on different cancers by the American Cancer Society shows that from 1950-52 to 1975-77 deaths from liver cancer has steadily decreased. For men it has gone down by over 38% and for women by 53%.[4] All this while the production of PCBs was increasing. If PCBs were as bad as the environmentalists claim, why should liver cancer be decreasing?

Many epidemiologic studies have tested for impaired liver functions. Most deal with increased levels of enzyme production, something that occurs when a foreign substance enters the body. (Liver enzyme production is triggered, for example, by nicotine in cigarettes, charcoal-broiled meats with benzopyrene on it, birth control pills, BHT preservatives that are widely used in food, brussel sprouts, cabbage, and even stress.)[81] So what happens to workers exposed to PCBs? The next studies should reveal the answer to that question with respect to liver function.

HUMAN LIVER STUDIES

Puccinelli, 1954, Italy. Men were exposed to heated Aroclor 1254 at a factory that produced capacitors impregnated with the chemical. This Aroclor was present in concentrations from 5-7 mg/m³. (International standard is 1 mg/m³). Three men, ages 18-24, developed chloracne after working in this area for 2-4 years. "Other than chloracne, the men appeared healthy, and all findings, including liver function tests, were reported normal."[82]

Karppanen and Kolho, 1972, Sweden. Six persons handled PCBs in an analytical lab and 12 worked in a plant where Aroclor 1242 was impregnated into capacitors. The capacitor workers had been at their job for 4 years. Special care was given to protecting their skin and the workroom air met internationally accepted standards (1/mg/m³). Subcutaneous adipose tissue was taken from two lab workers. The capacitor workers had the highest concentration of PCBs in their tissue, having 160, 285 and 635 ppm. "All persons examined were in good health. The capacitor workers had been under special medical observation but the investigators were unable to detect any biologic effect from the PCBs."[83] No

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liver impairment was found.

SKIN EFFECTS

If the liver is not adversely affected, then what is? The skin, sometimes. In addition to the finding or non-findings of dermal disorders already mentioned in connection with the search for liver disorders and in Doctors Goldwater and Kelly's testimony, the remainder of cases of chloracne will presently be discussed.

In 1969, a 43 year old man got severe chloracne. He had put his hands in a PCB "mixture without skin protection for a long time . . . his clothes often became impregnated with the PCB. It was reported that this was the first case of chloracne in the plant. Where more than 100 workers had been engaged in the process for more than 20 years."[9]

In 1964, 13 of 15 workers exposed to an Aroclor which was a mixture of bi- and terphenyls (65% chlorine content) developed chloracne. "An enamel containing the Aroclor was painted onto glass and then baked in an oven. Faulty ventilation caused contamination by chlorinated hydrocarbon vapors."[9] Note that this was not an electrical-grade PCB.

The General Electric Company gave testimony in 1975 about what its records showed concerning persons working with PCBs (in the matter of General Electric Company, File No. 2833, New York State Department of Environmental Conservation). The testimony related to two manufacturing facilities that used PCBs. Their records showed that, during the previous 15 years, only 49 cases of skin rashes and dermatitis has been reported to the dispensary. Of these 49 visits, 22 were second visits. Of the 49 complaints, only the fingers and hands were involved in 21 of the cases, only the arms, and hands in 5, only the face and legs in 1. Fourteen complaints were about a generalized skin rash. On sixteen other occasions, things like a burning sensation in the eyes and nose, dizziness and nausea were reported. This latter group of people, along with those who complained twice, were taken off the job.[9] Think about these figures for a moment: Only 49 complaints in 15 years involving hundreds of people.

In 1950, Elkins reported that although the average concentrations of PCBs in the workroom air of several plants in Massachusetts ranged from 0.1 to 5.8 mg/m³, with maximums of 0.2 to 10.5 mg/m³. "No evidence of immediate toxic effects was observed except at PCB concentrations approaching 10 mg/m³, which the workers found unbearably irritating."[9]

The 1943 study by Karppanen and Kolho of Sweden, mentioned earlier, turned up no cases of chloracne. The subjects of the study had blood PCB levels of 74-1000 ppb and had been exposed to PCBs for 4 years in capacitor-impregnating operations.[9]

It should now be clearly evident why animal studies are so widely quoted by those trying to ban PCBs. No evidence, along human lines, exists to support their position, and this after 50 years of exposure to PCB under the worst possible conditions. Animal studies, they claim, are needed to determine the long-term effects (they are looking for cancer) of exposure to PCBs. But not only are the results of the animal tests extremely questionable in their relevance to human experience, they are unneeded. For (one), a long-term mortality study has been done (30,000 man-years of exposure, with a maximum of 38 years of exposure, seems pretty long-term to us) — probably the reason the results have not been very widespread related to its finding of "no long-term effects from exposure to PCB," (two), over a twenty-five year period beginning in 1950-52, deaths from almost all cancers had decreased, and liver cancer, the expected site for PCB-caused cancers, has decreased between 39 and 53%. (The exception, lung cancer, which has increased around 200%. The "increase seen in both sexes due to cigarette smoking.")" So if PCBs do cause cancer (which we are not in the least even intimating) then the number of cancers caused must not be very significant. The long-term effects of PCBs on humans is that literally thousands of people in the United States have worked with PCBs since 1929 with no evidence of health problems whatsoever. If the object is to determine deleterious effects of PCBs, then the simple thing to do would be to make a study of those people who died who worked with the PCBs and see what they died from.

These figures could then be compared to national average and we could see exactly how harmful this chemical is. As it is, however, the only such story published completely exonerates PCBs.

With respect to the carcinogenic evidence against PCBs, NIOSH gives what turns out to be the basis for the demands that PCBs be banned: "In humans, there are no adequate studies to confirm or deny carcinogenicity. . . ."[9]

Our claim about the foundation of their case will be discussed later, but first let's finish the quotation:

"Although preliminary data suggested that among Yusho patients, deaths due to cancers exceed expectations (168, 161) and preliminary studies of two occupationally exposed groups in the U.S. indicate that the occurrence of certain cancers may be excessive (H.A. Sinclaire written communication, June 1976; G. Roush, written communication, September 1976). However, the two reports are not consistent as to the types of cancers found to occur more frequently than expected."

Sinclaire searched through the records of a petrochemical facility for employees who were "likely to have been exposed to Aroclor 1254 for various periods between 1949 and 1957." The 8 cancers that occurred out of a group of 92 "were not significantly more than would be expected," but the 3 melanomas and the 2 pancreatic cancers were significantly different from what calculated expections."

Two observations about this study would be appropriate at this point. First at a petrochemical factory there are many other chemicals present

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and it would therefore be both unreasonable and unscientific to use an example such as this to make any judgment on the carcinogenic potential of PCBs. Second, even in the Yusho incident, where all those other contaminants were present "there were no deaths from malignant melanoma, a tumor previously suspected to be linked to PCB exposure."[100]

Roush studied 300 current and former employees engaged in the manufacture of PCBs and found no malignant melanomas or pancreatic cancers. There was an excess of lung cancer, but these were not corrected for age or smoking habits.[101]

This sums up the epidemiologic evidence of what PCBs do to human beings. NIOSH says that "since 1970, PCBs have been one of the more thoroughly investigated environmental and occupational hazards."[102] This was in 1977. As late as January of 1981, however, statements such as "since the published epidemiologic evidence is limited we have utilized animal toxicology studies where appropriate in anticipating potential biologic effects in humans."[103] were still being thrown around. One has to wonder if it is not the fact that a lack of epidemiologic studies that show that PCBs are harmful is what these people mean, for we have listed here 15 separate studies of the effects of PCBs on humans and have yet to turn up anything more serious than chloracne. (Impotence and jaundice have been laid at PCBs doorstep. But the case against them involved a mixture of 90% tetra- and pentachloronaphthalenes and only 10% PCB.[104])

ENVIRONMENTAL CONCERN

Earlier in this article it was mentioned that those pushing for a total ban on PCBs had built their case on two foundations. The one foundation mentioned, which was discussed was animal studies, to which we added the epidemiologic studies to determine the relevance of the tests to human experience. The other foundation upon which their cases is built is that of environmental concern. "Concern about PCBs has, therefore, focused on their resistance to bio-degradation with the consequent potential for long-term or delayed health effects."[105] It is to this concern that we would now like to address ourselves.

The major environmental (and occupational) concern is the great stability of PCBs: They biodegrade *very* slowly, if at all. Due to their stability and to the fact that they are not readily metabolized, PCBs tend to build up (bioaccumulate) in the tissues of living organisms, so that if a person ate 27 μg/day for 70 years he would have close to all 0.68965 g ingested in him at his 70th birthday. He would lose some of it along the way, but how much, is not known.[106]

This bioaccumulation is especially marked in the lower-life forms. Suppose some seaweed has PCB in it. A little fish comes and eats lots of seaweed and the PCB from that seaweed accumulates in him. A bigger fish eats him and others like him and he ends up with an even higher PCB level. Then a bird or human eats that fish and ends up with a major share of all the PCBs that those under him consumed. That's what is meant by bioaccumulation.

Let's see how this looks in a real life situation. The Great Lakes area, for example, one of the most PCB-contaminated bodies of water in the U.S. contains fish that have over 90 ppm PCB in their edible tissues.[107,108] The FDA at that time prohibited the sale of fish with over 5 ppm in their edible tissues.

Not all PCBs (209 different PCBs are possible) are alike. However, the degree of chlorination seems to be a determining factor in the stability of the PCB as it relates to metabolism and biodegradation. It appears that the lower chlorinated PCBs (e.g., Aroclors 1242 and 106) cause less severe reactions in animal tests[109] and as for the environment, the following statement was made at the 1975 EPA Conference on PCBs:

At least 1500 reports in scientific literature have been written relating to PCBs and, whereas most of these deal with only the question of reporting the presence of PCBs in the environment, invariably these reports describe and relate Aroclors 1254 and 1260, the more highly chlorinated PCBs produced by Monsanto.[110]

It was also claimed that the EPA has stated that Aroclor 1242 bioaccumulates on the order of 8 times less than the more highly chlorinated Aroclors."[111]

Aroclor 1242 contains about 91% and Aroclor 1016 about 94% of the lower isomers, containing 4 chlorines or less that more readily biodegrade in the environment. Therefore, if some of these mixtures do enter into the environment "over 90% of the small amount that did enter into the environment would have relatively low persistence."[112]

Bernard Kerns of Westinghouse has stated that "Westinghouse utilized primarily only two askarels (generic name), Aroclor 1242 for transformers and Aroclor 1016 for capacitors."[113]

NIOSH says that, prior to 1971, Aroclor 1242 was the major capacitor impregnant and after that date, it was Aroclor 1016. Transformers contained the more highly chlorinated Aroclors.[114]

It would thus seem that Aroclors 1242 and 1016 could be used in transformers and capacitors, providing the needed safety and minimizing the persistent elements that could enter the environment. And this is exactly what we believe should be done. It is the height of stupidity to persecute a chemical (and indirectly, the chemical industry) that has saved thousands of lives and thousands of buildings from destruction (NIOSH's evaluation):

There are currently no commercially available fluids which can be considered as totally acceptable substitutes for PCBs in the broad range of AC capacitors, nor are there substitute dielectric systems which would satisfy reliability and safety requirements in most applications. Transformers containing PCBs represent only about 5% of the U.S. transformer market, and are used only where safety and reliability are of prime importance . . . Major construction changes would be required to compensate for the fire-resistance of

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askarel-filled units if existing askarel-filled transformers are to be replaced with oil-filled units of equivalent electrical ratings."[10]

Just because it might "potentially" harm someone, NIOSH recommends that a standard be set so that wherever more than one-millionth of a gram of PCB exists in 1000 liters of air only, "a self-contained breathing apparatus with a full face piece operated in the positive pressure mode will provide adequate protection (to) workers."[11] Why this recommendation? "NIOSH concludes that PCBs in workplace air are potential carcinogens." (Emphasis ours.)[12]

In fact, when it comes down to the bottom line, this is the whole argument of those who wish to ban PCBs. Not because they can prove that PCBs have harmed anyone, but because PCBs are "potentially carcinogenic." Why are PCBs considered "potentially carcinogenic?" For the same reasons that saccharin and formaldehyde[11] are considered potentially carcinogenic — if enough of a substance is fed to a rat, it will develop cancerous tumors. PCBs are considered "potentially carcinogenic" because some, but not all, animal tests have turned up cancerous, substances. No evidence exists, apart from the animal tests, that would link cancer and humans. So until a human cancer is linked to PCBs, PCBs will be called "potential carcinogens," and radical environmental groups like the EDF will want them banned.

THE REAL PROBLEM

But even this "ban" won't be complete. The EDF wants to destroy over 17 million mineral oil filled transformers that contain some PCBs in order to get rid of 400,000 pounds of PCBs. Yet 179,000,000 pounds of PCBs exist in the light fixtures, television sets and air conditioners that were manufactured up till the late 1970's.[13] Their proposed ban doesn't even mention these things even though they contain almost 450 times the amount of PCBs. Why don't they go after those household items? Because it would make them look like the extremists they are when government employees entered into every household in America checking to see if everything was properly labelled or, in the case of a ban, confiscating all those items. If they are going to call for a ban, why not be consistent?

Dr. Leonard Goldwater gave us the inspiration for our closing argument. He said:

"There is no substance known to man which cannot cause serious or fatal poisoning in large amount. And, by the same token, there is no substance known to man which cannot be tolerated in some dose."[14]

What we need to do is look at the risks involved with PCBs. They have saved thousands of lives. They have saved many buildings from destruction by fire. They have enabled America to grow industrially by answering the needs of the electrical industry. They have not seriously affected the health of anyone in their fifty-two years of existence. So before some law or regulation is passed that is going to cost one trillion dollars, or even one dollar, for that matter, those who want to pass it should be made to substantiate their case with more than a "potentially" this or that. The American justice system is based on the presumption that one is innocent until proven guilty. America has the right to see the dead bodies.[15] While they are vainly searching for their "dead body" or "smoking gun," let's discuss the costs of what will happen if the EDF gets its way and PCBs are banned from use in transformers and capacitors.

ECONOMIC IMPACT

Transformer Maintenance Institute's primary concern is that of PCB contaminated transformer oil. Pure PCB transformers and capacitors represent 750,000,000 pounds of PCB liquid to be destroyed along with the associated equipment (140,000 transformers, 10,000,000 capacitors).

But . . .

Oil filled transformers having 2 or 5 ppm of PCB contamination will probably be declared to be (outcome of EDF-EPA law suit) pure PCB as far as rules and regulations for marking, inventory storage and disposal is concerned. And here in lies the problem.

There is estimated to be 140,000 to 400,000 pounds of PCB in 1.4 billion gallons of transformer oil in 28 million transformers. To destroy this 400,000 pounds of PCB it will cost 1 TRILLION TWO HUNDRED SIXTY BILLION DOLLARS.

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WHAT'S THE STORY?

The PCB threat—fact or fiction?

Q Over recent years, wide publicity and much fanfare have accompanied the condemnation of askarel transformer liquid because a major component of such fluid is polychlorinated biphenyl,—commonly called PCB. Environmentalists have banded PCBs as a serious threat to public health, and the EPA (Environmental Protection Agency) has backed those charges with rigorous regulations aimed at eliminating the alleged threat of PCBs. But there have been challenges to the charges against PCBs, and responsible and authoritative people have characterized the case against PCBs as a typical "paranoid reaction of radical environmentalists" who have little or no evidence to support their charges. What evidence is there to rebut the EPA stand against PCBs?

A It is true that opposition has been mounted against the ban on PCBs. The information presented here is representative of the opposing viewpoint.

In the "Letters to the Editor" section of *The Review*, a newspaper in Hayward-San Leandro, CA, the following was published on September 14, 1981.

> Editor: When will the hysterical uproar about PCB end and where will it lead us?
>
> The medical literature contains not a single case of cancer in humans attributable to this substance, nor is there in the half century of its use any report of illness related to it.
>
> It is true that if we follow slavishly the policy in present favor to spare no cost to avoid all possible risk to health, we can indeed solve all our problems. Then there will be no energy left in any of us and no money to produce food for our population, and we shall all starve to death!
>
> We, the undersigned physicians, feel it is our responsibility to call on the news media to bring attention to these medical facts to avoid serious harm not only to our economy, but to the mental health of the public [and to a threat] of much greater magnitude than the remote fears on malathion.
>
> As stated in the current issue of the *Physicians Bulletin of Alameda County Care Services*, the greatest risk from spraying of malathion, a pesticide of known toxic potential, is unnecessary anxiety among the residents of the area.
>
> In the case of PCB, so glibly referred to by commentators as the well-known cancer-causing drug, the evil effects are only conjecture based on animal experiments with inordinately large amounts and long and unreasonable exposures.
>
> We can be assured that the money already expended and the costs of impending litigation will appear in the utility bills and taxes of all of us.
>
> *R.A. Medemba, M.D.*
> *Frank Franks, M.D.*
> *J.J. Robbins, M.D.*
> *Louis Brahos, M.D.*
> *Hayward*

In the January 9, 1982, issue of the *Cleveland Plain Dealer* a feature news article under the byline of James Lawless carried the headline "Studies cast doubt on hazards of PCBs." The article was as follows.

> Two major studies prepared for the U.S. Environmental Protection Agency contend the health effects of polychlorinated biphenyls (PCBs) have been seriously exaggerated, said a scientist heading one study.
>
> Dr. Seymour Friess, of Drill, Friess, Hays, Lennis and Schaefer, an Arlington, VA toxicological firm, said his firm extensively reviewed 400 studies of animal exposure and health effects of PCBs. He said the results were "quite surprising to us."
>
> Friess said he could not release details of the studies of toxicologists, done for the Edison Electric Institute and the National Electric Manufacturers Association. But he said, "As scientists, we were shocked that the health effects" are minimal.
>
> He said the U.S. EPA would receive his report in about three weeks, when details would be released.
>
> Under federal court order, EPA is reviewing PCB limits after losing a court battle with the Environmental Defense Fund. EDF contended EPA could not justify its limits, that enforcement was confusing, and that the limits were too lenient. By August, EPA is scheduled to produce new limits that are to be justified by scientific research.
>
> Asked if he agreed with previous industry contentions that PCBs have minor health effects, Friess said, "That is a damn good statement."
>
> Jackie Warren, a lawyer for the National Resource Defense Fund in New York City and lead lawyer in the EDF suit, said, "Unless there is new evidence, I am not at all surprised that PCB manufacturers are trying to get lesser standards. I find it (the study) nothing more than a political document, considering where it came from.
>
> "Previous industry reviews of the literature have shown that PCBs don't cause much harm. I am very suspicious of this."
>
> Friess, on the other hand, said, "Personally, I feel much better than I did before I started reviewing this literature."
>
> A second study has been done by the Chemical Manufacturers

EC&M EDITORS will discuss electrical design, construction and maintenance questions and problems on these pages each month. Readers who disagree with our conclusions or wish to add to them are encouraged to write. We will publish these responses in our "Letters" columns, space permitting. Send to: What's the Story? EC&M, McGraw-Hill, Inc., 1221 Ave. of the Americas, New York, N.Y. 10020.

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WHAT'S THE STORY?

Association (CMA). A source told *The Plain Dealer* it, too, would contend the health effects were minimal.

The source told *The PD* the Freiss study shows "PCBs are not a cancer-causing agent and not the health hazard once feared."

He said the CMA study confirmed these results and discounted EPA's broadrange fears that PCBs cause cancer in laboratory animals and health problems in humans.

The chemical, found in electrical transformers and capacitors and used as an industrial flame retardant, was the only substance specifically cited in the Toxic Substances Control Act in 1976. It was considered dangerous because it is particularly long-lived, stays in fatty tissues, and was found to cause serious health effects in a contamination of cooking oil in Japan.

In a January 1982 letter to *EC&M*, Mr. Stanley D. Myers, president of the Transformer Maintenance Institute, Cuyahoga Falls, OH, wrote the following.

We have recently made a 30-day study of what the news media (newspapers) have been saying about PCBs. This information was sent to us by a news clipping service that monitors 700 newspapers. Inasmuch as all of the following statements have absolutely no evidence to affirm them, the news media have shown that they report allegations as fact and have taken poetic license to describe PCBs in a most astounding manner.

For example, some of the ailments attributed to PCBs are the following:

Cancer	Respiratory problems
Liver damage	Serious health problems
Death	Behavior problems
Genetic damage	Birth defects
Gastrointestinal problems	Sterility
Brain damage	Deformation of bones
Miscarriages	Internal disorders
Cancer of the pancreas	High blood pressure
Abnormalities in blood fat	Permanent injury
Irritation of nose, lungs, eyes	

And here are the ways in which the news media describe PCBs:

Potential toxic substance	Hazardous
Poisonous	Suspected carcinogen
Known carcinogen	Cancer-causing PCB
Hazardous waste	Highly toxic
Extremely toxic	Dangerous pollutants
Dangerous chemicals	Cancer-linked chemical
Deadly cancer-linked chemical	Toxic chemical
Cancer-causing PCBs	Deadly PCBs
Worst chemical ever created	A carcinogen
A banned carcinogenic	An incredible health hazard
Deadly chemicals	A very potent toxic material
Potential disaster	Toxic acid-metal mixture

If all this were true we should declare war on PCBs, forget Russia, Castro, Libya, etc., and absolutely expunge PCBs from America.

However, the only people really agitating for the removal of PCBs appear to be the news media, resulting in fear, apprehension and terror on the part of the public. The astounding truth is that the total cost of removing PCBs would average out to over $30,000 for each individual in the United States. Can we really afford to spend our time, efforts and wealth on a chemical that has never harmed any person in the entire world? *There has never been a clinical report made by any hospital, clinic, doctor, toxicologist, etc., who have treated people exposed to PCBs that speaks to any symptoms, ailments or any effects at all.* Why don't the EPA, EDF, Sierra Club, Ralph Nader, Friends of the Earth, Toby Moffet, Representative Dingle, et al, produce evidence of the harmful effects of PCBs rather than allegations or animal studies?

Transformer Maintenance Institute has done a study documenting the source of information of PCBs used by the radical environmentalists to hound our country into economic chaos and upsetting the fragile base on which they build, with an eventual cost of $4,000,000,000,000 (four trillion dollars) to our country.

But money cannot solve the PCB dilemma. Here in capsule form are the factors involved.

The problem

Congress has called on EPA to ban PCBs based on the best information available at the time legislation was passed. The environmentalists entered the arena and made the allegations concerning PCBs that they are a "chemical catastrophe" and a "mad dog" turned loose. The terror word carcinogenic appears everywhere in relation to PCBs in the news media. The environmentalists have been unable to prove their allegations even though NIOSH states that PCBs are the most widely researched chemical in existence today (circa 1977). Their only argument is this: "Well, we don't really know what will happen 5, 10 or 40 years down the road."

Transformer Maintenance Institute has set up a PCB Information Center to challenge Congress, business, TV, newspapers, etc., in this manner. The environmentalists have not been able to prove their allegations and have violated the most vital canons of problem solving—common sense. They have chosen to ignore 50 years' experience with PCBs, during the time in which PCBs were used, misused, misapplied, dumped, burned, eaten, swallowed, etc., without any discipline or restraint by anyone, anywhere—but clearly without harm to anyone. The remarkable thing about the past 50 years is the *complete absence of any clinical evidence* of any harm to human beings or the ecology or any adverse effects on our lives whatsoever.

The only logical conclusion is that PCBs—and even their by-products, PCDF and PCDDs—have yet to cause any injury to anyone of a permanent or debilitating nature; and based on a 50-year data base, they are not likely to cause problems in the future.

The solution

Realizing that the PCB problem has become a highly emotional affair, that this emotion has been fostered by the environmentalists, and that the news media have sensationalized the PCB problem in a scandalous way, we believe that the economics of the proposed solution (total destruction of PCBs) is the issue to focus on to restore to sanity. The people of this nation cannot afford to pay $4,000,000,000,000 because the environmentalists contend that we won't really know the effect of PCB on human beings for 10, 20, or 40 years.

Congress must revamp its position on PCBs, change the law, and put this problem to bed. We have wasted millions of manhours of high-caliber men already trying to fight our way out of this dilemma. It's time the creative manpower being wasted on this foolishness gets back to work on innovations that will create real jobs for America. EC&M

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Role of Polychlorinated Dibenzofuran in Yusho (PCB Poisoning)

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ABSTRACT. In the blood of 15 patients with Yusho or "polychlorinated biphenyl poisoning" that occurred in 1979 in Taiwan, was found polychlorinated dibenzofurans (14 of 15) and polychlorinated quaterphenyls (15 of 15), as well as polychlorinated biphenyls (15 of 15). The mean concentration ratio of these substances was approximately 1 : 160 : 500. Based on the following evidence, we propose that polychlorinated quaterphenyls were major pathogenic substances in the development of Yusho: (1) Clinical manifestations and course of Yusho patients are disproportionately severe and persistent for the observed blood levels of polychlorinated biphenyls, while patients who were occupationally exposed to pure polychlorinated biphenyls take characteristically mild and benign clinical course despite polychlorinated biphenyl levels often much higher than those noted in Yusho patients; (2) Polychlorinated dibenzofurans show a marked tendency to accumulate in the liver, which might explain frequent presence of jaundice and other abdominal symptoms in Yusho, which are, again, not observed in those with occupational polychlorinated biphenyl poisoning; (3) Toxicity of polychlorinated dibenzofurans is a hundred to ten thousand times greater than that of polychlorinated biphenyls and polychlorinated quaterphenyls in animal experiments.

"YUSHO" is a poisoning characterized clinically by acne-like dermal lesions and a variety of constitutional symptoms. In 1968, an outbreak occurred in western parts of Japanese Archipelago and as a result, more than a thousand persons suffered. The outbreak was initially thought to result from polychlorinated biphenyl (PCB) poisoning[1-5] caused by the consumption of rice oil contaminated with Kanechlor 400, a heat transfer medium.[2] Subsequently, the "toxic" rice oil was also found to contain elevated levels of polychlorodibenzofurans (PCDFs)[6]; highly toxic contaminants of PCBs[3] and polychlorinated quaterphenyls (PCQs)[6]—toxicologically ill-defined, but substances perhaps equally toxic as PCBs.[7,8] Both PCDFs and PCQs can be generated when PCBs are heated.[9] The ratio of PCDFs or PCQs to PCBs can become high during the deodorization process of rice oil under high temperature and reduced pressure.[10] Toxicolog-

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ical contribution of PCDFs in Yusho was further implicated by discovering that the liver of Yusho patients who died from other causes after the outbreak of the poisoning contained PCDFs one thousand times higher than that found in normal populations.[11,12] A recent analysis of the blood of Yusho patients conducted 11 yr after the Yusho outbreak, detected PCQs but not PCDFs, suggesting the role of PCQs as a suitable marker for the past episode of exposure to toxic oils.[13]

During the spring and summer of 1979, cases of peculiar skin disease characterized mainly by acne; cheese-like discharge from the Meibomian gland; pigmentation of the skin, gingiva and the nail beds; and abdominal pains were reported in two prefectures in the middle part of Taiwan, Republic of China.[13] Samples of suspected "toxic" rice oil delivered to us in the autumn were found to contain PCBs. Gas chromatographic patterns of these samples were similar to those found in Japanese "toxic" rice oils. All blood samples from the patients likewise contained PCBs ranging from 54 to 136 ppb (unpublished data). The number of patients exceeded 1,800 at the end of 1980; a detailed epidemiological and clinical report will be made elsewhere.

Of the 15 patients in the present study, PCDFs were found in the blood of all except for one who had mild clinical manifestations. In this study strong evidence suggesting a major role of PCDFs in developing clinical manifestations and prognosis which characterize Yusho (not mere "pure PCB" poisoning) is presented.

METHODS AND MATERIALS

Three samples of "toxic" oil, and 10-ml blood samples were collected from 15 patients with various clinical severities, who were pupils living in the dormitories on the school campus. Every meal was provided by the school. The pupils were estimated to have consumed the contaminated oil in the school chow served during a 4-month period, up to 6 months prior to the time of blood sampling.

Analytical methods for PCBs, PCQs, and PCDFs. Ten milliliters of blood were saponified with 20 ml of 2 *N* KOH ethanol solution for 1 hr under refluxing. After addition of 20 ml water, the saponified sample was extracted with 30 ml of *n*-hexane. The *n*-hexane layer was then washed with 50 ml of water, dried over anhydrous sodium sulfate and concentrated to approximately 5 ml in a K.D. concentrator. The *n*-hexane extract was put on a Florisil column (20 g of 60–100 mesh Florisil, activation at 130°C overnight; 1.8 cm ID) and eluted successively with 130 ml of *n*-hexane, 50 ml of 2% diethyl ether in *n*-hexane, 100 ml of 5% diethyl ether in *n*-hexane, 250 ml of 50% diethyl ether in *n*-hexane, and 250 ml of acetone at a rate of approximately 2 ml/min. The first and second eluates were combined, concentrated, and analyzed for PCBs by gas chromatography (GC) on a Varian Aerograph 2100 machine equipped with ^{63}Ni-ECD, as described previously.[1]

Polychlorinated quaterphenyls eluted in the third fraction were chlorinated exhaustively with antimony-pentachloride, cleaned up on an alumina column to remove impurities, and determined by a Shimadzu GC-6A gas chromatograph fitted with ^{63}Ni-ECD.

Polychlorinated dibenzofurans were eluted in the fourth and fifth fractions from a Florisil column, and after careful evaporation to dryness were subjected to a final clean-up on an alumina column (20 g alumina, Merck Co., Ltd., Art. No. 1077; 1.5 cm ID). To achieve more efficient removal of impurities, 200 ml of 2% methylene chloride in *n*-hexane was used as the first eluate. Polychlorinated dibenzofurans were recovered with 200 ml of 20% methylene chloride in *n*-hexane. The eluate was evaporated to dryness and the residue was dissolved in *n*-hexane and analyzed by GC using a Varian Aerograph 2100 gas chromatograph machine with ^{63}Ni-ECD. Gas chromatograph conditions were as follows: column, 2 m X 2 mm glass column packed with 2% OV-17 on Gas Chrom Q (100/120 mesh); injection, detection, and column temperatures, 255, 320, and 250°C, respectively; carrier gas, N_2 (30 ml/min). Polychlorinated dibenzofurans were estimated by comparing the peak heights of the gas chromatogram with those of PCDF standard, assuming that PCDF isomers with the same number of chlorine atoms all had the same sensitivity, regardless of the sites of chlorine substitution (Fig. 1).

Polychlorinated dibenzofurans in the samples were identified (Fig. 2) by gaschromoto-masspectrographic (GC-MS) methods, using a JEOL-20KP gas chromatograph-JMS-300 mass spectrometer with a computer system. The conditions were as follows: column, 2 m X 2 mm glass column packed with 3% OV-17 on Gas Chrom Q (80/100 mesh); inlet and column temperatures, 300 and 300°C, respectively; ionizing energy, 70eV; accelaration, 3.0 KV, carrier gas, He (2.0 kg /cm^2).

GC-MS Chromatograms at m/z (M^+) and (M^+ – 63) of PCDF in "toxic" oil in Taiwan (Fig. 2). The PCDF fraction of the oil was injected into a GC-MS using a JEOL-20KP gas chromatograph-JMS-300 mass spectrometer with a computer system. The GC-MS conditions were as follows: column, 2 m X 2 mm glass column packed with 3% OV-17 on Gas Chrom Q (80/100 mesh); inlet and column temperatures, 300 and 300°C, respectively; ionizing energy, 70 eV; accelaration, 3.0 KV; carrier gas, He (2.0 kg/cm^2).

RESULTS

The gas chromatographic patterns of PCDFs (Fig. 1) extracted and purified from toxic oil samples in both countries show fair resemblance in peaks 10 through 16, indicating similarity of constituent isomers between the two. A GC-MS confirmation was also done on each PCDF peak for toxic oil and Taiwanese blood samples. A similar relationship was likewise present between the blood of a Yusho patient (No. 11) in Taiwan and the liver of a Japanese patient. A GC-MS chromatographic analysis of the toxic oil from Taiwan indicates that peaks 5 through 14 represent 4–6 chlorinated dibenzofurans with molecular ion (M^+) and fragmental ion (M^+ – 63) resulting from the deletion of COCl (Fig. 2).[5]

The blood concentrations of PCBs, PCQs, and PCDFs found in patients from Taiwan and Japan, Japanese workers occupationally exposed to PCBs, and control Japanese subjects are shown in Table 1. Polychlorinated dibenzofurans were detected in all the Taiwanese patients (6 months after termination of toxic oil ingestion), except for one who had

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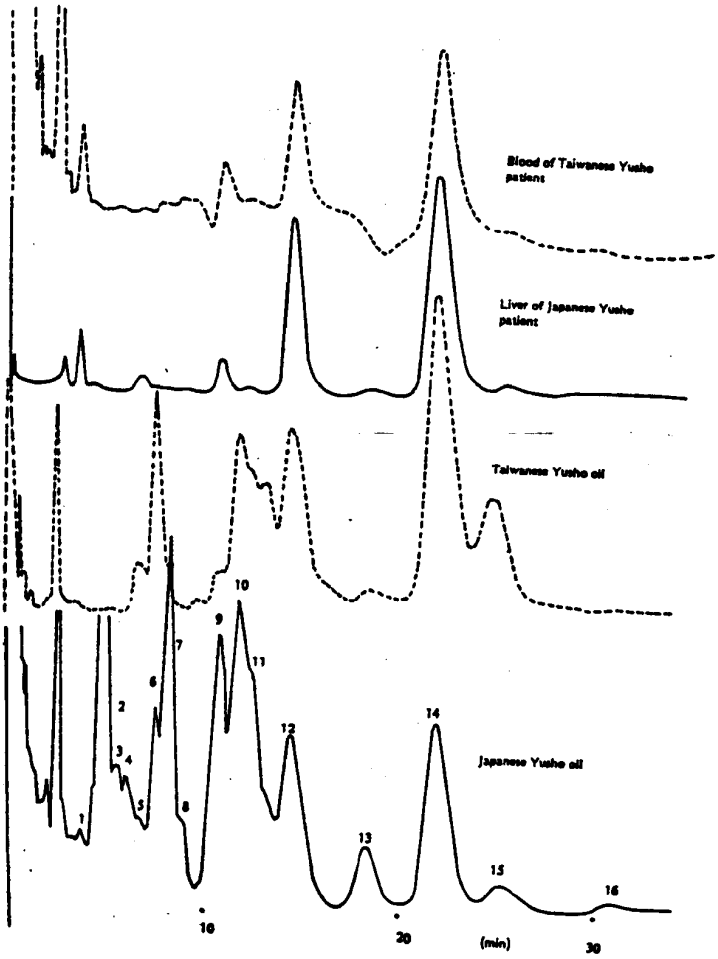

Fig. 1. Gas chromatograms of PCDF fraction: (A) blood of Taiwanese "Yusho" patient (No. 11); (B) liver of Japanese "Yusho" patient; (C) "toxic" oil in Taiwan; (D) "toxic oil in Japan. Compound of each peak shown in gas chromatograms was confirmed as follows by GC-MS analysis: peak No. 2, tetrachlorobiphenyl; peak Nos. 3–7, tetrachlorodibenzofuran; peak Nos. 8, 9, 11, and 12, pentachlorodibenzofuran; peak Nos. 13, 14, and 16, hexachlorodibenzofuran.

MONS 003913

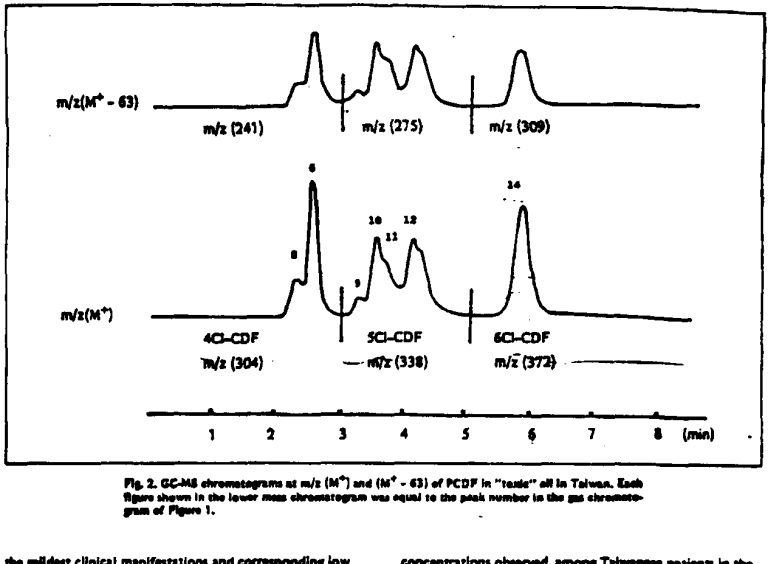

Fig. 2. GC-MS chromatograms at m/z (M^+) and (M^+ - 63) of PCDF in "toxic" oil in Taiwan. Each figure shown in the lower mass chromatogram was equal to the peak number in the gas chromatogram of Figure 1.

the mildest clinical manifestations and corresponding low levels of PCBs and PCQs.

DISCUSSION

Although attempts to identify PCDFs in the blood of Japanese Yusho patients 11 yr after the outbreak failed, PCDF concentrations exceeding those found in the present cases, in relation to PCB levels, would have been found shortly after the outbreak. Japanese toxic oils have a greater PCDF : PCB ratio, and roughly 10 times higher contamination with both substances than Taiwanese toxic oils (Table 1). However, a direct comparison between the two outbreaks with regard to the intake of toxic substances and severity of poisoning is impossible because only some "toxic" lot samples from the Taiwanese incident are available for analysis.

What do the PCDF levels noted in our study signify? We believe that the following evidence strongly suggests that PCDFs were mainly responsible in the pathogenesis of Yusho.

(1) Those individuals occupationally exposed to pure PCBs are usually symptom-free; sometimes mild dermal manifestations such as chloracne accompanied by mild constitutional symptoms occur.[14-16] This occurs despite that blood concentrations of PCBs in individuals 7 yr after leaving their workplace are equal to or greater by one digit than concentrations observed among Taiwanese patients in the current study (Table 1). When occupational exposure to PCBs was terminated, their dermal lesions quickly improved[17] in contrast to persistent clinical signs and symptoms among Yusho patients that have lingered more than 10 yr.[19] It should be again emphasized that most Japanese Yusho patients now have blood PCB levels that barely exceed those of the general population.[15]

(2) PCDFs show strong hepatotropism,[17] as do their analogue[18]—polychlorinated dibenzodioxines. While the PCDF to PCB ratio in the blood of Taiwanese patients is approximately 1 : 500, which is similar to that found in Taiwanese toxic oils (i.e., 1 : 300), the ratio found in the liver of Japanese patients, who succumbed 1 to 9 yr after the incident, is 1 : 5—a value far above the ratios noted in the Japanese toxic oils [1 : 100 in average (Table 1)]. Clinically, no abdominal pains nor jaundice have been described among the three series of workers occupationally exposed to PCBs,[14-16] while 45 to 73% of Japanese Yusho patients experienced abdominal pains[20] and 1-1% had jaundice accompanied by other abdominal symptoms.[21] Presence of hepatotropic and hepatotoxic[22] PCDFs in Yusho can account for this symptomatic discrepancy.

(3) Although toxicity of PCDFs varies depending on the biological parameters (e.g., chick embryo,[5] liver necrosis,[22] thymic atrophy,[23-26] animal species,[23] etc.) and on the structural difference of isomers,[23,26,28] most of these

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Table 1.—PCB, PCQ, and PCDF Levels in Taiwanese and Japanese Patients with "Yusho" Disease, Workers Occupationally Exposed to PCBs, Healthy Persons, and in Toxic Oils Causing "Yusho" Disease

	Sample	No.	Period after Termination of Exposure (yr)	PCB Level (ppb)	PCQ Level (ppb)	PCDF Level (ppb)	PCB : PCQ : PCDF
BLOOD	Yusho patients in Taiwan		0.5	42	14.5	0.072	100 : 30 : 0.16
				54	24.5	0.14	100 : 45 : 0.26
				48	24.0	0.008	100 : 50 : 0.21
				50	17.0	0.11	100 : 19 : 0.12
				54	8.4	0.006	100 : 16 : 0.12
				60	21.0	0.10	100 : 31 : 0.16
				35	11.0	0.17	100 : 31 : 0.17
				4	0.8	N.D.*	100 : 20 : < 0.13
				55	5.0	0.009	100 : 16 : 0.14
				64	15.5	0.009	100 : 20 : 0.11
				103	65.5	0.27	100 : 24 : 0.14
				34	16.9	0.001	100 : 50 : 0.27
				75	24.4	0.006	100 : 31 : 0.11
				37	16.0	0.007	100 : 40 : 0.10
				23	14.4	0.026	100 : 27 : 0.07
		16		60 ± 39§	19.3 ± 13.0§	0.14 ± 0.07§	100 : 30 : 0.16
	Yusho patients in Japan	56	11	5.0 ± 4.4	2.0 ± 2.0	N.D.†	100 : 24 : < 0.17
	Japanese workers occupationally exposed to PCB[1,2]	17	4	50.3 ± 23.0	N.D.‡	—	100 : < 0.05 : —
	Japanese workers occupationally exposed to PCB[1,2]	19	7	140.4 ± 170.2	—	—	
	Healthy subjects in Japan	60		2.0 ± 1.5	N.D.‡	—	100 : < 1 : —
UPPER	Yusho patients in Japan	3	1	223.5	217.5	64.7	100 : 90 : 30
			7	114.4	51.7	6.2	100 : 45 : 5
			9	60.4	27.0	6.1	100 : 30 : 9
				120.7 ± 75.0	98.7 ± 84.6	25.7 ± 27.5	100 : 70 : 30
	Healthy subjects in Japan[1,2]	4		27.5 ± 15.4	—	0.0061 ± 0.0063	100 : — : 0.02
	Toxic oil in Taiwan	3		43,000 ± 15,400	17,300 ± 5,500	141 ± 23	100 : 30 : 0.30
	Toxic oil in Japan[3]	3		430,000 ± 391,000	630,000 ± 167,000	3,550 ± 2,510	100 : 147 : 0.90

*0.005 ppb detection limit.
†0.01 ppb detection limit.
‡0.02 ppb detection limit.
§Values expressed as mean ± SD.

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toxicological comparisons were made using the PCDF and PCB samples which directly arrived "fresh" and "pure" from manufactures. Utilizing PCDFs and PCBs that were either reproduced under the original conditions (i.e., temperature and coexisting air) at the rice oil production plant, or purified from the "toxic oil"—the causative agent of Japanese Yusho—we observed that the toxicity of PCDFs was hundreds to ten thousand times greater than PCB toxicity in monkey,[5] mouse,[26] and rat.[7] In addition to hepatotrophism of PCDFs, the blood PCDF : PCB ratio in the current study alerts one to the toxic contribution of PCDFs in Yusho.

(4) Although the toxicologic role of PCQs has been barely defined in Yusho, the present study shows that the toxicity of PCQs is almost equal to that of PCBs.[7,8] Therefore, the blood levels of PCQs observed among Taiwanese Yusho patients suggest that these substances were relatively insignificant in toxicological contribution. Perhaps PCQs are more important in enabling one to differentiate whether the patient has had a past exposure to "Yusho-type" chlorinated hydrocarbons (i.e., PCBs, PCQs, and PCDFs) or to only PCBs.[13]

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Role of Polychlorinated Dibenzofuran in Yusho (PCB Poisoning)

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ABSTRACT. In the blood of 15 patients with Yusho or "polychlorinated biphenyl poisoning" that occurred in 1979 in Taiwan, was found polychlorinated dibenzofurans (14 of 15) and polychlorinated quaterphenyls (15 of 15), as well as polychlorinated biphenyls (15 of 15). The mean concentration ratio of these substances was approximately 1 : 160 : 500. Based on the following evidence, we propose that polychlorinated quaterphenyls were major pathogenic substances in the development of Yusho: (1) Clinical manifestations and course of Yusho patients are disproportionately severe and persistent for the observed blood levels of polychlorinated biphenyls, while patients who were occupationally exposed to pure polychlorinated biphenyls take characteristically mild and benign clinical course despite polychlorinated biphenyl levels often much higher than those noted in Yusho patients; (2) Polychlorinated dibenzofurans show a marked tendency to accumulate in the liver; which might explain frequent presence of jaundice and other abdominal symptoms in Yusho, which are, again, not observed in those with occupational polychlorinated biphenyl poisoning; (3) Toxicity of polychlorinated dibenzofurans is a hundred to ten thousand times greater than that of polychlorinated biphenyls and polychlorinated quaterphenyls in animal experiments.

"YUSHO" is a poisoning characterized clinically by acne-like dermal lesions and a variety of constitutional symptoms. In 1968, an outbreak occurred in western parts of Japanese Archipelago and as a result, more than a thousand persons suffered. The outbreak was initially thought to result from polychlorinated biphenyl (PCB) poisoning[1-3] caused by the consumption of rice oil contaminated with Kanechlor 400, a heat transfer medium.[3] Subsequently, the "toxic" rice oil was also found to contain elevated levels of polychlorodibenzofurans (PCDFs)[4]; highly toxic contaminants of PCBs[5]; and polychlorinated quaterphenyls (PCQs)[6]—toxicologically ill-defined, but substances perhaps equally toxic as PCBs.[7,8] Both PCDFs and PCQs can be generated when PCBs are heated.[9] The ratio of PCDFs or PCQs to PCBs can become high during the deodorization process of rice oil under high temperature and reduced pressure.[10] Toxicolog-

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ical contribution of PCDFs in Yusho was further implicated by discovering that the liver of Yusho patients who died from other causes after the outbreak of the poisoning contained PCDFs one thousand times higher than that found in normal populations.[12,13] A recent analysis of the blood of Yusho patients conducted 11 yr after the Yusho outbreak, detected PCQs but not PCDFs, suggesting the role of PCQs as a suitable marker for the past episode of exposure to toxic oils.[12]

During the spring and summer of 1979, cases of peculiar skin disease characterized mainly by acne; cheese-like discharge from the Meibomian gland; pigmentation of the skin, gingiva and the nail beds; and abdominal pains were reported in two prefectures in the middle part of Taiwan, Republic of China.[13] Samples of suspected "toxic" rice oil delivered to us in the autumn were found to contain PCBs. Gas chromatographic patterns of these samples were similar to those found in Japanese "toxic" rice oils. All blood samples from the patients likewise contained PCBs ranging from 54 to 136 ppb (unpublished data). The number of patients exceeded 1,800 at the end of 1980; a detailed epidemiological and clinical report will be made elsewhere.

Of the 15 patients in the present study, PCDFs were found in the blood of all except for one who had mild clinical manifestations. In this study strong evidence suggesting a major role of PCDFs in developing clinical manifestations and prognosis which characterize Yusho (not mere "pure PCB" poisoning) is presented.

METHODS AND MATERIALS

Three samples of "toxic" oil, and 10-ml blood samples were collected from 15 patients with various clinical severities, who were pupils living in the dormitories on the school campus. Every meal was provided by the school. The pupils were estimated to have consumed the contaminated oil in the school stew served during a 4-month period, up to 6 months prior to the time of blood sampling.

Analytical methods for PCBs, PCQs, and PCDFs. Ten milliliters of blood were saponified with 20 ml of 2 *N* KOH ethanol solution for 1 hr under refluxing. After addition of 20 ml water, the saponified sample was extracted with 30 ml of *n*-hexane. The *n*-hexane layer was then washed with 50 ml of water, dried over anhydrous sodium sulfate and concentrated to approximately 5 ml in a K.D. concentrator. The *n*-hexane extract was put on a Florisil column (20 g of 60-100 mesh Florisil, activation at 130°C overnight; 1.8 cm ID) and eluted successively with 130 ml of *n*-hexane, 50 ml of 2% diethyl ether in *n*-hexane, 100 ml of 5% diethyl ether in *n*-hexane, 250 ml of 50% diethyl ether in *n*-hexane, and 250 ml of acetone at a rate of approximately 2 ml/min. The first and second eluates were combined, concentrated, and analyzed for PCBs by gas chromatography (GC) on a Varian Aerograph 2100 machine equipped with ^{63}Ni-ECD, as described previously.[1]

Polychlorinated quaterphenyls eluted in the third fraction were chlorinated exhaustively with antimony-pentachloride, cleaned up on an alumina column to remove impurities, and determined by a Shimadzu GC-6A gas chromatograph fitted with ^{63}Ni-ECD.

Polychlorinated dibenzofurans were eluted in the fourth and fifth fractions from a Florisil column, and after careful evaporation to dryness were subjected to a final clean-up on an alumina column (20 g alumina, Merck Co., Ltd., Art. No. 1077; 1.5 cm ID). To achieve more efficient removal of impurities, 200 ml of 2% methylene chloride in *n*-hexane was used as the first eluate. Polychlorinated dibenzofurans were recovered with 200 ml of 20% methylene chloride in *n*-hexane. The eluate was evaporated to dryness and the residue was dissolved in *n*-hexane and analyzed by GC using a Varian Aerograph 2100 gas chromatograph machine with ^{63}Ni-ECD. Gas chromatograph conditions were as follows: column, 2 m X 2 mm glass column packed with 2% OV-17 on Gas Chrom Q (100/120 mesh); injection, detection, and column temperatures, 255, 320, and 250°C, respectively; carrier gas, N_2 (30 ml/min). Polychlorinated dibenzofurans were estimated by comparing the peak heights of the gas chromatogram with those of PCDF standard, assuming that PCDF isomers with the same number of chlorine atoms all had the same sensitivity, regardless of the sites of chlorine substitution (Fig. 1).

Polychlorinated dibenzofurans in the samples were identified (Fig. 2) by gas chromoto-massspectrographic (GC-MS) methods, using a JEOL-20KP gas chromatograph-JMS-300 mass spectrometer with a computer system. The conditions were as follows: column, 2 m X 2 mm glass column packed with 3% OV-17 on Gas Chrom Q (80/100 mesh); inlet and column temperatures, 300 and 300°C, respectively; ionizing energy, 70eV; acceleration, 3.0 KV, carrier gas, He (2.0 kg /cm^2).

GC-MS Chromatograms at m/z (M^+) and (M^+ - 63) of PCDF in "toxic" oil in Taiwan (Fig. 2). The PCDF fraction of the oil was injected into a GC-MS using a JEOL-20KP gas chromatograph-JMS-300 mass spectrometer with a computer system. The GC-MS conditions were as follows: column, 2 m X 2 mm glass column packed with 3% OV-17 on Gas Chrom Q (80/100 mesh); inlet and column temperatures, 300 and 300°C, respectively; ionizing energy, 70 eV; acceleration, 3.0 KV; carrier gas, He (2.0 kg/cm^2).

RESULTS

The gas chromatographic patterns of PCDFs (Fig. 1) extracted and purified from toxic oil samples in both countries show fair resemblance in peaks 10 through 16, indicating similarity of constituent isomers between the two. A GC-MS confirmation was also done on each PCDF peak for toxic oil and Taiwanese blood samples. A similar relationship was likewise present between the blood of a Yusho patient (No. 11) in Taiwan and the liver of a Japanese patient. A GC-MS chromatographic analysis of the toxic oil from Taiwan indicates that peaks 5 through 14 represent 4-6 chlorinated dibenzofurans with molecular ion (M^+) and fragmental ion (M^+ - 63) resulting from the deletion of COCl (Fig. 2).[5]

The blood concentrations of PCBs, PCQs, and PCDFs found in patients from Taiwan and Japan, Japanese workers occupationally exposed to PCBs, and control Japanese subjects are shown in Table 1. Polychlorinated dibenzofurans were detected in all the Taiwanese patients (6 months after termination of toxic oil ingestion), except for one who had

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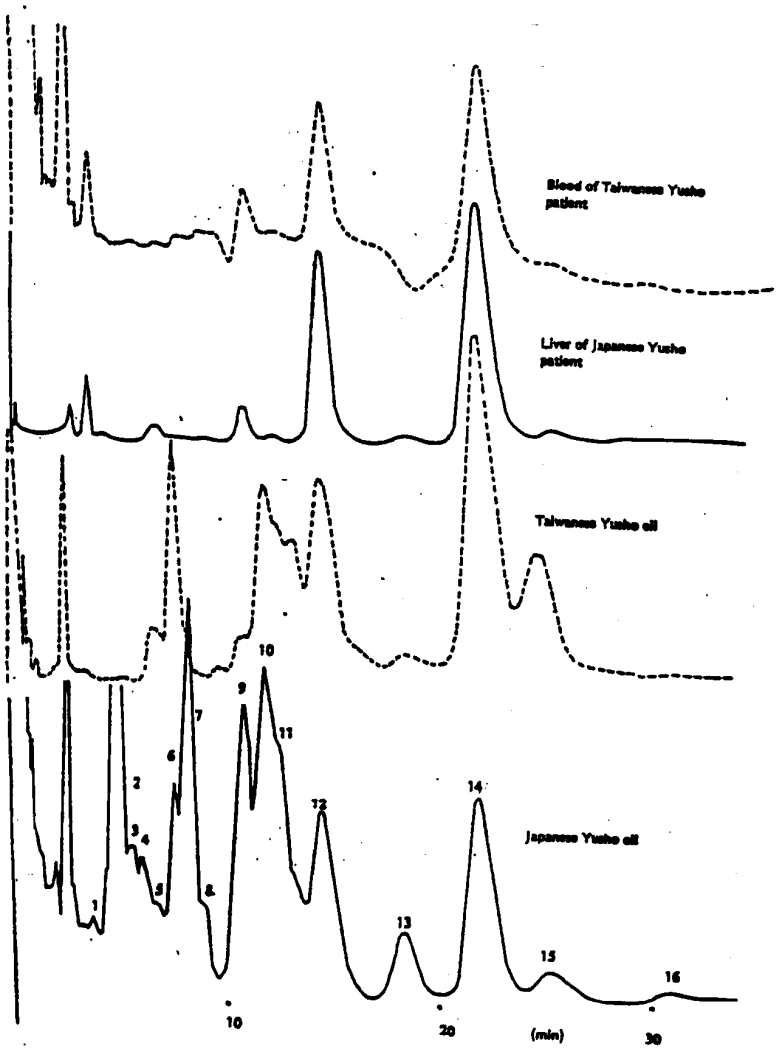

Fig. 1. Gas chromatograms of PCDF fraction: (A) blood of Taiwanese "Yusho" patient (No. 11); (B) liver of Japanese "Yusho" patient; (C) "toxic" oil in Taiwan; (D) "toxic oil in Japan. Compound of each peak shown in gas chromatograms was confirmed as follows by GC-MS analysis: peak No. 2, tetrachlorobiphenyl; peak Nos. 3–7, tetrachlorodibenzofuran; peak Nos. 8, 9, 11, and 12, pentachlorodibenzofuran; peak Nos. 13, 14, and 16, hexachlorodibenzofuran.

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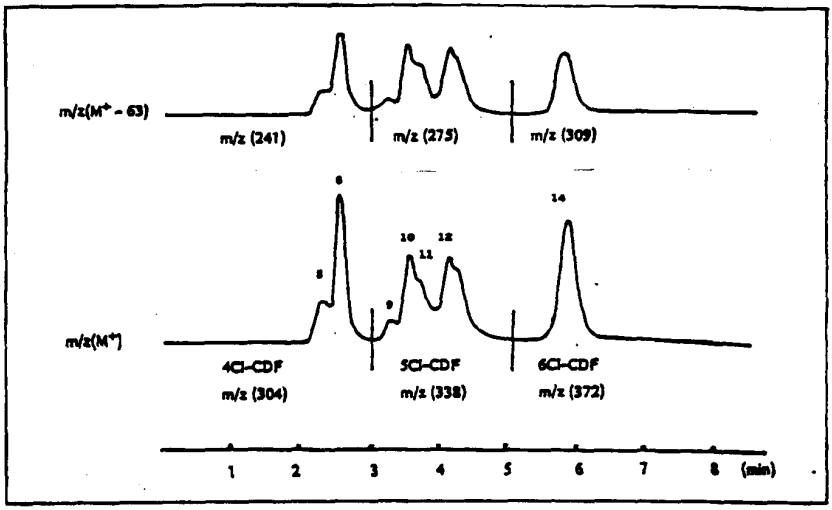

Fig. 2. GC-MS chromatograms at m/z (M^+) and (M^+ - 63) of PCDF in "toxic" oil in Taiwan. Each figure shown in the lower mass chromatogram was equal to the peak number in the gas chromatogram of Figure 1.

the mildest clinical manifestations and corresponding low levels of PCBs and PCQs.

DISCUSSION

Although attempts to identify PCDFs in the blood of Japanese Yusho patients 11 yr after the outbreak failed, PCDF concentrations exceeding those found in the present cases, in relation to PCB levels, would have been found shortly after the outbreak. Japanese toxic oils have a greater PCDF : PCB ratio, and roughly 10 times higher contamination with both substances than Taiwanese toxic oils (Table 1). However, a direct comparison between the two outbreaks with regard to the intake of toxic substances and severity of poisoning is impossible because only some "toxic" lot samples from the Taiwanese incident are available for analysis.

What do the PCDF levels noted in our study signify? We believe that the following evidence strongly suggests that PCDFs were mainly responsible in the pathogenesis of Yusho.

(1) Those individuals occupationally exposed to pure PCBs are usually symptom-free; sometimes mild dermal manifestations such as chloracne accompanied by mild constitutional symptoms occur.[14-16] This occurs despite that blood concentrations of PCBs in individuals 7 yr after leaving their workplaces are equal to or greater by one digit than concentrations observed among Taiwanese patients in the current study (Table 1). When occupational exposure to PCBs was terminated, their dermal lesions quickly improved[15] in contrast to persistent clinical signs and symptoms among Yusho patients that have lingered more than 10 yr.[10] It should be again emphasized that most Japanese Yusho patients now have blood PCB levels that barely exceed those of the general population.[11]

(2) PCDFs show strong hepatotropism,[17] as do their analogue[18]—polychlorinated dibenzodioxines. While the PCDF to PCB ratio in the blood of Taiwanese patients is approximately 1 : 500, which is similar to that found in Taiwanese toxic oils (i.e., 1 : 300), the ratio found in the liver of Japanese patients, who succumbed 1 to 9 yr after the incident, is 1 : 5—a value far above the ratios noted in the Japanese toxic oils [1 : 100 in average (Table 1)]. Clinically, no abdominal pains nor jaundice have been described among the three series of workers occupationally exposed to PCBs,[14-16] while 45 to 73% of Japanese Yusho patients experienced abdominal pains[20] and 11% had jaundice accompanied by other abdominal symptoms.[21] Presence of hepatotropic and hepatotoxic[22] PCDFs in Yusho can account for this symptomatic discrepancy.

(3) Although toxicity of PCDFs varies depending on the biological parameters (e.g., chick embryo,[5] liver necrosis,[22] thymic atrophy,[23-26] animal species,[25] etc.) and on the structural difference of isomers,[23,26,28] most of these

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Table 1.—PCB, PCD, and PCDF Levels in Taiwanese and Japanese Patients with "Yusho" Disease, Workers Occupationally Exposed to PCBs, Healthy Persons, and in Tooth Bile Causing "Yusho" Disease

	Sample	No.	Period after Termination of Exposure (yr)	PCB Level (ppb)	PCD Level (ppb)	PCDF Level (ppb)	PCB : PCD : PCDF
BLOOD	Yusho patients in Taiwan		0.5	43	14.5	0.073	100 : 33 : 0.16
				54	34.5	0.14	100 : 45 : 0.26
				43	34.0	0.092	100 : 56 : 0.21
				58	17.0	0.11	100 : 19 : 0.12
				54	8.4	0.046	100 : 16 : 0.12
				45	21.0	0.10	100 : 31 : 0.16
				35	11.0	0.17	100 : 30 : 0.17
				4	0.9	N.D.*	100 : 26 : < 0.13
				56	9.0	0.059	100 : 16 : 0.14
				64	15.5	0.069	100 : 24 : 0.11
				105	43.5	0.27	100 : 34 : 0.14
				34	16.9	0.091	100 : 50 : 0.27
				76	24.4	0.086	100 : 31 : 0.11
				37	16.9	0.067	100 : 46 : 0.18
				53	14.4	0.036	100 : 27 : 0.07
		15		60 ± 39§	19.3 ± 13.0§	0.14 ± 0.07§	100 : 32 : 0.16
	Yusho patients in Japan	56	11	5.9 ± 4.4	3.0 ± 3.0	N.D.†	100 : 34 : < 0.17
	Japanese workers occupationally exposed to PCB[1]	17	4	30.3 ± 22.5	N.D.‡	—	100 : < 0.05 : —
	Japanese workers occupationally exposed to PCB[1]	10	7	140.4 ± 170.3	—	—	
	Healthy subjects in Japan	60		2.0 ± 1.3	N.D.‡	—	100 : < 1 : —
LIVER	Yusho patients in Japan	3	1	205.0	217.5	64.7	100 : 98 : 29
			7	114.4	51.7	6.2	100 : 45 : 5
			9	65.4	27.0	6.3	100 : 39 : 9
				150.3 ± 72.0	95.7 ± 54.6	25.7 ± 27.6	100 : 70 : 20
	Healthy subjects in Japan[2]	4		27.3 ± 15.4	—	0.0001 ± 0.0003	100 : — : 0.00
	Tooth oil in Taiwan	3		43,000 ± 12,400	17,300 ± 5,500	141 ± 35	100 : 39 : 0.32
	Tooth oil in Japan[3]	3		430,000 ± 391,000	630,000 ± 167,000	3,350 ± 2,510	100 : 147 : 0.80

*0.001 ppb detection limit.
†0.01 ppb detection limit.
‡0.05 ppb detection limit.
§ Values expressed as mean ± SD.

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toxicological comparisons were made using the PCDF and PCB samples which directly arrived "fresh" and "pure" from manufacturers. Utilizing PCDFs and PCBs that were either reproduced under the original conditions (i.e., temperature and coexisting air) at the rice oil production plant, or purified from the "toxic oil"—the causative agent of Japanese Yushō—we observed that the toxicity of PCDFs was hundreds to ten thousand times greater than PCB toxicity in monkey,[6] mouse,[20] and rat.[7] In addition to hepatotroplism of PCDFs, the blood PCDF : PCB ratio in the current study alers one to the toxic contribution of PCDFs in Yushō

(4) Although the toxicologic role of PCQs has been barely defined in Yushō, the present study shows that the toxicity of PCQs is almost equal to that of PCBs.[7,8] Therefore, the blood levels of PCQs observed among Taiwanese Yushō patients suggest that these substances were relatively insignificant in toxicological contribution. Perhaps PCQs are more important in enabling one to differentiate whether the patient has had a past exposure to "Yushō-type" chlorinated hydrocarbons (i.e., PCBs, PCQs, and PCDFs) or to only PCBs.[19]

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CHAPTER 20

THE EPIDEMIOLOGY OF PCBs

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INTRODUCTION

This is a review and evaluation of the epidemiologic evidence concerning the health effects of exposure to polychlorinated biphenyls (PCBs), particularly at levels that do not cause acute toxic effects. A study is considered "epidemiologic evidence" if it measures, directly or indirectly, the differences in the risk of ill health among populations with different exposures to PCBs.

In the past several decades there have been many clinical studies of the effects of heavy exposures to PCBs (e.g., Von Wedel et al., 1943; Schwartz, 1936). Such studies are extremely useful in identifying the kinds of effects that should be investigated. However, they do not address the question of the risk of incurring such effects and are, therefore, not included in this review.

The studies reviewed here fall into three categories. First, there are studies of accidental heavy exposures and the resulting acute and chronic effects. In each case the study was prompted by an outbreak of illness or the occurrence of a death in an exposed population, after which the population was studied.

Second, there are studies of the relationship between exposure to PCBs and the resulting body burden of PCBs in serum or adipose tissue. Strictly speaking, these are not epidemiologic studies since they do not deal with health effects. However, if a relationship between level of exposure and body burden cannot be verified, the interpretation of epidemiologic studies becomes difficult if not impossible.

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The third category is studies that were done because the populations in question were known or suspected to be exposed to PCBs, rather than because some untoward health outcome had been observed first.

Many published reports combine some or all of these types of investigations. In the sections that follow, we consider first the studies of accidental over exposure; second, the studies of PCB exposure versus body burden; and third, the epidemiologic studies of exposed populations. In the latter section, the discussion will be organized with respect to the health effects that were investigated. These are:

(1) dermatologic symptoms,
(2) biochemical alterations,
(3) other symptoms and illnesses,
(4) carcinogenicity.

ACCIDENTAL HEAVY EXPOSURE

Two epidemiologic studies of accidental exposure have been reported. The first (Meigs et al., 1954) described an outbreak of chloracne in a plant in which a process change had introduced an unspecified PCB compound into the work environment. Breathing zone levels of PCB were stated to be 0.1 mg/m^3. Seven of 14 exposed workers developed chloracne, but liver function tests were normal in six of these, with some borderline abnormalities in the seventh. The chloracne disappeared after treatment, and the single borderline liver function abnormality improved, but did not disappear after 13 months. Improved process control prevented any recurrence.

Although the estimated PCB level must be accepted with reservation because of the state of the art at that time, it is clear that the chloracne resulted from the PCB exposure. Given the lack of controls and the small rate of abnormal liver function, it is unlikely that the PCB exposure had any connection with the liver function findings.

The second incident is the now famous Yusho incident in 1968 which has been documented in many reports (Kuratsune et al., 1972; Urabe et al., 1979) in which some thousand Japanese became ill after eating cooking oil which had been contaminated with Kanechlor 400, a PCB compound of Japanese manufacture.

The most common acute symptoms observed were hyperpigmentation and acne-like lesions, discharge from the eyes, central nervous system symptoms, and vomiting and diarrhea. There was a dose-response relationship between the amount of oil ingested

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and the proportion of persons reporting symptoms. Three years later about half the patients had improved, but still had symptoms. Six years later many patients still reported such symptoms as headache, stomach pain, numbness of the extremities, joint pain, and respiratory symptoms.

Out of ten live births to women affected by Yusho, nine showed hyperpigmentation and most had increased eye discharges. These symptoms later disappeared. Although there have been reports of premature eruption of teeth (2 children out of a series of 13) and unusually wide fontanelles and sagittal sutures (3 out of 13), it is not at all clear that these findings represent any more than the normal variation to be expected, since no control observations were made (Funatsu et al., 1972).

In general, laboratory tests of the Yusho victims showed elevated serum triglyceride levels, low serum cholesterol in serious cases, and elevated serum glutamic-oxaloacetic transaminase (SGOT) and serum glatamic-pyruvic transaminase (SGPT) levels in serious cases (Higuchi, 1976).

As of the end of 1977, fifty-one deaths among Yusho patients had been identified. The percentage of cancer deaths (35.4) exceeded that of the prefecture in which the deaths occurred (21.1). However, the figures do not appear to be very useful for several reasons. First, after the original incident, the criteria for diagnosis of Yusho had been changed, so that it is impossible to determine the denominator which produced this number. The completeness of ascertainment of the deaths is unknown. In addition, no adjustment for age appeared to have been made in the above comparison. Finally, the average elapsed time from exposure to death was less than ten years, and cannot be calculated precisely because the dates of death are not provided. This may well be too short a period for cancers resulting from the exposure to show up.

Although the Yusho incident represented a massive ingestion of PCBs, recent reanalysis of the cooking oil and of the estimated intake by the patients shows that the exposure to polychlorinated dibenzofurans (PCDFs) and polychlorinated quaterphenyls (PCQs) was about equal to the exposure to PCBs, and current determinations of PCQs in blood and other tissues of Yusho patients have shown levels similar to that of PCBs (Kimbrough, 1980). It is, therefore, doubtful whether any generalization can be made from this incident to lower level environmental or occupational exposures to PCBs.

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ENVIRONMENTAL LEVELS AND BODY BURDENS

Two studies of the relationship between ingestion of PCBs and blood levels of PCBs have been reported (MDPH, 1975; Kreiss et al., 1981). In each case the study was concerned with ingestion of fish known to contain relatively high levels of PCBs. In the first, an association was found between blood PCBs and exposure level as estimated by the amount of Lake Michigan sport fish consumed. In the second, the relationship between blood PCBs and a complex of factors was examined in a population in an area with high levels of environmental contamination. Age, sex, and fish consumption, in that order of importance, were associated with blood levels of PCBs. To the extent that fish consumption measures ingestion of PCBs, these studies confirm that blood PCBs are a function of ingestion of PCBs as well as of age and sex. Other associated variables were examined in Kreiss et al. (1981) but will be discussed in the following section.

A number of studies of blood PCBs and exposure to PCBs have been made, most of them in conjunction with studies of health effects. The portions of the studies relevant to this section are reviewed here.

There are three types of studies. The first compares groups which have had different exposure levels as estimated from process considerations or environmental measurements. For convenience, such a study design will be called Type A. The second, which we will designate Type B, measures the change over time in a single group after PCBs have been removed from the environment (or after the group has left the environment). The third, Type C, compares groups that have had different durations of exposure. Often the same report will contain more than one type of study. For example, an exposed group may be compared with an unexposed group (Type A) and within the exposed group long-term exposed workers may be compared with short-term workers (Type C).

The measure of body burden has in most cases been a single number representing, depending on the study, blood PCBs, plasma PCBs, serum PCBs (all of which are called "blood" PCBs in this review), or level of PCBs in adipose tissue. Analytic methods have varied over time and among investigators. More recently, measures of body burden have sought to determine separately the levels of higher chlorinated biphenyls (5 or more chlorine atoms per molecule) and lower chlorinated biphenyls.

Table 1 lists the studies considered in this section, with the type of design and whether or not separate determinations of higher and lower chlorinated biphenyls were made. All of the studies except Baker et al. (1980) are occupational.

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TABLE 1

Studies of Environmental Levels and Body Burden of PCBs by Type of Body Burden Measure

Study	Study Type[a]	High and Low Chlorinated PCBs	Adipose PCBs
Baker et al., 1980	A	No	No
Bumgarner et al., 1973	C	No	No
Chase et al., 1982	A,C	No	Yes
Hara et al., 1974, 1975	B,C	No	No
Hasegawa et al., 1972	A,B,C	No	No
Inoue et al., 1975	A,C	No	No
Karppanen and Kolho, 1973	A	No	Yes
Kitamura et al., 1973	B	No	No
Maroni et al., 1981a	A,C	Yes	No
Ouw et al., 1976	A,B	Yes	No
Smith et al., 1982	A,C	Yes	No

[a]A = Comparisons of groups with different exposure levels
B = Evaluation of results of decreasing or removing exposure
C = Comparisons of groups with different durations of exposure

All of the Type A studies agree in showing a higher body burden of PCBs in populations with higher environmental exposure, except for one anomaly in Baker et al. (1980). There, persons exposed to sludge containing PCBs had slightly lower blood levels than the controls, on the average. However, the sludge-exposed persons and the controls were not matched for age, which Kreiss et al. (1981) showed to be the most important factor associated with blood PCB level. It, therefore, appears unequivocal that higher exposure to PCBs means a higher body burden, all other things being equal.

The Type B studies appear at first glance to be more equivocal (Table 2). Two studies show a decrease when exposure ceased or decreased and two do not. However, the studies showing no decrease remeasured their study groups within a month or two after exposure changed. The ones showing a decrease remeasured after three months and one year.

The fact that Ouw et al. (1976) found no decrease after two months while Kitamura et al. (1973) found over a 50 percent

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TABLE 2

Studies of Blood PCB Levels Before and After Exposure Levels Changed, and Interval From Exposure Change to Remeasurement

Study	Exposure Change	Interval to Remeasurement (months)	Decrease in Blood PCB Level (percent)
Hara et al., 1974, 1975	Ceased	12	ca75
Hasegawa et al., 1972	Ceased	1	None
Kitamura et al., 1973	Ceased	3	>50
Ouw et al., 1976	Decreased	2	None

decrease after three months gives rise to some uneasiness. However, in the former study exposure was decreased but still present, while in the latter study PCB use had ceased. Ouw et al. (1976) also suggest that after exposures in their study plant had decreased, workers did not wear gloves as recommended, so that the blood PCB levels may have resulted from skin contact.

Table 3 shows the findings for the Type C studies other than Maroni et al. (1981a) and Smith et al. (1982) that is, for those that compared duration of exposure with a single measurement of blood PCB level. The results are not consistent. The study of Bumgarner et al. (1973) found very low levels (average 4 ug/kg) in exposed workers, which may have accounted for their failure to find a relationship with duration. On the other hand, the exposed workers in Hasegawa et al. (1977) had an average level of 370 ug/kg and still showed no relationship with duration.

The studies of Maroni et al. (1981a) and Smith et al. (1982) suggest a possible explanation. Maroni et al. (1981a) made separate comparisons of high chlorinated PCBs and low chlorinated PCBs between workers with present and past exposures. They found differences in the low chlorinated PCBs but not in the high chlorinated compounds. Even though their analysis did not adjust for age, it suggests that the relationship between blood PCB levels and duration and recency of exposure may be a function of the level of chlorination of the PCBs. Smith et al. (1982), however, in an elaborate analysis

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TABLE 3

Studies of PCB Levels by Duration of Exposure

Study	Relationship of Blood PCB to		
	Duration of Exposure	Age	Race
Bumgarner et al., 1973	No	No	No
Chase et al., 1982	Yes	Yes	--
Hara et al., 1974, 1975	Yes	--	--
Hasegawa et al., 1972	No	--	--
Inoue et al., 1975	Yes	--	--

of high and low chlorinated blood PCBs versus present and past exposure, found no "evidence either to support or refute different accumulation kinetics in humans for the lower and higher chlorinated biphenyls". Nevertheless, they found a significant correlation between current personal air PCB levels and low chlorinated blood PCBs, but no significant correlation with high chlorinated blood PCBs.

In summary, body burdens of PCBs are clearly related to the level of exposure to environmental PCBs. Observations of a decrease in the burden of PCBs after exposure is eliminated or decreased are not consistent. The lack of consistency may be due to the short periods of observation of some of the studies, or possibly to differences in the average chlorination of the PCBs involved. Studies of the relationship of PCB burden to duration of exposure again are not consistent. There is a suggestion that this may be due to the confounding effects of age and sex, or to differences in the metabolism of high and low chlorinated PCBs, with the higher PCBs being more likely to accumulate in adipose tissue.

EPIDEMIOLOGIC STUDIES OF PCBs AND HEALTH

Excluding mortality studies, there are 18 epidemiologic studies of health effects related to PCB exposure. The accident report of Meigs et al. (1954) is included since it did not

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differ in design from many of the studies that were not motivated by accident reports.

These studies are listed in Table 4 with a summary of the findings by major category. Five of the reports are in Japanese (Hara et al., 1974, 1975; Hasegawa et al., 1972; Inoue et al., 1975; Kitamura et al., 1973). The details of those studies are taken from the NIOSH criteria document for PCBs (NIOSH, 1977).

Two of the studies (Kaappanen and Kolho, 1973; SCDH, 1978) are not specific as to health effects. The first of these is a comparison of groups with different work exposures and different blood PCB levels (74-1900 ug/kg in the 12 persons with the greatest exposure) in which the authors simply state that all persons studied were in good health. The second is a study of 32 workers in a capacitor plant, 10 of whom were exposed regularly to PCBs. The authors state that there is "no evidence of physical harm resulting from working with PCBs".

The remaining 16 studies in Table 4 are reviewed below with respect to their findings in each major category of health effects. The studies are considered in the order of their population.

Dermatologic Effects

There are 12 studies of dermatologic effects associated with PCB exposure. The first is Meigs et al. (1954) described in the above section on accidental heavy exposure, who found that 7 of 14 exposed workers got chloracne where the PCB concentration in their breathing zones averaged 0.1 mg/m^3. Hasegawa et al. (1972) reported an unstated number of cases of hyperpigmentation of the hands, and acne-like lesions of the jaw, back, and thighs in exposed workers. The average blood PCBs in the workers was 370 ug/kg. However, the authors state that skin complaints were unrelated to blood PCB levels and appeared to be due to skin contact. Kitamura et al. (1973) reported a range of skin disorders in 10 of 13 exposed workers with an average blood level of 820 ug/kg. The disorders occurred on parts of the body not normally in direct contact with PCBs. Hara et al. (1974, 1975) reported that about 45 percent of 118 capacitor workers complained of blackheads and other acne-like symptoms while working with PCBs. The complaints were not related to blood levels of PCBs, and virtually disappeared within a year after exposure had ceased.

Inoue et al. (1975) reported one case of chloracne in an exposed worked whose blood PCBs were in the 190 to 210 ug/kg range, but no symptoms in the rest of a small work force whose

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TABLE 4

PCB Epidemiology Studies (Other Than Mortality) and Summary of Findings[a]

Study	Dermatologic Findings	Physiological Parameters	Symptoms and Illness	Other
Alvares et al., 1977	--	Y	--	--
Baker et al., 1980	N	Y	N	--
Bumgarner et al., 1973	--	N	--	--
Chase et al., 1982	Y	Y	Y	--
Fischbein et al., 1979	Y	N	Y	--
Hara et al., 1974, 1975	Y	Y	--	--
Hasegawa et al., 1972	Y	Y	--	--
Inoue et al., 1975	Y	--	--	--
Kaappanen and Kolho, 1973	--	--	--	N
Kitamura et al., 1973	Y	N	--	--
Kreiss et al., 1981	--	Y	N	N
Maroni et al., 1981b	Y	Y	Y	--
Meigs et al., 1954	Y	Y	Y	--
MDPH, 1975	N	--	N	--
Ouw et al., 1976	Y	N	--	--
Smith et al., 1982	Y	Y	Y	--
SCDH, 1978	--	--	--	N
Warshaw et al., 1979	--	Y	Y	Y

[a]Y = Findings associated with exposure
N = No findings associated with exposure
No entry = No data presented

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blood PCBs ranged from 130 to 520 ug/kg. The Michigan Department of Public Health (1975) reported no relationship of any Yusho symptoms to consumption of fish with high levels of PCBs. Ouw et al. (1976) reported 14 cases of dermatitis, eye irritation, or burning sensations on the skin out of 34 exposed workers, where air levels of PCBs ranged from 0.32 to 2.22 mg/m^3. The complaints appeared to occur more often in those with higher blood PCB levels. Fischbein et al. (1979) reported that about 50 percent of 326 capacitor manufacturing workers reported a history of dermatological symptoms, the most common symptom being a rash. Those with symptoms had higher blood levels of high chlorinated PCBs. Baker et al. (1980) reported no chloracne in 18 exposed workers (average blood PCBs 75.1 ug/kg) or 19 members of their families (average blood PCBs 33.6 ug/kg). Maroni et al. (1981b) reported 10 cases of dermatitis (5 diagnosed as active or past chloracne) out of 80 exposed workers. The average blood PCB level in the study was 342 ug/kg. Chase et al. (1982) observed chloracne and an increase in other dermatological findings in 86 exposed male railroad workers, but the findings were not significantly associated with blood or fat PCB levels. Smith et al. (1982) found no chloracne in a study population of 324 exposed workers in capacitor manufacturing and transformer repair, whose average blood PCBs ranged from 38 to 546 ug/kg. However, there was a significant association of skin rash or dermatitis with blood levels of high chlorinated PCBs.

Interpretation of this mass of data is complicated by the difficulty of diagnosing chloracne, the uncertainties of blood PCB determinations, and the changing technology for making such determinations. Nevertheless, the data suggest strongly that when PCB blood levels exceed about 150 to 200 ug/kg, chloracne can occur. However, most studies have shown that the occurrence of chloracne is not further associated with blood PCB levels. This suggests that (a) personal idiosyncratic factors may be involved and/or (b) that the high blood levels are an indicator of the existence of environmental contamination which actually produces chloracne by skin contact.

The reports of dermatitis other than chloracne suffer from an additional complication. According to the National Health Survey, about one-third of all Americans of working age have at least one current skin condition serious enough to warrant evaluation by a physician (NCHS, 1979). Clearly, substantially more than one-third must have either a current condition or a history of such a condition in the past. The prevalence figures reported by Maroni et al. (1981b) and Fischbein et al. (1979) are, therefore, not in themselves remarkable, but the agreement of Fischbein et al. (1979) and Smith et al. (1982) on the relationship between dermatitis and

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high chlorinated blood PCBs suggests that this association may be real.

Liver Function

Ten studies examined liver function. Meigs et al. (1954) found one borderline abnormal liver function in 14 exposed workers. Hasegawa et al. (1972) found mild disturbances in exposed workers (increased SGOT, SGPT, SAP, decreased serum cholninesterase) which they did not consider to be clinically significant. Kitamura et al. (1973), Ouw et al. (1976), Fischbein et al. (1979), and Baker et al. (1980)(a non-occupational study) found no abnormalities associated with exposure, except that Ouw et al. (1976) found a high BSP retention in four out of seven workers with blood levels above 500 ug/kg.

Maroni et al. (1981b) found 16 out of 80 workers with abnormalities in GGT, OCT, and transaminases. Their blood PCB levels were higher than those in the workers with normal liver function. Kreiss et al. (1981)(non-occupational study) found no relation between liver function and blood PCBs when age and alcohol consumption were taken into account. Chase et al. (1982) found among exposed railroad workers a statistically significant correlation between blood PCBs and SGOT after adjusting for age. Smith et al. (1982) found elevated SGOT and GGT levels in persons with higher blood PCB levels.

In summary, six studies of the ten found some mild liver function abnormalities, none of which were associated with any measurable adverse health effects. The two non-occupational studies, Baker et al. (1980) and Kreiss et al. (1981), found no abnormalities associated with blood PCB leve. Fischbein et al. (1979) in their study of capacitor manufacturing workers, noted that "there was a paucity of abnormal results in the biochemical studies."

Finally, Alvares et al. (1977) reported that in five workers occupationally exposed to PCBs, the rate of drug metabolism was significantly higher than in a group of controls matched for age, sex, and smoking and drinking habits.

Fat Metabolism

Six studies considered cholesterol levels. One (Bumgarner et al., 1973) found no relationship between cholesterol level and blood PCB level. One of the remaining five (Hasegawa et al., 1972) found a decreasa in cholesterol, glycerides,

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phospholipids, and beta-lipoprotein in exposed workers. Three of the other four studies found no relationship of cholesterol to blood PCBs (Baker et al., 1980; Chase et al., 1982; Smith et al., 1982) while one found increased cholesterol at higher blood levels of PCBs (Kreiss et al., 1981). Kreiss et al.)1981) and Smith et al. (1982) also present contradictory findings with respect to HDL cholesterol levels; the former found no relationship to blood PCBs, but the latter found an inverse relationship.

Five studies examined triglyceride levels. Four found an increase with increased blood PCBs (Hara et al., 1974, 1975; Baker et al., 1980; Chase et al., 1982; Smith et al., 1982) while one found no relationship when cholesterol level was taken into account (Kreiss et al., 1981).

In summary, most studies, including one non-occupational study, have associated increased triglycerides with PCB exposure. The data on cholesterol show no relationship in three studies, and an increase and decrease in one study each. HDL cholesterol either decreased or was unchanged (one study each). Even if PCB exposure has some effect on fat metabolism, it appears to be without any apparent clinical significance.

Blood and Blood Pressure

There are five studies of blood chemistry: Bumgarner et al. (1973), Kitamura et al. (1973), Fischbein et al. (1979), Baker et al. (1980), and Maroni et al. (1981b). None of them report any relationship of blood chemistry to PCB levels.

Bumgarner et al. (1973) and Kreiss et al. (1981) measured blood pressure in exposed persons. Bumgarner et al. (1973) found no association with PCBs, but Kreiss et al. (1981) found a statistically significant association between diastolic blood pressure and blood PCBs. Since there was no control group and since Kreiss et al. (1981) are the only investigators to report this finding, its significance is not clear at this time.

Symptoms, Illness, and Other Conditions

Eight studies investigated symptoms, illness, and other conditions in persons exposed to PCBs. None of the three non-occupational studies reported any abnormalities. The first of these (MDPH, 1975) compared the incidence of 18 conditions, most of which were reported in Yusho disease, in consumers of sports fish containing PCBs and in controls. The second (Baker et al., 1980) found none of the following conditions were associated with blood PCB levels in a community study; fever,

weight loss, anorexia, fatigue, headache, eye irritation, cough, shortness of breath, nausea, vomiting, diarrhea, abdominal pain, arthralgia, and persistent rash. The third study (Kreiss et al., 1981) reported the same thing for prevalence of illness or weight loss in the preceding year, use of medication, use of medical care, history of heart disease, and percentage of pregnancies ending in miscarriage, stillbirth, or infant death.

Of the occupational studies, Chase et al. (1982) reported no evidence of organ toxicity from the medical histories or physical examinations of 86 exposed railroad workers. Three studies reported various symptoms. Fischbein et al. (1979) reported a history of gastrointestinal symptoms in 18 percent of 326 capacitor manufacturing workers, a prevalence of from 3.0 to 15.2 percent of various musculoskeletal symptoms, and a prevalence of from 4.8 to 27.8 of various neurological symptoms. These were, however, unrelated to duration of employment or to level of blood PCBs. Maroni et al. (1981b) reported eight cases of gastrointestinal complaints in 80 exposed workers, with no indication of whether there was a relationship to duration of employment. They also reported two bleeding haemangiomas and one case of chronic myelocytic leukemia. These findings do not appear to have any significance, since they apparently are unrelated to the circumstances of exposure.

Smith et al. (1982) reported an increased prevalence of general malaise and possibly altered peripheral sensation with increased blood PCB levels among occupationally exposed workers, but found no clinical abnormalities on physical examination.

Finally, one study of pulmonary function (Warshaw et al., 1979) reported decreased vital capacity in 243 capacitor workers. However, the pulmonary function values in the study population, most of whom were current or ex-smokers, were compared with a standard population of non-smokers, so that the effect of smoking was not taken into account.

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."

There appear to be no significant clinical effects associated with the occupational or environmental exposures studied in these reports.

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Carcinogenicity

It is generally agreed that epidemiologic evidence for carcinogenicity should fulfill certain requirements in order to be acceptable. These requirements, presented below, deal with the study design, the logic of the observed pattern, and the repeatability of the results (Doll, 1981).

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

(2) That are not explained by bias in recording or detection.

(3) That are not explained by confounding.

(4) That are not explained by chance.

(5) That vary appropriately with dose.

(6) That vary appropriately with period of exposure.

(7) That are observed repeatedly in different circumstances.

There are four studies directed solely or primarily to the question of the carcinogenicity of PCBs. Table 5 lists the studies and their findings. They are reviewed here keeping in mind Doll's requirements.

The most obvious feature in Table 5 is that no study agrees with any other. That is, the requirement of repeatability is not met.

The first study by Bahn et al. (1976, 1977) observed three melanomas in a group of 92 research and development and refinery workers. These workers had an unknown exposure to other possible carcinogens, so that there could have been confounding. In any case, the study was withdrawn for revision in the definition of the exposed population, and has not yet been released (NIOSH, 1977).

Zack and Musch (1982) studied 89 Monsanto workers exposed for at least six months between 1945 and 1965 inclusive. There were no deaths from cancer of the liver or cirrhosis. The excess in respiratory cancer was based on four deaths and was not statistically significant. As with Bahn et al. (1976, 1977), there was confounding because of other chemical exposure at the plant and, in this case, possibly cigarette smoking.

TABLE 5

Inconsistencies in Studies of Cancer in PCB Exposed Populations With Findings

Study	Number Studied	Findings
Bahn et al., 1976; 1977	92	Melanoma[a]
Zack and Musch, 1982	89	Lung
Brown and Jones, 1981	2,567	Liver
		Rectum
Bertazzi et al., 1981	1,310	Digestive[b], Lymphatic, and hematopoietic

[a]Significant at 1 percent level
[b]Significant at 5 percent level

Brown and Jones (1981) studies 2,567 workers in a capacitor plant. About half the cohort had a latency period of 20 yrs or more. Although there was an excess of liver cancer deaths, it was inversely related to duration and latency of exposure, which does not support an occupational explanation. There was also an excess of rectal cancer. However, the two plants studied are located in an area whose mortality from rectal cancer is greater than the U.S. average (Mason et al., 1975). Since U.S. population rates were used as a basis for comparison, the rectal cancer excess is at least partly an artifact.

Bertazzi et al. (1981) studied 1,310 workers with at least six months employment in capacitor manufacturing between 1946 and 1970. Although excess digestive cancer was observed, there were no liver cancer deaths. The total number of deaths was small (27) and the excess cancer observed was based on two or three deaths for each of the two major sites involved. There is no indication of the duration or latency of exposure for the cancer deaths. The authors state that there were no other major exposures at the plant, and propose to continue the study with a larger cohort. In spite of the statistical significance of the excesses from all cancers, this study must be considered a preliminary report, particularly since it shares with the other studies a failure to agree on any particular pattern of mortality.

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The existing mortality studies of occupational exposure do not show the agreement that would lead one to infer an excess risk of cancer. Much of the conflicting findings can be attributed to the possible effect of confounding exposures, and to the "noise level" of sporadic excesses which would be expected in the absence of any occupational hazard.

SUMMARY AND CONCLUSIONS

The epidemiologic studies of exposure to PCBs show that the body burden in exposed persons, whether the exposure is by ingestion, inhalation, or skin contact, is related to the environmental levels and distribution of PCB. The relation of body burden to duration of exposure is less clear, and appears to differ depending on the degree of chlorination of the PCBs. Nevertheless, the evidence is clear that higher exposures mean higher blood PCB levels, and that persons with occupational exposures have blood PCB levels that may be an order of magnitude greater than that of environmentally (that is, non-occupationally) exposed persons.

Occupational exposure to PCBs at high levels has been associated with the occurrence of chloracne, but the relationship is not straightforward, suggesting that the actual risk of chloracne is also a function of individual susceptibility and personal work habits, as well as possible exposure to other contaminants.

Dermatologic problems other than chloracne are associated with occupational exposure, and may be related to exposure to high chlorinated PCBs.

Alterations of liver function and fat metabolism associated with PCB exposure have been observed in several studies, but are characterized by investigators as mild and of no clinical significance.

The one fact on which all occupational studies of health effects agree is that there has been no clinical illness associated with PCB exposure other than dermatitis. Studies of non-occupationally exposed populations have found neither dermatitis nor other clinical evidence of exposure-related effects, with the exception of a single study which suggests that diastolic blood pressure may be related to blood level of PCBs.

Mortality studies concerned primarily with cancer present problems of interpretation due to the small sample size of some of the studies, and to the confounding effect of other

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exposures. However, they do exhibit a pattern, which is that none of the studies agree on the cancer sites at which an excess mortality was found, and the excesses that were found are in general not statistically significant. One must conclude that the findings of the mortality studies reflect a sporadic pattern of excess mortality at different sites which is not consistent with a carcinogenic effect of PCBs. In addition, where an examination of duration and latency of exposure was possible, no association with these variables was found (Brown et al., 1981).

Taken as a whole, the epidemiologic studies find that high occupational exposures to PCBs may cause dermatitis of various kinds, but that there are no other clinically observable effects, including the occurrence of cancer.

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