STATEMENT OF ROBERT RISEBROUGH BEFORE THE COMMITTEE ON COMMERCE

UNITED STATES SENATE, AUGUST 4, 1971

My name is Robert Risebrough. Since 1965 I have been employed by the Institute of Marine Resources at the University of California, Berkeley. On July 1, 1971 I joined the staff of the Bodega Marine Laboratory of the University of California, where I am an Associate Research Ecologist. With colleagues from several universities and other scientific institutions I have carried out research in the field of environmental pollution, primarily with the chlorinated hydrocarbons. These substances include several of the insecticides and the polychlorinated biphenyls. With my colleagues I have prepared approximately forty technical and semi-technical papers in this field.

In 1970 I was a member of the panel on Monitoring Persistent Pesticides in the Marine Environment of the Committee on Oceanography of the National Academy of Sciences. I also served as chairman of the Committee on Halogenated Hydrocarbons of the Seminar on Methods of Detection, Measurement and Monitoring of Pollutants in the Marine Environment, organized by the Food and Agriculture Organization of the United Nations in Rome, Italy. Currently I am a member of the panel on Marine Aquatic Life and Wildlife of the Committee on Water Quality Criteria of the National Academy of Sciences.

I am appearing before this committee as a scientist and a private individual. I welcome the opportunity to discuss before this committee the polychlorinated biphenyls, a class of industrial chemicals that have become widespread pollutants in the global environment. The polychlorinated biphenyls, or PCB, are among the toxic substances that would be regulated under the provisions of Senate Bill 1478. I shall therefore try to review briefly our current knowledge of the distribution and environmental effects of PCB. Scientific statements are documented by reference to the original research. The interpretation of these studies and all opinions expressed are necessarily my own, and not those of any public or private organization.

CHEMISTRY OF THE POLYCHLORINATED BIPHENYLS

The structure of chlorinated biphenyls is shown in Figure 1. Chlorine atoms may occupy any combination of the positions of the biphenyl molecule. Many such combinations containing one to 10 chlorine atoms in the various positions are therefore possible. PCB consists therefore of a mixture of chemical compounds. Commercial preparations in the United States, where the Monsanto Company is the only manufacturer, are designated by the trademark "Aroclor" followed by a number that indicates the approximate chlorine composition of the preparation. Thus, "Aroclor 1254" contains approximately 54% chlorine by weight.

PCB is frequently compared to the DDT compounds. Like them it is very insoluble in water, but highly soluble in oils, fats and non-polar liquids. It is highly resistent to degradation by heat, by the biological processes that degrade most chemicals released into the environment, and to chemical attack. These properties have made them useful in many industrial applications.

INDUSTRIAL USE OF PCB

PCB has been used as an additive to sealants, rubber, paints, plastics, adhesives, printing inks, insecticides, and many other commodities. The major uses, however, have been as insulating fluids in electrical equipment and as heat transfer fluids. These are considered to be "closed" systems.

Following the discoveries first made in Sweden (1) and later in other parts of Europe (2,3) and in North America (4) that PCB had become a widespread and locally abundant environmental pollutant, the Monsanto Company recognized the ecological hazard of PCB, and sought to restrict all usage to that of closed systems. The sale of PCB for plasticizer use was discontinued on August 30, 1970. The industrial and hydraulic fluids manufactured by Monsanto were reformulated to exclude PCB, and a high temperature incineration unit was built by Monsanto at Sauget, Illinois, to destroy waste PCB (5).

OCCUPATIONAL EXPOSURE TO PCB

Occupational exposure to PCB has in the past produced a high incidence of chloracne, a severe skin disease, among factory workers. Although known as "chloracne" the skin lesions are not produced by chlorine gas, nor by inorganic compounds of chlorine. The disease has also been prevalent among workers in factories manufacturing chlorinated naphthalenes, chlorinated phenols, and compounds derived from them(6). The literature in this field was very adequately reviewed during the hearings held by this committee in April of 1970 which dealt with the effects of 2,4,5-T on man and the environment. Several of the most important papers in the field were reprinted in the Record of those hearings. The production of chloracne by chlorinated compounds among industrial workers has been extensively studied in Germany. The most potent inducers of chloracne were found to be the chlorinated dibenzodioxins. The chlorinated dibenzofurans were also shown to be potent inducers of chloracne. The structures of both the chlorinated dibenzodioxins and the chlorinated dibenzofurans are shown in Figure 1. Both groups of compounds are highly poisonous (7,8). Very small amounts of the chlorinated dibenzodioxins will produce embryonic defects in chicks, as Dr. Verrett described to this committee last year (9).

The chlorinated dibenzofurans have recently been discovered to be contaminants in some PCB preparations. The toxicity of three PCB

preparations containing approximately the same amounts of chlorine was found to vary widely and to correlated with very small amounts of the benzofurans present as contaminants. The French preparation was found to be more poisonous than that one from Germany, while the American preparation possessed the lowest toxicity when all three were fed to chickens (10,11). The contaminent in the French preparation was estimated to be present in the order of 20 ppm of the PCB preparation (11). So poisonous are the dibenzofurans that the Dutch scientists studying the relative toxicity of various PCB preparations did not wish to work with the pure dibenzofurans.

From the literature it is not clear whether chloracne conditions produced in the past by PCB preparations have been caused by the PCB or by very minute amounts of the contaminants. The Monsanto Company no doubt possesses considerable data on this subject.

A CASE OF NON-OCCUPATIONAL EXPOSURE TO PCB

In 1968 an outbreak of a peculiar skin disease occurred in Japan. More than 300 people were affected. Exhaustive studies were made by a number of Japanese scientists and an extensive series of papers describing their findings has been published. Unfortunately they are in Japanese, but each has an English summary. Several of these are attached as part of my testimony. The results may be summarized briefly. The disease was traced to a shipment of commercial rice oil used for cooking that had been contaminated with PCB used as a heat transfer fluid in the manufacturing equipment. Nine of the women affected were pregnant. Two of the babies were dead at birth, and all showed symptoms of chlorobiphenyl poisoning. None, however, possessed birth defects. Feeding of the oil to mice and monkeys produced comparable symptoms. One of the studies reported that the concentration of PCB in the rice oil was about 200 ppm, and that the amount of oil consumed by a family of four for over a month was in the order of 1.8 liters. The first symptoms appeared in March 1968 and it had been determined that the contamination occured on or about February 5, 1968. It is of interest to make a very rough calculation to determine how much fish or poultry contaminated with PCB at a level of 5 ppm in the edible flesh a person would have to eat to ingest a comparable amount of PCB. On the assumption that half a liter of cooking oil was sufficient to produce the symptoms, 45 pounds containing 5 ppm of PCB of contaminated fish or poultry might produce the same effect.

It is not clear, however, whether the symptoms produced by consuming the contaminated cooking oil in Japan were in fact produced by the PCB or by very small amounts of the contaminants mentioned above. Studies to determine this should clearly receive a high priority.

It is also of interest to compare the levels found in the fat of the people affected with the few values so far obtained for PCB concentrations in human fat in the United States. Biopsy of samples of subcutaneous fat in the affected Japanese ranged from 13 to 76 ppm.

In our laboratory we have measured PCB in nine samples of human milk from Berkeley, California, and 7 samples from nursing mothers in the Los Angeles area. Concentrations in the fat portion of the milk averaged 2.6 ppm in the Berkeley samples, and 2.1 in the Los Angeles samples. Minute amounts were also detected in cows' milk. Confirmation of the occurrence of PCB in human fat by mass spectrometry has recently been reported by research workers of the Food and Drug Administration (12).

BIOLOGICAL EFFECTS OF PCB

An earlier study sponsored by the Monsanto Company of the toxicity of the vapors of two PCB preparations, Aroclor 1242 and Aroclor 1254 (13) demonstrated no injury to several animals at concentrations in the air of Aroclor 1242 below 8.6 micrograms per liter. Reversible degenerative changes in some of the viscera were produced by comparable concentrations of Aroclor 1254. A study reported by the United States Public Health Service in 1944 (14) showed that liver and skin damage was produced in guinea pigs, rats and rabbits exposed to Aroclor 1242 by subcutaneous injections and applications to the skin. The skin lesions were comparable to those found in the chloracne condition in man.

Three studies have recently reported data on the toxicity of PCB to birds. A British study carried out at the Monks Wood Experimental Station (15) found that Aroclor 1254 had only 1/13 the toxicity of DDT to Bengalese finches. The Bureau of Sport Fisheries and Wildlife at the Patuxent Wildlife Research Center has reported toxicity data of 6 PCB preparations to mallard ducks, pheasants, bobwhite quail and Japanese quail. In general, the insecticide dieldrin was approximately 4 times as toxic as any of the PCB preparations, and DDT was approximately 4 times as toxic. Toxicity tended to increase with increasing chlorine content of the PCB (16). A Dutch study (10) found, however, that toxicity of PCB preparations containing the same amount of chlorine varied widely because of different concentrations of chlorinated dibenzofurans present as contaminants. Until the amounts of these contaminants have been measured in each PCB preparation, it is not possible to determine how much toxicity derives from PCB and how much from the contaminants. The recent studies of the Monsanto Company, which I have not yet had the opportunity to examine will no doubt clarify this point.

PCB, like most other chlorinated hydrocarbons, and like many chemicals foreign to the animal body have the capacity to increase the activity of certain liver enzymes (4, 17). This is an adaptive response since the enzymes permit the body to eliminate foreign compounds which are frequently harmful. The more highly chlorinated of the PCB preparations, however, appear to induce the sythesis of these enzymes and the associated supporting material known as the smooth endoplasmic reticulum without being degraded themselves. Some of the naturally occurring compounds in the body, including the sex hormones and other steroids, are then broken down at more rapid rates than is usual. The regulatory mechanisms,

however, cause more of the steroidal compounds to be synthesized so that the levels frequently do not change. The significance of the phenomenon either to the environment or the human health is not clear. One of the Japanese studies of the Yusho disease reported hypertrophy of the smooth endoplasmic reticulum in liver biopsy specimens. Much of the cholesterol synthesized by the liver is produced in the smooth endoplasmic reticulum fraction. A study carried out in Israel (18) on the effect of organochlorine insecticides on serum cholesterol level in people occupationally exposed to those compounds reported that while serum cholesterol increased with age in both the exposed workers and the control groups, the increase was greater in exposed workers. These scientists concluded that the organochlorine compounds induced an increase of cholesterol synthesis, but that the regulatory processes of the body were able to increase the rate of breakdown in younger people. In older people past the age of 45, however, the regulation mechanism worked less well, and cholesterol levels increased.

Several studies (11,19) have shown that PCB may produce symptoms in chicks identical to those produced by chick edema factors. These were discussed extensively in the hearings held by this committee to consider the environmental effects of the herbicide 2,4,5-T (9). It is likely that this effect is caused by the dibenzofuran contaminant (11).

PCB IN THE NORTH AMERICAN ENVIRONMENT

PCB is now universally distributed in the terrestrial, fresh-water and marine environments of North America. On the west coast concentration of PCB in a collection of northern anchovies from coastal waters of southern California was in the order of 1 ppm (20). Concentration in the body of a Peregrine Falcon in California was 65 ppm of the wet weight and 2000 ppm of the body fat (4). All specimens examined of several species of petrels and shearwaters from the Pacific Ocean all contained PCB (4). These are pelagic species which rarely approach land. Sixty-five eggs of the Brown Pelican from Anacapa Island, California, almost all of which were broken when found because of excessive shell thinning contained an average concentration of 210 ppm of PCB in the yolk lipid (21). PCB levels in the coastal waters of southern Florida are somewhat lower. 87 eggs of the same species from Florida, obtained in 1969 and 1970, contained an average concentration of 77 ppm (21). PCB concentrations in the yolk lipids of Ospreys, like the Brown Pelican a fish-eating bird, were highest in Massachusetts and Connecticut with values ranging between 500 and 2500 ppm and lower in New Jersey (22). PCB was found in all plankton analysed from the coastal waters of the north-eastern United States. Concentrations were highest in the waters off Long Island and decreased southwards towards Maryland and eastwards towards the deeper water of the Atlantic (21). The PCB detected in the human milk samples most likely came from residues in food.

MOVEMENT OF PCB THROUGH THE ENVIRONMENT

In November and December of 1970 we measured PCB concentrations in the effluent waters of the major sewage treatment plants of California(23). Several were discharging PCB into the local marine environments at rates equivalent to a ton per year. The high concentrations found in the waters near New York and New England most likely derive mainly from sewage outfalls. Once in an aqueous medium PCB readily passes into the fatty tissues of organisms because of its very low solubility in water and high solubility in fat and oils.

In order to look for the origins of the PCB reported in polar bears by the Canadian Wildlife Service we travelled in the summer of 1970 to remote lakes in the arctic terrain of northern Quebec. These are rarely if ever visited by man. All of the fish so far analysed contained measurable amounts of PCB. One lake trout had 12 ppm in its fat, equivalent to the amounts found in many ocean fish (21). We concluded that PCB, like the DDT compounds, is also dispersed through the atmosphere to fall out in areas remote from the sites of application.

ENVIRONMENTAL EFFECTS OF PCB

In attempting to assess the environmental impact of PCB, the pollution ecologist finds himself in a position he was in up to 1968 in attempting to assess the environmental effects of the DDT compounds. Circumstantial evidence suggested effects upon wildlife populations, but the conclusive scientific evidence was not yet in. The most spectacular and the best documented of the environmental effects of pollutants has been the shell thinning of a number of species of fish-eating and raptorial birds. Several laboratories have investigated the possible effects of PCB upon shell-thinning, but no significant effect has been found. The laboratory of the Bureau of Sport Fisheries and Wildlife has fed PCB to mallard ducks and bobwhite quail but has found no shell thinning effects(16). Similarly the PCB given to doves produced no changes in shell thickness whereas the DDT compound DDE did so(24).

In attempting to determine the environmental pollutants responsible for phenomena such as shell thinning in birds, ecologists have attempted to correlate the magnitude of the effect with the measured concentrations of one or more pollutants. Frequently high amounts of PCB are found together with high amounts of other pollutants such as the DDT compounds. It is therefore difficult to sort out the effects of each pollutant. In examining both west coast and east coast samples of Brown Pelican eggs, containing many different proportions of PCB and the DDT compounds, we have concluded that PCB has no significant effect upon the shell thinning, which correlated well with the measured concentrations of the DDT compound DDE.(25).

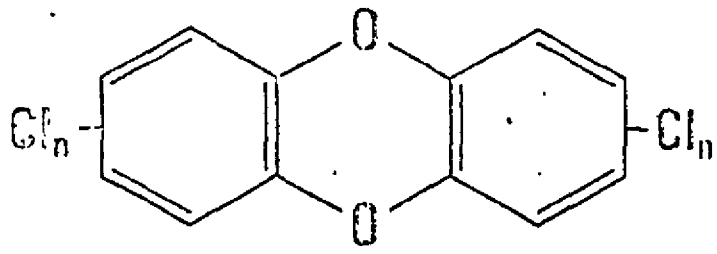
An environmental effect that may be caused by PCB is the lowered fertility of some bird populations and the failure of eggs to hatch.

This effect was noted by a commercial poultry breeder who became alarmed at the low rate of hatching of his eggs. The hens were subsequently shown to have accumulated PCB from contaminated fish meal. The source of this PCB was traced to a leak from the heat-exchange fluid in the factory equipment(26). This observation appears to be inconsistent with the findings reported by the U.S. Bureau of Sport Fisheries and Wildlife (16) that PCB had no significant effects upon the reproduction of mallards and of bobwhite quail. The inconsistency could be explained if the PCB in the heat-transfer fluid leaking from the machinery had higher amounts of the dibenzofuran contaminants.

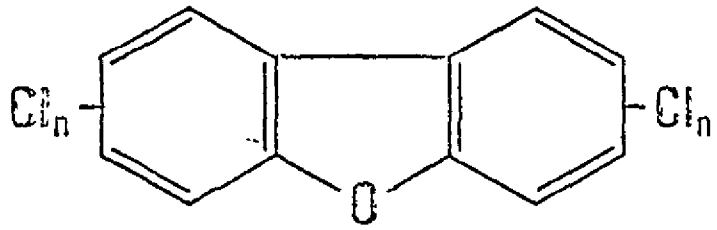
A crucial factor in assessing the environmental impact of PCB is the accumulation rate in "sinks" such as the sea which receive waste materials, including waste chemicals. We can assume that most of the PCB produced is so far still on the land, from which it is slowly leaching into the atmosphere, into sewage systems, and into rivers. It is difficult to estimate the fraction of the PCB now on the land that will eventually enter the sea, because of insufficient data on the amounts of PCB present and on the systems in which it is used. One of the principal research problems in pollution ecology at the present time is to predict future rates of accumulation in the world-wide environment of synthetic chemicals. Most degrade readily to primary materials, but those with certain chemical properties that are produced in large amounts inevitably become pollutants. Necessary data are production figures, reliable estimates of the proportion of total production that will eventually enter the environment, present distribution of the pollutant in the water, air, soil, and biomass, and estimates of the rates of degradation and withdrawal into the sediments of the ocean bottom.

If the amounts of PCB already manufactured are sufficient to increase concentrations in fish in the coastal waters by a factor of 10 over the next 25 or 50 years, the coastal fisheries would be destroyed for the purpose of human consumption. We can not seriously approach this problem, however, without knowing how much is already on the land and how much more is introduced each year. If input were sufficiently low, a steady-state concentration could be maintained in the sea that would be acceptable in environmental terms. We can not yet estimate, however, the magnitude of this concentration. When countries such as Brazil which are now in a state of rapid development begin to manufacture PCB in large quantities, pollution of the sea will undoubtedly become worse.

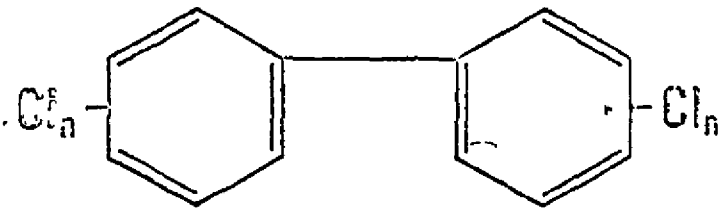
The toxicity of the degradation products of PCB has not been determined, and I am not aware of any studies in progress. It is not known whether the highly toxic dibenzofurans can be derived from PCB by biological or chemical means in the environment.



Chlorinated dibenzodioxin, n=1,2,3,4



Chlorinated dibenzofuran, n=1,2,3,4



Chlorinated biphenyl, n=1,2,3,4,5

Figure 1

Structures of chlorinated biphenyl, chlorinated dibenzofuran, and chlorinated dibenzo-p-dioxin. Chlorine atoms may occurv any of the available positions.

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