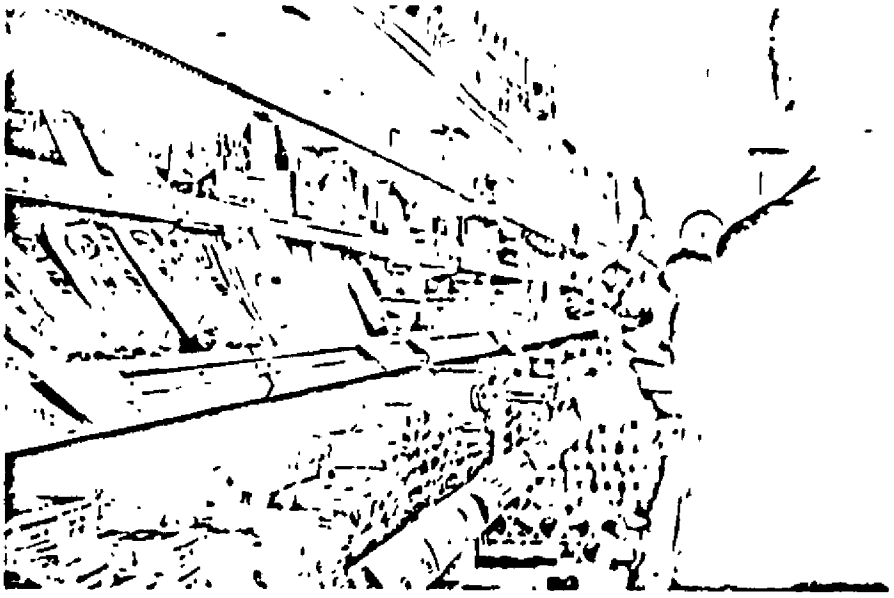
BY KEVIN P. SHEA

PCB

The worldwide pollutant that nobody noticed.



THE POLYSTYRENE COFFEE cup you used this morning, plastic liners for baby bottles, frozen-food bags, bread wrappers, and many other products which come into contact with food contain PCBs, or polychlorinated biphenyls. While recently issued federal standards restrict these toxic materials to much less than 1 part per million in most foods, PCB concentrations in the food containers just named have been measured at as high as 6.6 parts per million. No one knows whether these PCBs leach out into the milk held by baby-bottle liners or the coffee held in polystyrene cups; this is just the latest in a long series of riddles surrounding the use of PCBs.

Polychlorinated biphenyl compounds had been in use for 36 years before their discovery in 1966[1] in the flesh of fish from the Baltic Sea set in motion an intensive worldwide research effort to determine the extent of their distribution in ecological systems and the possible danger their distribution may pose to human health. Since their belated discovery as important global pollutants, PCBs have chalked up an impressive record of finding their way into human food chains, sometimes by totally unexpected routes, and have been responsible for huge economic losses in the food industry and, at least in one case, for a serious epidemic of human poisoning. Although important steps have been taken to prevent such episodes from recurring, there remain a number of questions to be answered regarding the uses of PCBs, their effects on biological systems, and their routes of entry into the environment.

Oddly, it was the experience gained in the pursuit of information about another group of environmental contaminants, the chlorinated synthetic pesticides (DDT and its relatives), that both helped and hindered in tracking down the various pathways through which PCB might enter and spread throughout ecological systems. By the middle 1950s analytical techniques had been developed (gas chromatography) that were able to detect very small amounts of environmental contaminants. Analytical chemists, however, could not, with any certainty, identify a particular compound unless they knew precisely what they were looking for. In the case of DDT, for example, they could compare known samples of DDT with results obtained from chromatographic analysis of environmental samples.

In 1964 the Swedish Royal Commission on Natural Resources em-

ployed Dr. Soren Jensen of the University of Stockholm to begin a study of chlorinated pesticides in the Swedish environment. It was during this study that Dr. Jensen and his colleagues began to see evidence on their chromatograms of several substances totally unknown to them but apparently related to the chlorinated pesticides.[1] At first it was thought that the compounds might be unidentified breakdown products of chlorinated pesticides. To check this idea, Dr. Jensen collected from the Swedish Museum of Natural History feathers from specimens of the white-tailed eagle for each year dating back to 1888. By this technique it was learned that the unknown compounds first occurred in feathers in 1942 and could therefore not be chlorinated pesticides or their breakdown products. It was not until two years later, after a great deal of detective work, that the contaminants were fully verified as polychlorinated biphenyls.

Part of the problem in identifying PCBs was the difficulty during analysis of separating DDT residues from those of PCBs. With analytical equipment widely used until 1965, the identity of certain PCB compounds and one of the breakdown products of DDT could easily be mistaken for each other. In Dr. Jensen's opinion it is quite likely that many DDT analyses made prior to 1965 could have been wrong. However, since 1966, with a steady improvement in analytical methods and a rapidly growing interest in the biological activity of PCBs, it has now been established that PCBs are among the most abundant, persistent, and widely dispersed environmental contaminants.

A comparison of production figures for the U.S. alone sheds some light on the relative importance of DDT and PCBs. During the period 1945 to 1958, U.S. production of DDT was about 933 million pounds.[2] It is certain that nearly all of that production was sprayed into the environment somewhere in the world. During the years 1930 to 1970, total U.S. production of PCBs was approximately one billion pounds, of which, it has been estimated, 780 million pounds has entered the environment via the atmosphere, water systems, and dumps.[3] Add to these figures the production of PCBs in Europe, Russia, and Japan, and the total amount of PCBs entering the environment on a global basis may approach that of DDT. Japan, for instance, produced 114,660,000 pounds of PCBs between 1954 and 1971.

Physiological Effects

While the amounts of PCBs and DDT released to the environment may be similar, other information indicates that PCBs may be the most important in terms of ecological damage. First, PCBs seem to be far more stable – that is, because of their chemical structure they are less susceptible to being broken down by sunlight, water, and microbiological action than are the chlorinated pesticides. When DDT and PCBs are added to an activated sewage sludge and are digested anaerobically, DDT is broken down, mainly to DDD and other compounds, within a few days. PCB compounds, however, will remain intact for as long as 40 days.[3] Anaerobic digestion in sewage sludge is one of the most rigorous techniques for determining the stability of a potential pollutant.

Another difficulty is that PCBs, rather than being one specific chemical, are a mixture of a large number of closely related but different chemicals. For example, a careful analysis of Aroclor 1254,[4] a widely used commercial PCB preparation showed that it is actually composed of 69 different chemical structures. Nineteen of these structures were considered major constituents of the mixture, while 50 were classified as minor constituents. For the most part, analytical and toxicological studies have been conducted with commercial PCB preparations while little attention has been given the individual chemicals that make up these preparations, many of which may have unique toxicological characteristics.

Finally, while PCBs are not quite as poisonous as DDT when consumed orally, they do possess similar ability to alter important biological functions. Like DDT, PCBs have been shown to be potent inducers of liver enzymes in a number of laboratory animals. That is, when fed to laboratory animals in small doses they stimulate the liver to produce enzymes which then act on other body substrates. The clearest measure of this effect is that which occurs in PCB-treated rats when they are subsequently given a barbiturate. A measured amount of phenobarbitol, for example, will produce a standard sleeping time in laboratory rats. The sleeping time is a function of the rate at which liver enzymes break down the barbiturate to non-narcotic substances. When rats are treated with a combination of PCBs and barbiturates, sleeping time is greatly reduced because of the increased enzyme activity brought on by the presence of PCBs in the liver.

The metabolism of barbiturates is of no importance to laboratory animals, but the enzymes induced by this effect are the same ones that metabolize certain hormones and thereby regulate important physiological functions. Their overabundance in the liver at certain times could cause serious disruptions of normal activities such as reproduction. In the past, DDT has been implicated in the disruption of the reproductive cycle of some birds, and it is thought that the mechanism has something to do with hormonal imbalance brought about by the increased production of liver enzymes at the wrong time.

PCBs have other effects on the liver.[4] In both Japanese quail and laboratory rats, dietary levels of 100 parts per million (ppm) fed for two months resulted in enlarged livers, an increase in liver fats, and a sharply reduced liver content of vitamin A. In rats the decrease in liver vitamin A storage was 50 percent that of untreated animals.

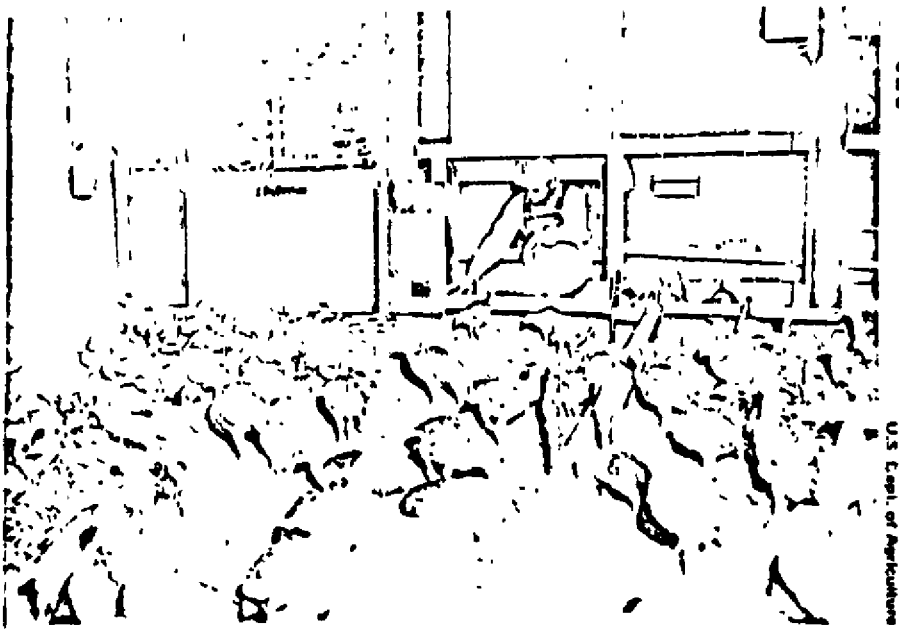
Other subtle physiological effects have been observed in laboratory animals fed various PCB compounds. Interference with the production of adenosine triphosphatase (ATPase) in fish is a good example. ATPase is an enzyme which makes energy available for important biological reactions such as in the production of proteins, muscle contractions, the conduction of nerve impulses, and glandular secretions.[5] At levels as low as 0.6 ppm, Aroclor 1242 inhibited the ATPase system in bluegill tissue by as much as 50 percent.

Still other effects that have been reported include edema (the accumulation of fluids) of the tissues surrounding the heart, porphyria (an abnormality of blood pigment metabolism), thickening of the skin, and a reduced resistance to certain kinds of parthogenic organisms. When ten-day-old ducklings were fed Aroclor 1254 at levels of 25, 50, and 100 ppm in their diet they suffered no apparent clinical symptoms of poisoning, but five days later, when they were exposed to duck hepatitis virus, they suffered significantly higher rates of mortality than did birds that were given the virus but were not pre-exposed to PCBs.[6]

Food Contamination

While a great deal of research is still being conducted on the various sublethal effects of PCBs on laboratory animals, the accidental presence of PCBs in human food and animal feeds remains the most important aspect of their distribution. Over the past few years a number of incidents have been recorded in which significant quantities of PCBs have contaminated human food. Most of these incidents were preventable, but in some cases it is still a mystery as to what was the source of contamination.

In December 1970 the Campbell Soup Company of Camden, New Jersey, through a routine monitoring program discovered excessive PCB residues (up to 268 ppm) in chickens grown in New York State. The state of New York



U.S. Dept. of Agriculture

Excessive levels of PCBs have been found in turkeys and chickens in several cases as the result of contaminated feed.

placed a quarantine around a three-county area and required pretesting before slaughter of all chickens originating from that area. As an interim guideline the Federal Food and Drug Administration (FDA) agreed to allow normal marketing of all chickens containing less than 5 ppm PCB. On the basis of the pretesting program 140,450 chickens were slaughtered and buried. The source of contamination in this case was thought to be plastic wrappers on stale bakery goods that were ground, wrapper and all, for use as a feed.

In July of 1971, Holly Farms of Wilkesboro, North Carolina, notified personnel of the Meat and Poultry Inspection branch of the U.S. Department of Agriculture that they had been alerted to a PCB problem by a decrease in egg hatchability (a typical PCB-poisoning symptom) in their poultry operation. The source of the contamination was traced to a fish meal pasteurization plant in Wilmington, North Carolina, where a leaky heating system was dripping Aroclor 1242 into the finished product. The leak began in April 1971 and continued through July. A total of 12,000 tons of feed were contaminated, and more than 2,000 tons were recalled by the company. In the meantime, broiler and egg producers suffered tremendous losses as a result of contaminated meat and eggs. A single producer was forced to destroy 88,000 broilers, and 123,750 pounds of eggs were removed from the market.

In August 1971, Swift and Company and the Department of Agriculture notified the FDA of excessive levels of PCB found in turkeys in Minnesota. Over 100 flocks in Minnesota, North Dakota, and South Dakota were tested, but only the flock in which the original discovery was made showed high residues (20 ppm). In this case, over one million turkeys approaching market weight were held off the market until the residue levels were reduced to less than 5 ppm. The source of the contaminant was not determined.

In a routine food surveillance program, Baltimore health authorities discovered excessive PCB levels in milk, and the dairy farms involved were taken out of production by state officials. An investigation revealed that the source was spent transformer fluid (a major use of PCBs) used as a carrier for a herbicide sprayed on a power line right-of-way near Martinsburg, West Virginia.

Finally in Ohio, Florida, and Georgia, contamination of milk was traced to a PCB-containing sealant used to seal the inside of silos. The silo coating was composed of 11 percent PCB, some of which had migrated into the silage and was subsequently consumed by dairy cattle.

Yusho Disease

While North Americans have so far avoided a massive outbreak of human poisoning from PCB-contaminated food, Japan has not been so lucky. In October 1968 a number of reports of a skin disease similar in appearance to chloracne (a disease often observed in workers who handle PCBs and other chlorinated aromatic compounds) began to appear in Fukuoka perfecture in western Japan. It wasn't long before twenty other perfectures in the same general area also began reporting similar cases, and it was soon apparent that a full-blown epidemic was underway.[1] Aside from the symptoms of chloracne, which include acne-like skin eruptions and excessive pigmentation of the skin, patients afflicted with the disease suffered from increased eye discharges, swelling of the upper eyelids, headaches, vomiting, diarrhea, fever, visual disturbances, and a number of other unpleasant effects. A team of specialists in medicine, pharmacology, agriculture, and engineering was quickly assembled from the faculties of the University of Kyushu, and the cause of the massive outbreak (1,057 patients had been reported by 1971) was soon discovered. By carefully interviewing both individuals suffering from the disease and a matched number of healthy individuals, it was found that of 60 personal traits used to compare healthy and affected individuals only one trait, that of eating fried foods or tempura daily, differed greatly between healthy and stricken people. It was also discovered that 96 percent of the diseased individuals came from households where a rice-bran oil produced by a company in Kitakyushu City and known as K rice oil was used regularly. Further investigation showed that 143 of 155 patients who had used the oil purchased it from a lot that had been produced and shipped between February 5 and 15, 1968. Finally, analysis of some of the recovered K rice oil showed that it contained from 2,000 to 3,000 ppm of Kanechlor 400, a Japanese produced PCB that was used in the heating system at the K rice oil production plant in Kitakyushu City. Since the cause of the outbreak was discovered the disease has been known as "Yusho" or oil disease.

The effects of this outbreak are still evident in Japan. By the summer of 1970 half of a closely studied population of 159 individuals seemed to be improving, while the other half showed no improvement, and 10 percent seemed to be worsening. There is also some evidence that babies born to women who had consumed the contaminated oil while pregnant exhibit some of the symptoms of Yusho. Thirteen women, eleven of them suffering from Yusho, and two who had no symptoms but had consumed some K oil, gave birth to ten live born and two stillborn children between February and December of 1968. Nine of the ten children showed positive signs of Yusho, and Japanese authorities plan extensive follow-up studies of the children to detect any physical or mental impairment that may develop in later years.

Ironically, six months prior to the outbreak of Yusho, Japanese health authorities had a clear warning that such an epidemic was possible. Beginning in February an epidemic of chick edema

disease occurred in the same western Japan perfecture. More than two million chickens were involved and over 400,000 died. The cause of the disease proved to be PCB-contaminated feeds made by two feed manufacturers that had purchased "dark oil" for use as an ingredient in their products. The "dark oil" was produced by the same company that produced the poisonous K rice oil implicated in the outbreak of Yusho.

Regulating PCBs

Since the repeated episodes of food contamination and the outbreak of Yusho in Japan, a number of actions have been taken both by government agencies and the major manufacturer of PCBs in the U.S., the Monsanto Chemical Company, to prevent further incidents

Before 1971, about 40 percent of the PCB compounds produced in the U.S. were used in applications where losses to the environment were probable These included plasticizers, lubricants, hydraulic fluids, carbonless carbon paper, and adhesives.[10] Since that time, however, Monsanto has agreed to sell PCB compounds for use only in closed systems from which spent PCBs are theoretically recoverable The company has also constructed a high-temperature incinerator, in which Monsanto will destroy spent PCBs for customers that purchase large amounts of the materials for use in electrical transformers and capacitors The expense of shipping the materials to the company and the cost of incineration must be paid for by the customer.

In January 1973 the National Electrical Manufacturers Association published their "official guidelines" for handling and disposing of capacitor and transformer grade PCB compounds. In the guidelines seven different locations for disposal of PCB compounds were listed, two of which have high-temperature incinerators. The other five are described as having "scientific landfills" for PCB disposal. In one facility at Sheffield, Illinois, the disposal system was described in a telephone conversation as an open pit into which contained hazardous chemicals are placed and then covered with soil. While placing spent PCBs in landfills is far cheaper than incineration at high temperatures, the security of materials handled in this way is open to question In 1970 alone, 36 million pounds of PCBs were disposed of in dumps and landfills, and in 1972, sales of PCBs for use in transformers and capacitors were expected to be an additional 50 to 60 million pounds. Since high temperature incineration is expensive and limited (the Monsanto incinerator has an annual maximum capacity of one million pounds) it is likely that much of this production will eventually be disposed of in landfills

To prevent further contamination of food and animal feeds, the FDA recently (July 6) issued regulations placing restriction of the use of PCBs in the manufacture of food and feeds and also issued tolerance limits on the amount of PCBs that are acceptable in food as unavoidable contaminants[11]

The new regulations forbid the use of PCBs for use in new equipment in food processing plants and require PCBs used in old equipment to be replaced with non-PCB-containing materials PCBs are also to be eliminated from surface coatings and lubricants used for handling or processing food Similar regulations were to go into effect by September 4, 1973, for plants processing or handling animal feeds.

Restrictions were also placed on equipment used to manufacture paper food containers and a tolerance limit of 10 ppm was placed on paper materials intended for use in packaging both human and animal foods Manufacturers of plastic food containers were also included in the regulations excluding PCBs from use in equipment used to manufacture food wrappings, but no tolerance on plastic wraps was set The regulations specifically point out that no PCBs whatever are allowable in plastic food wraps either by purposeful or accidental introduction.

The new FDA regulations, if strictly enforced, will almost certainly reduce human exposure to PCBs from accidental contamination, but they are unlikely to have an immediate effect on unavoidable contamination from environmental sources. For that reason the FDA has also established tolerance limits for foods and animal feeds in which a certain amount of PCB contamination is inevitable because of the already existing amounts of PCBs in the environment. The tolerance limits are: milk, 2.5 ppm (fat), finished dairy products, 2 5 ppm (fat), poultry, 5 ppm (fat); eggs, 0.5 ppm; finished animal feeds, 0.2 ppm; animal feed components, 2.0 ppm (fish meal, and so on); fish and shell fish, 5 ppm.

One area the regulations do not cover, however, is containers and wraps for home use. Whether or not there is widespread contamination of these materials is not known, but an analysis[12] of a number of such items in 1971 indicates that it is quite likely The following levels were found in various plastic containers other than commercial food wraps: a plastic bag, 2.4 ppm; a plastic bottle used to contain an eyewash, 3.7 ppm; a polystyrene cup, 6.6 ppm, a baby-bottle liner, 2.7 ppm; a frozen-food bag, 4 8 ppm; a sandwich wrap, 3 2 ppm, a sheet plastic wrap, 3.7 ppm. None of these items are included in the regulations covering food wraps, and since none of the materials are actually plasticized with PCBs it is likely that the contamination is a result of various PCB uses in the processing machinery.

A further complication in tracking down all sources of human exposure is the fact that PCBs are now manufactured in Great Britain, France, Germany, the Soviet Union, Japan, Spain, Italy, Czechoslovakia, and the United States. It is virtually impossible at this time to determine whether or not finished consumer items that might lead to human exposure are being imported into the U.S., but the FDA plans to investigate this possibility in the future

While both regulatory and voluntary actions were long in coming, they will quite likely reduce the immediate hazards associated with PCBs both to human beings and ecological systems On the other hand, because of the enormous amounts of PCBs already circulating in the environment, it will be some time before they will be considered insignificant contaminants. □

NOTES

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