PCB General

CONSUMER HEALTH AND PRODUCT HAZARDS - CHEMICALS, ELECTRONIC PRODUCTS, RADIATION

Vol. 1 of THE LEGISLATION OF PRODUCT SAFETY

Samuel S. Epstein and Richard D. Grundy, editors

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Dedicated by Samuel S. Epstein to his children:
Mark, Julian, and Emily

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Polychlorinated Biphenyls Probably no other class of organic chemicals in industrial use more clearly illustrates the need for effective environmental control of industrial chemicals than the polychlorinated biphenyls (PCB's). While PCB's have a history of use (9) dating back to 1929, it was not until 1966 that they became suspect as widespread environmental contaminants. Since then, PCB's have been discovered as contaminants in food (10), wildlife and fish (11), marine and fresh water, and in human tissues (12). They now appear to be one of the most widely dispersed and abundant environmental contaminants.

While it is clear that PCB's are widely dispersed in the environment, it is less clear how it happened. Given their wide application in industry, the pathways through which they can reach the environment are numerous. Although no quantitative source data are available, the following appear to be the most important (13): open burning and incineration of industrial and municipal wastes; vaporization from paints, coatings, and plastics; municipal and industrial sewers; accidental spills; disposal of sewage sludge; and migration from surface coatings and packaging materials into foods and feeds. It also is very likely that a significant amount of PCB's used in "closed"

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systems such as capacitators, transformers, and hydraulic systems have been released, either by inadvertent leakage from such devices, by disposal directly into sewer systems, or by migration from land fills where spent devices containing PCB's have been buried.

It has been estimated that over the past 40 years about 30,000 tons of PCB's have been lost to the atmosphere, 60,000 tons to water systems, and 300,000 tons to dumps (14). These estimates are crude, and a recent report adds an element of confusion. It has recently been suggested that DDT can be converted to PCB's by irradiation with ultraviolet light of wavelengths found in the lower atmosphere (15). If these observations are confirmed, estimates of the total amount of environmental PCB's may be drastically changed.

Because of their unusual properties -- stability at high temperature, nonflammability, cooling and insulating capacity -- PCB's are now alleged to be essential or (at least for the time being) nonreplaceable in the electrical industry. (Although the silicones do appear to be possible substitutes.) This fact, together with a lack of adequate biological and ecological information about PCB's, has made dealing with them on an ad hoc basis even more difficult. Their value to the electrical industry can be detailed at length, while the "externalized" costs of those uses, especially in terms of human health, is at present unclear.

The most extensive adverse human experience with PCB's began in Japan (16). In October 1968 an outbreak of a peculiar skin disease known as chloracne was reported in Fukuoka Prefecture. Shortly after, twenty other prefectures in western Japan reported the disease and by August 1971 some 1,057 cases had been reported.

The initial signs and symptoms of the disease are swelling of the upper eyelids and eye discharge, acneiform skin eruptions, follicular accentuation and skin pigmentation, disturbances of digestive functions, and numbness and other neurological disorders. No sex differences in the distribution of the disease were noted, but it seemed to occur within particular families. For instance, 325 patients examined in Fukuoka Prefecture between October 1968 and January 1969 belonged to 112 families.

Soon after the epidemic became evident, a study group composed of staff members of various faculties at the University of Kyushu was formed to determine the cause of the epidemic and to find means to control it. A thorough case study was undertaken of 121 patients and 121 healthy controls, matched by age and sex, in which individuals were asked 60 questions

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concerning their occupation, medical backgrounds, health status, diets, and other personal habits.

It was discovered that the only significant difference between the patient's and the control's habits was the fact that a greater number of patients reported eating fried foods or tempura nearly every day. A further test matched 69 households with patients against 207 households without patients; 96 percent of the patients' households reported using a brand of rice oil designated as "K rice oil," while only 31 percent of the control households reported such use.

It was eventually established that the source of the disease (now called Yusho) was a supply of rice oil produced by a single company and shipped between February 7 and 10, 1963. The oil had become contaminated with as much as 3,000 ppm of PCB's during the process of heating the oil at reduced pressure to remove unwanted odors. It is believed that the PCB's leaked from the heating pipe and contaminated the oil.

In the summer of 1970, a mass clinical examination of Yusho patients was conducted. About 50 percent of the patients showed some improvement, despite the fact that no cure is known for the disease; the remaining 50 percent of the patients showed no improvement, with 10 percent of this group apparently deteriorating.

Ironically, six months before the outbreak of Yusho, parts of western Japan were affected by an outbreak of a disease in chickens as a result of which 400,000 birds died. This outbreak was eventually traced to feeds in which "dark oil" was used as an ingredient. It was further discovered that the "dark oil," which was contaminated with 1,300 ppm of PCB's, was purchased between February 6 and 27 from the same company that produced K rice oil.

While the United States fortunately has been spared the suffering associated with a massive outbreak of Yusho, there have been numerous cases paralleling the outbreak in Japanese chickens. Some of these incidents are rather bizarre and clearly indicate the need for overall regulation of toxic materials.

In one case, the state of New York placed three counties under quarantine when surveillance data collected by the Campbell Soup Company indicated excessive PCB levels in chickens raised in those counties (13). The federal Food and Drug Administration (FDA) advised the State and the United States Department of Agriculture (USDA) that the distribution of poultry containing less than 5 ppm of PCB's would be acceptable, implying of course that anything exceeding that level would be subject to confiscation. Apparently all parties,

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including the producers, agreed to this arrangement. On the basis of subsequent analyses performed by the State of New York, 140,450 chickens had to be slaughtered. It is interesting to speculate on the outcome of the situation had the producers been less cooperative. It is certain that the rationale for allowing PCB concentrations of no more than 5 ppm in chickens would be difficult to defend on scientific grounds. It is just as certain that defending the harmlessness of this level of PCB's would be equally difficult. The source of PCB's in this case was thought to be plastic bakery wrappers, as stale bakery goods were ground up, wrappers and all, and used as chicken feed.

In another case (13), an estimated 12,000 tons of fish meal were contaminated with PCB's by a leaky heating system in a fish-meal pasteurization plant at Wilmington, North Carolina, between April and July of 1971. Of this, about 2,000 tons were recalled from the market. Samples of the meal indicated PCB contamination ranging from 14 to 30 ppm. Not only did this result in the recall of fish meal, but it also affected the hatchability of eggs in at least one large poultry operation that used the meal as a poultry ration. Furthermore, one broiler chicken producer had to destroy 88,000 birds, also the result of contaminated rations. In these incidents, all actions were based on the FDA's 5 ppm guideline.

Further incidents involved poultry in Oklahoma, Minnesota, Mississippi, and California; milk in West Virginia, Ohio, Florida and Georgia; and meat meal in Illinois. The source of milk contamination in Ohio, Florida, and Georgia was thought to be from a PCB-containing sealant used in silos (13).

Because of these unexpected incidents, the FDA has initiated an extensive national surveillance program to monitor animal feeds and milk on a state-by-state basis.

Aside from the direct contamination of consumer products with PCB's, there are the longer-range environmental effects. Because of the stability of PCB's, even a total shutdown of all sources of environmental contamination would not result in an immediate decrease in PCB levels. Here the picture is even less clear than it is with immediate human health problems. While laboratory studies indicate a number of potentially adverse effects of PCB's, such as microsomal enzyme induction, estrogenic activity, and immunosuppression (17), there is a noteworthy sparcity of toxicological data; moreover, the eventual effect of even the present burdens of PCB's on the environment is far from clear.

In light of the aforementioned experiences, the Monsanto Chemical Company, the sole producer of PCB's in the United

States, has agreed to voluntarily limit the distribution of PCB's to those uses which are considered "closed systems." However, PCB's are now also manufactured in Great Britain, France, Germany, the USSR, Japan, Spain, Italy, and Czechoslovakia.

What is clear, as will be subsequently discussed, is that existing federal authority is inadequate to restricting more PCB's from entering the environment. Additionally, the federal authority cannot at present control the importation of PCB's manufactured in foreign countries. The result is that in the United States there exists an almost universal presence of PCB's in the environment which, under existing authority, can be controlled only on a piecemeal basis.

Phthalic Acid Esters Another group of chemicals to gain recent attention of environmentalists (18) are the phthalic acid esters (PAE's). Attention was first drawn to PAE's when, following a widely publicized report in 1970, they were found as a contaminant in whole blood stored in polyvinyl chloride transfusion bags (19). Before long, other scientists began to report the occurrence of PAE's in a diversity of environmental samples: water taken from the Charles River in Massachusetts (20), fish tissue from the Great Lakes (21), and heart tissue from cows (22).

The mechanism by which PAE's enter the environment is unclear. A very small amount of the total production is applied directly to the land in the form of pesticide carriers; evaporation from plastics is another source; and some PAE's are probably released during the manufacturing process. A number of reports indicate that some PAE's also are produced naturally by a few species of plants, fungi, and bacteria. These reports, however, are now open to question since contamination of laboratory equipment during analysis has been common.

Phthalates are incorporated into polyvinyl plastics to give the end product its desired pliability. Oddly, however, the plasticizer does not actually combine chemically with the polymer resin, but simply acts to "lubricate" the polymer molecules in such a way as to allow them to be flexible. The plasticizer materials can sometimes account for as much as 50 percent of the entire weight of a plastic product. These phthalate plasticizers are able to migrate from the plastic into either the air or a surrounding liquid.

PAE's are one of the most widely used plasticizers. In 1968, there were at least 10 different phthalate compounds in production, totalling 715 million pounds (23). This is not total production, however, because information about many

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phthalate compounds is proprietary information. Therefore the actual substances are not identified; these unidentified PAE's account for another 125 million pounds annually (23).

The PAE's generally have been found to be of very low toxicity, when administered orally or by injection. When some eight phthalates were examined (24), their acute LD_{50}, when injected into the peritoneum, ranged from 1.58 grams per kilogram of mouse body weight to 14.19 grams per kilogram. In other words, by this route these compounds were virtually nontoxic from the standpoint of immediate effects. In the same study, however, some observations were made on more sensitive indices of subacute toxicity (24).

A factor complicating toxicological research on phthalates has been the difficulty in their precise chemical characterization (25). It was found, on careful analysis, that besides commercially prepared medical-grade plasticizer [di(2-ethylhexyl)phthalate (DHEP], several other related chemicals also were found in blood stored in plastic bags (25). The other contaminating chemicals were thought to be closely related compounds (homologues) of the plasticizer that became incorporated into the product during the production process. Most of the homologues were about as toxic as their plasticizing relatives. Special note was made of the fact that specially synthesized pure di-n-decyl phthalate was not toxic, while a commercial preparation was extremely toxic to mice. In conclusion, the authors stated (25) that:

"A pure plasticizer may have no demonstrable toxic response, but the presence of related homologues and isomers could add toxic potential to the plasticizer. To this possibility one must also include potential toxic effects from degradation products present or forming in the plasticizer during manufacture of a plastic device, or occurring after the device had been stored under various conditions for undetermined amounts of time."

More recent experiments have indicated that some PAE's were teratogenic (causing birth defects) following administration to laboratory animals (26). Eight different PAE compounds were injected into pregnant rats (at doses of 0.3 to 10 milliliters per kilogram of body weight) on the fifth, tenth, and fifteenth day of gestation. Among the effects induced were fetal death, decreased fetal size, and abnormalities such as twisted hind legs, fused ribs, and absence of eyes or tails.

Two PAE compounds were also tested for mutagenic (causing genetic damage) effects in male mice (26). A single dose of

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DHEP was injected into male mice prior to mating. Litters from females mated to DHEP-treated males showed a significantly higher percentage of fetal deaths at all dosages tested. These results indicate that PAE's induce dominant lethal mutations.

Here again, as with PCB's, is a case in which a group of chemicals have become widely dispersed in the total environment before they were recognized as a potential hazard. Only minimal control has been exercised over the use of PAE's and those controls only apply to situations where the material comes in direct contact with food (27). It is likely that exposures from other sources, especially consumer products made from polyvinyl chloride plastics, are of much greater significance. Automobiles, for example, whose interiors are lined with plasticized materials, are probably a major source of inhaled PAE's and data on inhalation exposures are absent from the available toxicological literature.

Freons Another class of synthetic organic chemicals, in widespread use before it came under careful examination, is the fluoroalkanes (Freons). These chemicals are almost universally used as the propellant in aerosol products. Yet they are now under strong suspicion. Since the 1950's, there has been a steady increase in the numbers of sudden deaths among asthmatics that is closely related to the increased use of pressurized aerosol bronchodilators. At present, the precise cause of these sudden deaths is unclear, but evidence is accumulating that they may be the result of cardiac arrhythmias (28). A recent FDA report indicated that 11 sudden deaths have been associated with the use of one of these products (29).

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