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persons who had been exposed in earlier years. There is no evidence that the presence of DDT in human fat is harmful at the levels reported in the general population.

THE POLYCHLORINATED BIPHENYLS (PCBs)

The PCBs are a family of chemicals known as chlorinated biphenyls and naphthalenes and were used in large quantities early in World War II to provide nonflammable insulation for electrical equipment. The products were sold under the trade name "Halowax," and a number of workers who were exposed to their vapors and dusts died from liver damage. The chlorinated naphthalenes proved to be so toxic to humans working with them that their use as insulating materials was voluntarily discontinued, but the chlorinated biphenyls now known as PCBs (polychlorinated biphenyls) continued to be used in electrical equipment, and for numerous other purposes.

The PCBs comprise more than 200 chemical species, each distinguished by differences in the number of chlorine atoms and the position in which they have been incorporated into ringlike organic molecules known as "phenyls." Large-scale production of these compounds began around 1930 and reached a maximum in 1970, afterwhich production was voluntarily curtailed by the Monsanto Chemical Company, the principal manufacturer, because of developing concern about the effects of the PCBs on human health and wildlife. Sale of PCBs was thereafter limited to sealed systems from which environmental contamination could be minimized.

During the many years in which they have been manufactured, the PCBs had been found to have remarkable properties that favored their use for many purposes. Because the compounds are excellent electrical insulators and are not flammable, they have been used as dielectrics in transformers and large electrical condensers. These are sealed systems in which the PCBs can remain in place for the life of the equipment and can then be purified for reuse when the equipment is scrapped. However, some leakage can be expected to occur during the lifetime of the equipment, and PCBs may eventually enter the environment via sewers or surface runoff to streams.

PCBs are also used in small electrical capacitators, such as those in fluorescent-lamp boosters, that have a relatively short life and ultimately find their way to the scrap heap for incineration or land disposal. The compounds are also used in lubricating and cutting oils, as plasticizers in paints, in pesticides, in the manufacture of paper, adhesives, and plastics, as well as in heat transfer and hydraulic fluids. The many uses of the PCBs, plus the fact that they are highly

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stable chemicals, assured their role as ubiquitous environmental contaminants.

The danger of PCBs to humans came to worldwide attention as a result of an accident in Japan in 1968, in which rice oil was contaminated by PCBs leaking from a heating system.[30] The first cases had unusual skin complaints, and upon investigation it was found that all patients had consumed rice oil that was contaminated by PCBs. The disease was soon found to involve more than skin complaints and was given the name "yusho" (oil disease). The patients suffered from fatigue, nausea, swelling of the arms and legs, and liver injury. Babies born to mothers with yusho were smaller than normal. In addition, their subsequent growth and development were diminished, all of which indicated that the PCBs had caused fetal injury after being transported through the placenta. By 1973, about 1,200 cases of yusho had been reported as a result of this mishap.[31]

Shortly after the yusho incident occurred, it became apparent that the analytical procedures used to detect DDT were being affected by the presence of another class of chlorinated organic compounds whose behavior in the analytical procedures was very similar to that of DDT. The results of tests for DDT were being biased by the presence of substances which were soon found to be PCBs. Thus, coincidentally with worldwide awareness of the human toxicity of PCBs, it was found that they, like DDT, were persistent in the environment and were present as contaminants of fresh water and marine organisms.

Like DDT, the PCBs tend to accumulate in the fatty tissues of plants and animals. They are also capable of biomagnification: PCBs, at each step in the food chain, can be concentrated from ten to 100 times the concentration in the previous step. At the top of the food chain, in the sea birds and large carnivorous fish and sea mammals, the concentration can be 10 million times that in the surrounding water.[32] As a result, although the concentration of PCBs in the ambient water might be only one part per billion, fish from the waters and bays of Europe, North America, and Japan contain several parts per million. Concentrations as high as 800 parts per million have been found in the fish from some inland waterways.[33] The higher levels tend to be found near centers of industrial activity, but worldwide contamination exists at levels on the order of one part per million.

The toxicity of PCBs is variable, depending on the biological species and the particular molecular form of PCB to which they are exposed.[34–36] Some organisms are able to tolerate concentrations of PCBs that kill others. One of the most susceptible is the pink shrimp, among which complete mortality can be produced in water containing

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100 parts per billion. The stage of development is also important: Many fish that can tolerate several parts per million as adults are seriously affected in earlier stages of growth. Among the mammals, the mink has proved to be markedly sensitive, a fact that was discovered in 1967 when commercial mink ranchers fed their stock food prepared from Coho salmon contaminated by PCBs in a Lake Michigan tributary. Subsequent laboratory experiments demonstrated that PCBs were extraordinarily toxic to mink, and even more so to the embryos.[37]

The acute toxicity of PCBs to humans was demonstrated in the yusho incident, but in that instance the levels of exposures were high and therefore do not shed light on the toxicity of PCBs to humans at the levels "normally" found in the environment. The main source of PCBs for humans is the diet, and FDA market-basket studies since 1969 have shown that fish are the major source of PCBs.[38,39] The daily intake of PCBs by the average teenage American male has dropped from 15 (micrograms/day) micrograms per day in 1971 to about 8.7 in 1974 and 1975. This is due to reduced contamination in food other than fish and poultry, which have not changed significantly.

Studies in Canada[40] and the United States[41] have shown that PCBs tend to concentrate in body fat and, because of the high fat content of human milk, can be passed in this way to breast-fed babies. The actual PCB content of the fat in human milk is approximately the same as in other fatty tissues of the body and has averaged about 1.7 parts per million in North America during the period between 1969 and 1974. Samples containing as much as ten parts per million have been reported. People who eat fresh fish, particularly from lakes known to be contaminated with PCBs, tend to have the highest levels.

There is reason to be concerned about these reports. Experiments with rhesus monkeys have shown that severe PCB intoxication occurs in the rhesus infants born to mothers fed PCB-contaminated food. The levels of PCBs in the rhesus milk are comparable to the highest values found in human milk in the United States and Canada. Nevertheless, the consensus among informed individuals is that the presence of PCBs in human milk does not warrant changes in nursing practice.[42] However, an advisory committee to the New York State Health Department recommended that if mothers have been consuming fish from waters known to be contaminated with PCBs on a regular basis, nursing should be discontinued after three months.[43]

It has also been found that 2.5 to five parts per million of PCBs in the diet of rhesus monkeys caused toxic symptoms to develop within one to two months.[44] The rhesus monkey is a relatively sensitive species, and is affected at concentrations as low as 1 percent of those

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that affect rats.[45]

On the assumption that a teenager consumes 2,000 grams of food per day, his daily intake of PCBs is of the order of 1 percent of that known to have caused toxic symptoms in monkeys. This may seem to be a safe level, but it should be noted that the market-basket estimates are applicable to "average" people, and not to people who consume large quantities of fresh-water fish. Moreover, since PCBs are passed to breast-fed infants, infants may be relatively heavily exposed.

The infants of rhesus monkey mothers fed about 2.5 parts per million of PCB in their diet were undersized at birth, generally debilitated, and subject to frequent infections. We cannot be certain that somewhere in the United States there are not nursing mothers with a preference for fish contaminated with PCB, whose nursing babies are showing the effects of PCB toxicity.

The basic concerns about PCBs are over the relative susceptibility of humans compared to rhesus monkeys, and to what extent there are deviations from the national average. There have been no reports of PCB poisoning in nursing infants, but there had been no epidemiological studies as of the end of 1977. Only by careful and sufficiently extensive studies can assurances be gained that occasional cases of PCB poisoning are not occurring in the infant population. The fact that these studies have not been done is an example of the ineffectiveness of the means for epidemiological research for environmental health in the United States.

The PCBs are a particularly difficult class of compounds to evaluate from the toxicological point of view. As noted earlier, their chemical complexity is such that they exist in more than 200 forms (isomers), and the toxicity of a particular isomer, as well as the rate at which it will degrade in the environment, depends on the chemical species involved. A further complication arises because PCBs can be transformed into a class of highly toxic impurities, polychlorinated dibenzofurans (PCDFs). These PCDFs can be formed in the process of manufacture or by subsequent degradation processes. It is known that the contaminated rice oil that caused yusho in Japan contained high levels of dibenzofurans, and it has been suggested that because the PCBs were used as a heat-transfer medium, the high temperatures may have degraded the PCBs to produce PCDFs. The toxicity of PCBs may thus depend, not only on their particular chemical identity, but also on the extent to which they are contaminated with dibenzofurans.[46] The role of PCDFs in the epidemiology of PCB poisoning is not well understood. Unfortunately, the PCDFs are extremely difficult to measure in trace amounts and very few studies on the effects

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of PCDFs have been reported.

In 1973, the FDA issued regulations that banned the industrial uses of PCBs in establishments manufacturing, handling, or storing human food, animal feeds, and paper food-packaging materials.[47] The regulations also proposed temporary limits on PCBs in food. The most important limitation was that pertaining to fish: a maximum of five parts per million in the edible portions. The Canadian authorities subsequently reduced the permissible PCB concentration in fish to two parts per million, following which the United States FDA proposed a similar change.[48]

The PCB content of a fish depends to a large extent on its feeding habits. For example, the American shad spends most of its life in the open ocean but comes to the estuaries to spawn. Since the shad are usually caught for market on their way to their spawning grounds, they have relatively low levels of PCBs. Catfish and eels, which tend to feed among the sediments of the estuaries, are among the fish that contain the highest concentrations.

It is ironic that, at the very time when benefits were being expected from the enormous expenditures for the clean waters programs of many states, the PCB and other similar problems have developed and, for some time to come, will deny the use of important waterways for some of the very purposes for which billions were spent for pollution control. As an example, the total expenditures by local, state and federal governments for water-pollution control in the Hudson River estuary were in excess of 3 billion dollars by 1976. However, the new sewage-treatment plants were not designed to remove toxic substances that accordingly are allowed to enter the lakes, rivers, or bays along with the treated sewage.

Because of the high concentrations of PCBs in Hudson River fish, it was decided to ban commercial fishing for an indefinite period starting late in 1975. The principal species affected was the striped bass, which is highly prized by both sports and commercial fishermen. Sports fishing was allowed, but the fishermen were cautioned not to eat more than one fish meal per week and were forbidden to sell the catch. Eels were not allowed to be taken by either sportsmen or commercial fishermen. An exception to the ban on commercial fishing was made in the case of shad which, for the reasons noted, proved not to be excessively contaminated.

The main sources of PCB contamination of the Hudson were believed to be two General Electric Company plants at Fort Edward and Hudson Falls, located in the upper reaches of the river. A prolonged hearing resulted from charges by New York State that the company polluted the waters of the river to such an extent as to be

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injurious to wildlife, and that the company should eliminate its discharge of PCBs and "restore the health of the Hudson River and other natural resources to the extent that PCB discharges have despoiled them. . . ."[48] The hearing that followed illuminated the history of PCB discharges by the company in a manner that was neither a credit to the company nor to the state and federal governments. As stated in the opinion of the hearing examiner, "these unlawful consequences are the product of both corporate abuse and regulatory failure: corporate abuse in that G.E. caused the PCBs to be discharged without exercising sufficient precaution and concern; regulatory failure in that G.E. informed the responsible Federal and state agencies of its activities, and they too exercised insufficient caution and concern. . . ."

The General Electric Company had purchased more than 41,000 tons of PCBs of various types from the Monsanto Chemical Company since 1966. The material was used in the manufacture of electrical condensers, and during much of the time it used them, the company had been discharging an average of about 30 pounds per day of PCBs to the river.

As a result of the 1972 amendments to the Federal Water Pollution Control Act, the states were required to adopt control programs based on effluent limitations. As a result, New York State required that a certificate be obtained that authorized the discharge of individual pollutants. A federal discharge permit was required following certification by the state. General Electric applied for such a permit in late 1971. Two years later, the state certified that no effluent standards were applicable to the proposed discharges, and the U.S. Environmental Protection Agency regional office issued a draft permit that proposed to authorize a daily average discharge of 30 pounds from the two plants. The draft permit also proposed that the chlorinated hydrocarbon discharges be reduced to zero within 21 months of the permit's issuance.[49]

A requirement for zero discharge is unrealistic. Large industrial plants can reduce their discharges to any desired level short of zero. The discharge of 30 pounds per day could have been reduced to three pounds per day, .03, .003 pounds, or lower, but some finite discharge would remain that could be detected. In practice, a waste-discharge limit should be based on the risks to human health and wildlife, technical feasibility, the characteristics of the receiving body of water, and cost. If the plant is to operate, the permissible discharge must be expressed in finite terms. It can be reduced to whatever level is required, but not to zero.

When the permit was finally issued in December of 1974, it ordered

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that the discharge of PCBs be reduced to 3.5 ounces by mid-1977. The position that General Electric took at the hearing rested substantially on the premise that since the Department of Environmental Conservation and the Environmental Protection Agency were fully aware of the PCB discharges, the discharges could not be illegal. The hearing examiner commented that "G.E.'s argument has more than superficial appeal."

By 1972 it was well known by the State Department of Environmental Conservation that PCBs were present in striped bass in concentrations ten times or more the FDA's limit of five parts per million. The high levels persisted through 1977, despite the fact that G.E. quickly reduced its releases. The PCBs were deposited in the sediments, where they have served as a reservoir for pollution of the biota, and it is not known how long it will take for the river to cleanse itself.

The state had the power all along to correct the problem, the existence of which was known six years before the matter received public attention. Since the matter of PCB pollution was a national problem, the state looked to the federal government for policy, but received only a guideline for the maximum permissible PCB content of fish, and no detailed instructions as to how the guideline should be interpreted. The federal government had the power to stop the sale of fish in interstate commerce, but failed to do so. The company presumably thought the problem could not be a serious one because they applied for and received permission to discharge PCBs in the stated amount. However, the large companies that manufactured and used the PCBs had the means to evaluate the impact of PCB discharges to the estuary. They did not do so on the assumption that generic questions of this kind have been dealt with traditionally by government.

Problems similar to those created by PCBs and DDT have been caused by other chlorinated organic compounds. These include kepone, and mirex. Most of the offending organic chemicals have been chlorinated hydrocarbons that are both toxic and persistent in the environment.

The episodes of pollution by chlorinated hydrocarbons served to emphasize the need for a National Toxic Substances Control Act that would prevent potentially catastrophic pollution with chemical wastes. A law finally was signed by President Ford in 1976. It requires that before they can be approved for use, all new chemicals must be tested for their human toxicity and ecological effects. This is a law that was long overdue and, if wisely administered, can greatly reduce, if not eliminate entirely, the danger that a new chemical will

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damage human health or wildlife. However, if it is administered too zealously, the world may be denied the use of new chemicals that could be of great benefit to mankind. Great administrative wisdom will be needed to provide the kind of balance that will give the protection needed, while at the same time assuring that the incentives to produce beneficial new chemicals will not be destroyed.

NOTES

1. Carson, Rachel. "Silent Spring," Houghton Mifflin Co., Boston, Mass. (1962).
2. Kilgore, W. W. and M. Li. "The Carcinogenicity of Pesticides," in: "Residue Reviews: Residues of Pesticides and Other Contaminants in the Total Environment" (F. A. Gunther, ed.), Springer-Verlag, New York/Heidelberg/Berlin (1973). Vol. 48, p. 141.
3. U.S. Environmental Protection Agency. "Report of the DDT Advisory Committee," Washington, D.C. (1971).
4. National Academy of Sciences. "Pest Control: An Assessment of Present and Alternative Technologies," Vol. V: "Pest Control and Public Health," National Research Council, Environmental Studies Board, Washington, D.C. (1976).
5. Deichmann, W.B. "Liver Cancer Deaths in the Continental USA from 1930 to 1972," *Am. Indus. Hyg. Assn. J.* (September 1976), p. 495–498.
6. Hayes, W. L. "Mortality in 1969 from Pesticides, Including Aerosols," *Arch. Env. Health* (March–April, 1976), pp. 61–72.
7. World Health Organization. "Health Hazards of the Human Environment," B. Carcinogens, Geneva (1972), pp. 219–228.
8. Bevenue, A. "The 'Bioconcentration' Aspects of DDT in the Environment," in: "Residue Reviews: Residues of Pesticides and Other Contaminants in the Total Environment" (F. A. Gunther, ed.), Springer-Verlag, New York/Heidelberg/Berlin (1976), Vol. 61, p. 37.
9. Woodwell, G. M. "Toxic Substances and Ecological Cycles," *Scientific American* 216(3):24–31 (1967).
10. Ware, G. W. "Effects of DDT on Reproduction in Higher Animals," in: "Residue Reviews: Residues of Pesticides and Other Contaminants in the Total Environment" (F. A. Gunther, ed.), Springer-Verlag, New York/Heidelberg/Berlin (1975), Vol. 59, p. 119.
11. Cooke, A. S. "Shell Thinning in Avian Eggs by Environmental Pollutants," *Environmental Pollution* 4:85–152 (1973).
12. "U.S. Department of Health, Education and Welfare Report of the Secretary's Commission on Pesticides" (December, 1969), p. 50.
13. Robinson, J. "Organochlorine Insecticides and Bird Populations in Britain," in: "Chemical Fallout: Current Research on Persistent Pesticides" (M. Miller and G. Berg, eds.), Charles C. Thomas, Springfield, Ill. (1969), p. 113.

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14. Devlin, R. M. "DDT: A Renaissance?" *Environmental Science and Technology* 8:322–325 (1974).
15. Wurster, Charles F. "DDT Reduces Photosynthesis by Marine Phytoplankton," *Science* 159:1474 (1968).
16. Wagner, R. H. "Environment and Man," W. W. Norton, New York (1971).
17. Commoner, Barry. "The Closing Circle," Knopf, New York (1971).
18. Massachusetts Institute of Technology. "Man's Impact on the Global Environment: Report of the Study of Critical Environmental Problems" (SCEP) (1970).
19. "DDT Residues in Marine Phytoplankton," in: "Residue Reviews: Residues of Pesticides and Other Contaminants in the Total Environment" (F. A. Gunther, ed.), Springer-Verlag, New York/Heidelberg/Berlin (1972), Vol. 44, p. 23.
20. National Academy of Sciences. "Pest Control: An Assessment of Present and Alternative Technologies." Vol. I. "Contemporary Pest Control Practices and Prospects: The Report of the Executive Committee." National Research Council, Environmental Studies Board, Washington, D.C. (1975).
21. Wurster, Charles F. "DDT Goes to Trial in Madison," *BioScience* 19:809–813 (1969).
22. Wurster, Charles F. "American Scientists Take Legal Action to Improve Pesticide Regulation," presented at the International Conference on The Responsibility of Science in Modern Society, Florence, Italy (October 4, 1976).
23. Federal Register. "Environmental Protection Agency. Consolidated DDT Hearings," *Federal Register* 37 (131):13369–13375 (July 7, 1972).
24. Council on Environmental Quality. "Sixth Annual Report," U.S. Government Printing Office, Washington, D.C. (1975), p. 368.
25. Johnston, D. W. "Decline of DDT Residues in Migratory Songbirds," *Science* 186:841–842 (1974).
26. Nickerson, P. R. and K. R. Barbehenn. "DDT Residues in Starlings, 1974," *Pest. Monit. J.* 9(1):1 (1975).
27. Anderson, D. W., J. R. Jehl, R. W. Risebrough, L. A. Woods, L. R. Dewees and W. G. Edgecomb. "Brown Pelicans: Improved Reproduction off the Southern California Coast," *Science* 190:806–808 (1975).
28. Woodwell, G. M., P. P. Craig and H. A. Johnson. "DDT in the Biosphere: Where Does It Go?" *Science* 1974:1101–1107 (1971).
29. Ware, G. W., B. J. Estesen and W. P. Cahill. "DDT Moratorium in Arizona—Agricultural Residues after 4 Years," *Pest. Monit. J.* 8(2):98 (1974).
30. Higuchi, Kentaro (ed.). "PCB Poisoning and Pollution," Kodansha Ltd., Tokyo and Academic Press, New York (1976).
31. Kuratsune, Masanori. "Epidemiologic Studies on Yusho," in: "PCB Poisoning and Pollution" (K. Higuchi, ed.), Kodansha Ltd., Tokyo and Academic Press, New York (1976).

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32. Nisbet, I. C. T. and A. F. Sarofim. "Rates and Routes of Transport of PCBs in the Environment," *Environmental Health Perspective*, Experimental Issue No. 1 (April, 1972).
33. Panel on Hazardous Trace Substances. Report of Panel on Hazardous Trace Substances to Ad Hoc Committee on Environmental Health Research (David P. Rall, Chairman). *Environ. Res.* 5:249–362 (1972).
34. Peakall, D. B. "Polychlorinated Biphenyls: Occurrence and Biological Effects," in: "Residue Reviews: Residues of Pesticides and Other Contaminants in the Total Environment" (F. A. Gunther, ed.), Springer-Verlag, New York/Heidelberg/Berlin (1972), Vol. 44, p. 1.
35. *Environmental Health Perspectives*, Experimental Issue No. 1 (April, 1972).
36. Environmental Protection Agency. *Proceedings of the National Conference on Polychlorinated Biphenyls* (November 19–21, 1975), Chicago, Ill., *EPA-560/6-75-004*, Environmental Protection Agency, Office of Toxic Substances, Washington, D.C. (March 1976).
37. Kornreich, M., et al. "Environmental Impact of Polychlorinated Biphenyls" *MITRE Technical Report 7006*, The MITRE Corporation, McLean, Va. (May, 1976).
38. Kolbye, A. C. "Food Exposures to Polychlorinated Biphenyls," *Environmental Health Perspectives*, Experimental Issue No. 1 (April, 1972).
39. Jelinek, C. and P. E. Corneliussen. "Levels of PCB's in the U.S. Food Supply," in: *Proceedings of the National Conference on Polychlorinated Biphenyls* (November 19–25, 1975), Chicago, Ill., *EPA-560/6-75-004*, U.S. Environmental Protection Agency, Office of Toxic Substances Washington, D.C. (March, 1976).
40. Grant, D., J. Mes and R. Frank. "PCB Residues in Human Adipose Tissue and Milk," in: *Proceedings of the National Conference on Polychlorinated Biphenyls* (November 19–21, 1975), Chicago, Ill. *EPA-560/6-75-004*, U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C. (1976), p. 144.
41. Kutz, F. W. and S. C. Strassman. "Residues of Polychlorinated Biphenyls in the General Population of the United States," in: *Proceedings of the National Conference on Polychlorinated Biphenyls* (November 19–21, 1975), Chicago, Ill., *EPA-560/6-75-004*, U. S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C. (1976), p. 139.
42. Miller, R. W. "Pollutants in Breast Milk," *Journal of Pediatrics* 90(3):510–512 (1977).
43. N.Y. State Health Planning Commission. "Report of the Ad Hoc Committee on the Health Implications of PCBs in Mothers' Milk," Albany, N.Y. (January, 1977).
44. Allen, J. R. "Response of the Nonhuman Primate to Polychlorinated Biphenyl Exposure," Research Activities at Regional Primate Centers, *Federation Proceedings*, Vol. 34, No. 8, pp. 1675–1679 (1975).
45. McNulty, Wilbur P. *Proceedings of the National Conference on Poly-*

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chlorinated Biphenyls (November 19–21, 1975), Chicago, Ill., EPA-560/6-75-004, U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C. (1976), p. 347.

46. Kuratsune, M., Y. Masuda, and J. Nagayama, "Some of the Recent Findings Concerning Yusho," in: *Proceedings of the National Conference on Polychlorinated Biphenyls* (November 19–25, 1975), Chicago, Ill., *EPA-560/6-75-004*, U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C. (1976).
47. Environmental Protection Agency, *Proceedings of the National Conference on Polychlorinated Biphenyls*, (November 19–25, 1975), Chicago, Ill., *EPA-560/6-75-004*, U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C. (1976).
48. Department of Health, Education and Welfare, Food and Drug Administration, "Polychlorinated Biphenyls (PCB's)," *Federal Register* 42(63):17487–17494 (April 1, 1977).
49. State of New York, Department of Environmental Conservation, Interim opinion and order, File No. 2833 (February 9, 1976).

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