THE PUBLIC HEALTH IMPLICATIONS OF POLYCHLORINATED BIPHENYLS (PCBs) IN THE ENVIRONMENT

Revised and Updated Edition

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Executive Summary

The environmental movement cut its eyeteeth on the controversy over polychlorinated biphenyls, or PCBs, which first came to public attention in the 1960s. PCBs were once widely used in industrial applications, particularly as electrical insulating and heat-exchange fluids. Concern over possible adverse effects on the environment and human health, however, resulted in the prohibition of PCB production in the U.S. and most other industrialized countries.

Even though complaints about PCBs still emanate from some so-called public interest groups, human exposure to these compounds is declining, continuing a trend that began decades ago. The reason this statement can be made with certainty is because environmental levels of PCBs have been measured for potential or possible exposure in a wide variety of systems – various aspects of the environment, food sources, and human and animal body fluids – and in each case, PCB levels have declined.

PCBs in the environment and animals (wildlife) have dramatically and consistently declined over the years. From a public health perspective, trends in human body content of PCBs are most relevant to the assessment of risk and potential for harm to human health. Here, the picture is encouraging and undisputed. Human exposure to PCBs is far lower than it was just 25 years ago. As a result, levels of PCBs in body tissue samples and breast milk have

consistently declined. There is indeed little supportive evidence to suggest that adverse effects are occurring from PCB exposure in today's environment.

An estimated 1.1 billion pounds of PCBs were produced in the U.S. between 1929 and 1977, when the main U.S. manufacturer voluntarily ceased production. Two years later, the U.S. Environmental Protection Agency (EPA) made the ban official, barring all PCB production and strictly regulating any continued use of the compounds.

While low levels of PCBs may remain in the environment for some time to come, their presence is continuing to decrease, primarily as a result of the cessation of production, along with the rise in environmental control measures and regulations that have been enacted over the past two decades. In turn, there is less uptake by lower-food-chain sources of exposure (fish) and lower amounts of PCBs in foodstuffs in general. This decreased exposure has resulted in lower human body burdens of PCBs.

It is ironic that concern in some quarters over PCBs in the environment seems to be growing, despite all the encouraging evidence to the contrary.

PCBs have been associated with health effects in laboratory animals, but typically these occur at very high dose levels relative to realistic human environmental exposures. PCBs have been shown to cause tumors in laboratory animals under conditions of high-dose lifetime exposure, conditions that are not relevant to human exposures. For purposes of regulation, several governmental or advisory agencies have concluded that there is sufficient evidence to consider PCBs to be animal carcinogens.

However, studies of workers who were exposed to high levels of PCBs by inhalation and/or skin contact over long periods of time have not demonstrated an increased risk of cancer. What is more, groups of exposed workers have been followed for years, and these individuals continue to show no statistically significant change in the incidence of cancer or death from other causes. In fact, skin and eye irritations remain the only consistent health effects in workers that can be definitively attributed to PCBs.

There is also no compelling reason to believe that PCBs exert any biologically significant endocrine-modulating (or hormonal) effect in humans exposed to realistic environmental levels. Although research continues today, notably on increasingly subtle effects such as neurobehavioral and neurodevelopmental effects in infants and children, there is no convincing, consistent evidence to support a causal connection between current low-level PCB exposure and clinically significant effects.

General population exposure to PCBs in fish and other foodstuffs has been significantly reduced, and PCB levels in humans are now typically very low. Thus, little benefit to public health can be expected to result from continued investigation into this class of chemicals. Furthermore, excessively stringent and expensive requirements for the removal of trace levels of PCBs from the environment cannot be justified by what is now known about PCBs and their potential effects on human health.

1. Introduction

Polychlorinated biphenyls, or PCBs, became a target of the environmental movement in the United States and elsewhere beginning in the 1960s. The low-level presence of PCBs in the environment, coupled with a perception that these compounds produce adverse health effects, has driven public concern and regulatory policy ever since.

Interest in PCBs first developed in the mid-1960s when researchers in Sweden found trace amounts of the compounds in fish, wildlife, and the environment (Jensen, 1966). Two years later, PCBs were confirmed in U.S. environmental samples. These discoveries soon gave way to concerns about worker safety and public health, eventually prompting regulators to ban PCB production in the U.S. (and other countries) and mandate environmental cleanup efforts.

Despite the torrent of publicity, research, and regulation, however, there is still no convincing, consistent evidence to support a causal connection to clinically significant health effects from exposure to PCBs. In other words, the public health worries about PCB exposure are largely without medical or scientific foundation, meaning that large amounts of scarce financial resources have been diverted over the years into costly PCB-related activities (e.g., cleanup, litigation, and compliance) that could have been put toward many more fruitful and beneficial uses.

2. Brief History of PCBs

PCBs, a group of 209 structurally related chemical compounds (or congeners), were discovered over a century ago, but their production and commercialization did not begin until 1929. They were utilized in the manufacture of a wide variety of products including plastics, adhesives, paints and varnishes, copying paper, newsprint, fluorescent light ballasts, and caulk.

More important, PCBs were found to possess remarkable electrical insulating properties and flame resistance, which led to their widespread use as insulators and coolants in transformers and other electrical equipment. They replaced combustible insulating fluids, thereby reducing the risk of fires in such facilities as office buildings, hospitals, factories, and schools. Some city codes, in fact, required that all capacitors and transformers be of the PCB type. Insurance companies similarly required PCB equipment in many locations. As superb fire-resistant insulators, PCBs surely saved many lives over the decades – lives that might otherwise have been lost in fires associated with less flame-resistant forms of electrical insulation and coolant.

An estimated 1.1 billion pounds of PCBs were produced in the U.S. between 1929 and 1977, when the main U.S. manufacturer, Monsanto Chemical Company, voluntarily ceased production of the compounds. Two years later, the U.S. Environmental Protection Agency (EPA) made the ban official, barring all production of PCBs in the U.S. and strictly regulating any continued use of the compounds.

PCBs nonetheless are still with us. For instance, they have been found, at some level, in roughly one-third of the sites on the Superfund's National Priorities List (NPL) (EPA, 2000). In addition, they often are characterized as ubiquitous environmental contaminants (Atlas and Giam, 1981; Bacon et al., 1992; Ballschmitter, 1991; Wasserman et al., 1979; Wolff et al., 1982), garnering extensive media attention and arousing widespread public concern. Interest in PCBs is not limited to the popular press, however. The number of scientific papers published on PCBs grows year by year, with the total now exceeding 10,000 articles (Erickson, 2001).

The most notorious incidents involving PCBs remain the accidental food poisonings that occurred in Japan in 1968 (Yusho) and in Taiwan in 1979 (Yu-cheng). Both involved eating rice oil that had been contaminated with heat-degraded PCB-containing fluid from leaky food-processing equipment. The two episodes firmly, albeit mistakenly, planted the perception in the public's mind that the PCBs themselves pose serious health risks.

Many of those who had consumed the oil became ill, as did some of the children later born to women who had consumed the contaminated oil during their pregnancies (Rogan et al., 1988; Yu et al., 1991). The symptoms associated with the exposure included various physical (somatic) complaints, low birth weights, chloracne (skin disorders), and excess skin pigmentation (hyperpigmentation), especially of newborns (Kuratsune et al., 1972).

The two poisoning episodes sparked international concern over the potential health risks of PCB exposure. As it turned out, though, the PCBs

themselves were not responsible for the ill health effects that occurred in Japan and Taiwan. The PCB mixtures contained other, more toxic compounds (e.g., polychlorinated dibenzofurans), and this confounded the alleged connection between PCBs and ill health. It is now widely accepted that the furans – and not the PCBs themselves – caused the symptoms.

Human exposure to PCBs occurs primarily by way of low-level food contamination. The compounds also have been found at trace levels in marine plant and animal species, fish, mammals, birds, and humans.

3. Occupational and Environmental Exposure

Prior to the imposition of the federal ban, workers safely manufactured and used PCBs for decades. Limits on occupational exposure were established in the early 1940s after PCBs were reported to cause such acute health effects as chloracne, an acne-like skin disorder. However, it was not until the 1960s, with the beginnings of the environmental movement, that concerns over PCBs extended beyond the workplace.

Early reports from Swedish researchers revealed the presence of PCBs in eagles and herring, as well as in soil and water samples, being screened for the controversial pesticide DDT (Jensen, 1966). In 1968, the presence of PCBs was confirmed in U.S. environmental samples.

Later studies demonstrated that the more highly chlorinated forms of PCBs biodegraded (i.e., broke down naturally) at very slow rates (Jensen, 1972). This finding helped to explain the stubborn persistence of certain types

of PCBs in the environment, a characteristic that elevated concerns about their potential public health effects.

In 1977, when the electrical industry notified Monsanto that suitable replacement fluids were available, Monsanto ceased manufacture of PCBs.

Earlier, in 1976, PCBs became the subject of specific regulations under the Toxic Substances Control Act (TSCA), because of their widespread and persistent presence in the environment and concerns about toxicity. TSCA authorized the EPA to issue rules to regulate the manufacture and use of PCBs in the U.S. In 1978, the EPA issued rules concerning the disposal and labeling of PCBs, and in 1979 it issued rules banning the manufacture of PCBs and placing stringent restrictions on their continued use.

Throughout the 1980s and 1990s, the EPA continued to issue new rules concerning PCBs (e.g., their use in electrical equipment and the cleanup of spills).

4. Results of Laboratory Animal Studies

While PCBs are present at only trace levels in many environmental areas, it has been the reported health effects in laboratory animal studies, often at dose levels far in excess of any human exposure, that have driven much of the public concern. A review of what is known about the effects of PCBs in laboratory animals is therefore relevant.

PCBs' ability to cause tumors in rodents was first reported in mice by Nagasaki et al. (1972) and Ito et al. (1973). These studies were followed by

reports of tumors in rats (Kimbrough et al., 1975; Schaeffer et al., 1984; Norback and Weltman, 1985; NCI, 1978). In many instances, study limitations made the interpretation of the whole body of data difficult and imprecise.

The rat studies nonetheless formed the basis for the EPA's classification of PCBs as probable human carcinogens (EPA, 1988). Besides the EPA, the International Agency for Research on Cancer (IARC, 1987), the National Toxicology Program (NTP, 1989), and the American Conference of Governmental Industrial Hygienists (ACGIH, 1996) have similarly concluded that sufficient evidence exists to classify PCBs as known animal carcinogens.

The EPA's longstanding policy is to consider chemicals that cause cancer in animals to be probable human carcinogens. However, when interpreting results from animal toxicity studies for purposes of assessing human relevance, a number of important factors should be taken into account. These include the major physiological and metabolic differences between the laboratory animals used in testing and humans; variation within the human population, including differences in age, sex, and individual susceptibility; and the very large doses of the agent administered to laboratory animals.

What is more, toxicology tests are designed specifically to elicit and detect adverse effects, and for this reason, high dose levels, unrealistic for humans, are employed. These doses invariably exceed the level of realistic human exposures, often by hundreds or thousands of times. Because of the vast difference in exposures between laboratory animals and humans, the EPA's cancer classification effort cannot reflect the true risk to humans.

While PCBs can cause tumors in laboratory animals, the question is, at what dose level and under what types of exposure does this occur? And do these conditions realistically reflect human environmental exposures? (Laboratory animals typically are exposed daily for their lifetimes.)

In a recent noteworthy action, the EPA lowered the cancer slope factor for PCBs, meaning the agency now considers the carcinogenic potential of PCBs to be lower than previously thought (EPA, 2003a).

The latest scientific evidence further indicates that PCBs can cause a variety of health effects in laboratory animals under defined conditions of exposure, including cancer and effects on the immune system, reproductive system, nervous system, and endocrine system. Whether any of these effects are relevant to humans at environmentally realistic exposure levels is doubtful given what is known about the evidence in humans and the differences in exposure.

5. Exposure-Related Effects in Humans

Human data, when available, are always preferable to laboratory animal data when assessing potential health risks from environmental or occupational exposures. And for PCBs, there is a robust body of human epidemiological literature, primarily from occupational studies.

Some investigators have examined the relationship between PCB exposure and potential human health effects using indirect or surrogate measures of exposure such as fish consumption. For example, there have

been recent reports of neurobehavioral and developmental effects in children (Jacobson and Jacobson, 1996; Korrick, 2001), memory and learning impairment in older adults (Schantz et al., 2001), endocrine-mediated effects including shortened menstrual cycle (Mendola et al., 1997), and other nonspecific reproductive effects. However, some of these studies have serious shortcomings with respect to confirmation and quantification of exposure, a critical facet in the assessment of causality between any suspected or supposed toxic compound and subsequent health effects.

For obvious ethical reasons, the types of studies conducted in laboratory animals are not conducted in humans. For relevant human data, we must therefore rely on (a) studies of workers occupationally exposed to PCBs, (b) studies involving large-scale or widespread accidental exposures, or (c) low-level environmental exposure studies, such as those involving individuals who consume food that contains PCBs.

The studies of PCB-exposed populations collectively suggest that the only consistent adverse health effects attributable to PCBs in humans are skin effects, including chloracne and other effects related to chronic skin and eye irritation (James et al., 1993). These effects occurred only in worker populations with relatively high skin and/or inhalation exposures or in individuals with high accidental exposures. In contrast, there is little documentation that environmental exposures, the type of exposure relevant for the vast majority of people, are associated with such effects.

A. Occupational Studies

Epidemiologists have evaluated the possible association between PCB exposure and effects in humans primarily by focusing on mortality (Kimbrough et al., 2003; Loomis et al., 1997; Sinks et al., 1992; Brown, 1987; Bertazzi et al., 1987; Gustavsson et al., 1986; Shalat et al., 1989; NIOSH, 1977; Davidorf and Knupp, 1979).

Significantly, more than 50 years have passed since workers were first heavily exposed to PCBs. This length of time should be sufficient to detect diseases, such as cancer, that are thought to have latency periods of 10 to 30 years (i.e., the period of time between initial exposure and development of the disease).

The most extensive occupational studies of long-term exposure to PCBs have focused on workers employed in the manufacture of electrical equipment. These individuals had daily skin contact with PCBs for many years, inhaled relatively high levels of the chemicals, and may have ingested some while eating near their work stations or smoking. Aside from skin and eye irritation, and perhaps transient elevations in some liver enzymes, no other acute or chronic health effects can be definitively attributed to PCBs from occupational studies. Despite high serum and fat concentrations of PCBs in these workers, the severe form of chloracne and other effects seen in the Yusho victims have not occurred (Rosenman, 1992).

Among the occupational health studies, several are particularly noteworthy because they found no adverse health effects associated with PCB exposure:

- The National Institute for Occupational Safety and Health (NIOSH) studied maintenance workers exposed to PCBs at two utility companies (Smith et al., 1981a). The workers did not exhibit any ill-health effects, even though the PCB concentrations found in their blood were well above the national average at the time.
- NIOSH also studied workers exposed to PCBs at an electrical equipment manufacturing plant and found some high PCB blood concentrations. However, no adverse effects were reported (Smith et al., 1981b).
- Two other studies (Lawton, et al., 1985) surveyed the health of workers heavily exposed to PCBs for an average of 15 years – and some for as long as 35 years. Once again, investigators found no evidence of PCB-related health effects among these workers.

Another study of workers exposed to PCBs looked specifically at rates of death (mortality). In that study, Sinks et al. (1992) compared the mortality of 3,188 electrical manufacturing workers with known exposures to PCBs (between 1957 and 1986) with appropriately matched controls in the U.S. They found a lower-than-expected number of deaths from all causes (192 deaths observed, 283 expected) and from all types of cancer (54 deaths observed, 64 expected). In yet another study, Loomis et al. (1997) examined deaths among

138,905 men employed at five electrical power companies for at least six months (between 1950 and 1986). The authors found no relationship between cumulative exposure to insulating fluids containing PCBs and deaths from all causes or specifically from cancer.

Between 1946 and 1977, the General Electric Company manufactured capacitors containing PCBs in two production plants in upstate New York. The workers in these two plants most likely represented the largest capacitor worker cohort in the world. Kimbrough et al. (1999) have followed this cohort, a group comprising more than 7,000 workers, to evaluate the six types of cancer in these workers that had been reported in the literature as being associated with PCB exposure: malignant melanoma, liver, rectal, gastrointestinal, brain, and hematopoietic (cancer of the blood-forming tissue) cancer.

In 1999, these investigators studied workers exposed to PCBs for at least 90 days (between 1946 and 1977) and found that deaths from all causes in male hourly workers were significantly below the expected number. No significant elevations were seen in the most highly exposed workers, and none of the previously reported specific excesses in cancer mortality were seen.

In a five-year update to this study (Kimbrough et al., 2003), the authors assessed a very large group of PCB-exposed electrical manufacturing workers. The authors report that they again failed to find any significant excess mortality from any type of cancer in the total group studied or specifically in workers with the highest PCB exposure. The study supports other occupational studies that

fail to demonstrate any link between PCB exposure and excess deaths from cancer or any other cause.

Collectively, these occupational studies have found no direct or discernible correlation between PCB exposure level and elevated risk of death from cancer. Given this, it is even more unlikely that a measurable or even plausible risk of cancer from environmental exposures to PCBs could be detected, a perspective recently acknowledged by the National Cancer Institute (Whelan and Lehr, 2001).

B. Accidental Environmental Exposures and Contamination

The serious health problems (acute and chronic) seen in victims of the accidental poisoning incidents in Japan and Taiwan have not been observed in people with occupational PCB exposure (Rosenman, 1992). This finding suggests that other chemicals were involved in the Japanese and Taiwanese incidents and/or that exposures were far higher than those typically encountered among people who are occupationally exposed. It is thus important to review in some detail those few cases involving high environmental exposure in order to learn about potential effects related to PCB exposure.

In 1968, some 1,300 people in Fukuoka, Nagasaki, and other areas of western Japan became ill after consuming rice bran oil contaminated with 2,000-3,000 parts per million (ppm) of a heat-degraded Japanese brand of PCB heat-transfer fluid (Kunita et al., 1984; Kuratsune, 1989). The victims

developed a very severe and persistent form of acne called chloracne. The disease symptoms soon progressed to include fatigue, nausea, and swelling of the arms and legs; some people developed liver disorders. Some newborns of exposed mothers were small in size, had discoloration of skin and nails, and showed premature eruption of teeth (Kuratsune et al., 1972).

A similar poisoning episode occurred in Taiwan in 1978 and was reported as “Yu-Cheng” disease. Once again, rice bran oil was contaminated by heat-degraded PCBs and subsequently consumed by more than 2,000 people. The Yu-Cheng victims displayed symptoms similar to those in the Yusho victims. Offspring of Yu-Cheng mothers exhibited symptoms of acute toxicity (Rogan, 1989; Rogan et al., 1988), as well as growth abnormalities, neurological effects, and a host of other non-specific findings (Guo et al., 1995). Elevated mortality from liver diseases other than cancer was observed within three years after the poisoning outbreak (Hsu et al., 1985; Hsieh et al., 1996).

The Yusho and Yu-Cheng incidents were originally attributed to PCBs, but later chemical analyses showed that the heat transfer fluid contained elevated levels of degradation products of PCBs (these included chemicals known as polychlorinated dibenzofurans – furans – and polychlorinated quaterphenyls). Comparisons of PCB body burdens of Yusho victims with those of Japanese electrical workers showed similar PCB levels, but the electrical workers had none of the symptoms of Yusho disease. However, the Yusho victims had much higher levels of furans in their bodies than did the

electrical workers. Similar studies of Yu-Cheng victims and workers showed the same pattern.

It is now largely accepted that the illnesses seen in Yusho and Yu-Cheng victims were caused by the thermal degradation byproducts, primarily the furans, and not PCBs. It should be further noted that PCBs heated during use in capacitors are not known to break down to the same types of degradation products as found in the Yusho and Yu-Cheng incidents. Therefore, the reports of health effects observed in the Japanese and Taiwanese incidents are not relevant to discussions of the potential health effects of PCBs in humans resulting from realistic environmental exposures.

C. Low-Level Environmental Exposure Studies

Even though environmental exposures are much below the levels to which persons were exposed occupationally, environmental incidents cause the most concern among the public, the most pressing attention from interest groups, and the highest level of attention from state and federal regulatory agencies. Over the years, numerous studies involving environmental exposure have been reported. However, the amount of human exposure has rarely been quantified or even estimated. This lack of adequate and accurate exposure monitoring is a serious problem when assessing the possible human health effects of PCBs.

In recent years, a number of studies have reported that prenatal exposure to PCBs among consumers of sport fish and certain other groups is

associated with subtle neurodevelopmental effects in newborns and children. Reports have appeared in the literature from studies in Michigan, North Carolina, and New York, as well as from the Netherlands, Germany, Canada, and elsewhere. While there have been similar findings in some of the studies, in general the results are inconsistent and the studies suffer from numerous, serious methodological deficiencies.

Many of the studies reported cord blood levels, maternal blood levels, children's blood levels, or maternal milk levels of PCBs, while others have used fish consumption as a substitute for PCB exposure. Other studies examined infants born to women with no known exposure other than to background levels of PCBs. In all studies, the mothers showed no signs of PCB toxicity. Lower birth weight has been reported in some of the infants born to mothers who consumed fish that contained PCBs, but the degree of weight reduction was not correlated with maternal exposure levels (Fein et al., 1984). In addition, there have been reports of various neurodevelopmental deficits among the more highly prenatally exposed infants from the various studies (Rogan et al., 1986; Gladen and Rogan, 1991; Gladen et al., 1988). A follow-up report indicated that some early deficits were no longer apparent at ages three, four, or five years (Gladen and Rogan, 1991).

Kimbrough et al. (2001) reviewed those studies that have looked at the possible relationship between PCBs and related chemical exposure in children and various neurobehavioral tests and concluded that "in the

aggregate, the studies reviewed here show no conclusive evidence that environmental exposure to these chemicals affects the neurobehavioral development of infants and children." The authors reported that statistically significant differences in some studies appear to be spurious and may have occurred by chance or been influenced by other factors. Because of declining body burdens of PCBs in the general population, which in turn make it difficult to achieve accurate exposure assessments, Kimbrough et al. (2001) suggest that additional studies will not yield more definitive results.

The absence of documented and confirmed effects in humans, despite evidence of effects in laboratory animals exposed to PCBs, should be reassuring. These findings are consistent with the major scientific reviews of the PCB literature and recent updates on important occupational cohorts. The divergence in the findings between animals and humans may result from several factors, some of which include differences in species sensitivity or susceptibility (e.g., Rhesus monkeys are much more sensitive to PCBs than humans or other animals), the very low environmental levels to which humans are exposed, or the high concentrations used in laboratory studies, far greater than even the highest occupational exposure levels.

Confidence in the absence of identifiable PCB-related disease and mortality in humans as a result of general environmental exposure is strengthened by the fact that the human evidence is based on studies of individuals with chronic, high exposure to PCBs in industries where PCB use was common for approximately 50 years. The occupational cohorts that have

been followed for years continue to demonstrate no significant change in either cancer incidence rate or other effects in humans (Swanson et al., 1995; Kimbrough et al., 2003; Sinks et al., 1992; Loomis et al., 1997).

6. Do PCBs "Disrupt" Endocrine Systems?

Significant public and regulatory attention is now focused on endocrine, or hormone, disruption as a measurable indicator of toxicity. Some scientists have hypothesized that hormonally-related health problems occur because trace environmental chemicals might have disruptive effects on the endocrine system. This speculation was widely publicized in 1996 with the release of *Our Stolen Future* (Colburn et al., 1996). The authors suggest that some chemicals, acting as hormonal disrupters, may lead to infertility, some cancers (e.g., cancers involving the breast, prostate, testes, ovaries, and uterus), and other hormone-related disorders.

Estrogens are produced both in males and, at much higher levels, in females. Estrogens are also found naturally in plants (phytoestrogens) and are consequently consumed in the diet.

There are concerns over a variety of environmental chemicals for their ability to cause endocrine changes to various levels of living organisms, but there has been no confirmed scientific evidence linking environmental exposures to such effects in humans (Juberg, 2000). Similarly, the evidence for effects of environmental estrogens on wildlife remains weak and circumstantial. Some have suggested, for example, that chemicals in the

environment acting as endocrine disrupters may be responsible for the production of female animals from eggs containing male embryos. However, it has been shown that the same sex change can occur in turtles when eggs containing male embryos are incubated at two degrees Celsius higher than normal (Crews et al., 1994). Thus, it is clear that some effects have been prematurely attributed to “endocrine disruption” based on incomplete evidence.

Given the notoriety that accompanies PCBs and given their persistence in the environment, it was not unexpected that they would be among those chemicals labeled “endocrine disrupters.” PCBs and some of the metabolites of PCBs do have weak estrogenic activity in laboratory tests, but their efficacy or potency as an estrogen is extremely small compared to the body’s natural estrogen (Safe, 1995). Most scientists today do not subscribe to the hypothesis that environmental estrogens cause increased rates of breast cancer or male reproductive problems, as scientific evidence of such is simply lacking and the biological plausibility for such is remote given the very low environmental levels.

Several studies have reported higher levels of organochlorines (PCBs and other chlorinated compounds) in fat tissue or blood serum of breast cancer patients, as compared to women without breast cancer. However, the overwhelming weight of the evidence on organochlorines suggests that there is no correlation between these chemicals and breast cancer. There have been dozens of studies examining the possible association between breast cancer (as cited in Kimbrough et al., 2003) and PCB exposure. Breast cancer and PCB

body burdens have been examined in at least 24 study groups comprising over 5,000 breast cancer cases, and the collective studies provide little support for the premise that body burdens of PCBs are associated with elevated risk for breast cancer.

Other related investigations on potential endocrine effects of PCB exposure have not demonstrated associations between PCB levels and miscarriage, stillbirth, or infant death (Kreiss et al., 1981). Great Lakes fish consumption has been examined in relation to spontaneous fetal death (Mendola et al., 1995), conception delay (Buck et al., 1997; Buck et al., 1999), and menstrual cycle length (Mendola et al., 1997). In these studies the only association detected was shorter cycle length (a mere one-half day delay), and this “finding” was described as an association, possibly influenced by confounding variables, not a causal relationship. Two other studies did not find associations between PCB serum levels and risk of endometrial cancer (Weiderpass et al., 2000; Sturgeon et al., 1998).

In summary, the weight of the scientific evidence, combined with consideration of the low levels of environmental exposure to PCBs and their very weak endocrine activity, does not provide compelling reason to believe that PCBs exert any biologically significant endocrine-modulating effect in humans exposed to realistic environmental levels.

7. PCBs in the Environment and Human Exposure

A significant part of the risk equation that is often overlooked when regulators, public health officials, and various public interest groups raise concern about continuing health risks from environmental PCB exposure is the element of human exposure.

In order for a risk to be present, there must be sufficient exposure to a biological or chemical agent. At a time when concern over PCBs continues to increase among some groups, the reality is that human exposure continues to decline, a trend that has been occurring for the past several decades. Environmental levels of PCBs have been measured in water, air, soil, food sources, and humans themselves – and in each case, PCB levels have been declining. Since PCB production and use has ended, air, water, and soil levels have naturally dropped. In turn, there is less uptake by lower food chain sources of exposure such as fish, and lower amounts in foodstuffs in general. Finally, these decreases have resulted in lower human body burdens of PCBs. It is ironic that in the face of all of this encouraging information, there is continued, often unwarranted, concern over PCBs in the environment.

8. PCB Concentrations in Environmental Compartments

To get a sense of what has occurred with respect to human exposure to PCBs over the years, it is helpful to look upstream, so to speak, at environmental levels and how those have changed. Because of the nature of PCBs, the major sources of environmental exposure are soils and water-

deposited sediments. Following the U.S. ban on PCB production over 25 years ago, PCBs levels in the environment have declined, particularly where their removal from transformers and capacitors has occurred on a large-scale basis, minimizing the risk of leaks or accidents. (PCB-containing equipment, it should be noted, is still in use in certain parts of the world.)

Over the past decade, the health of the Great Lakes ecosystem has been carefully studied. Most environmental indicators point to a decline in chemicals in the environment (SOLEC, 1995; SOLEC, 1997). For PCBs, much of the improvement came from decreased input to the environment. A major pathway by which some compounds can influence the Great Lakes is through rain and snow deposition, and the Integrated Atmospheric Deposition Network (IADN) has monitored precipitation and air concentrations of PCBs over the past decade. Levels of PCBs in air and precipitation have been decreasing steadily (IADN, 2003). Further, other researchers (Simcik et al., 1999) report that atmospheric concentrations of PCBs around the Great Lakes, specifically in such urban areas as Chicago, have decreased. Recent EPA monitoring and assessment of trends in sediment contamination throughout the U.S. also show declining concentrations of various chemicals in many regions (EPA, 2003b; Kuntz et al., 1999), including levels of PCBs in sediments (IJC, 1996; SOLEC, 1997). An IJC workshop in 1995 assessed contaminant trends in air, water, and soil, and for all three environmental compartments, chemical contaminant trends were clearly shown to be declining (IJC, 1996).

While low levels of PCBs will remain in the environment for years to come, their presence is continuing to decrease, largely as result of the cessation of production, along with the rise in environmental control measures and regulations that have been enacted over the past two decades.

9. PCBs in Fish and Other Foods

As fish eat other fish or bottom-dwelling organisms, they take on the body burden of PCBs present in their prey. Fish are able to metabolize (break down) some PCBs; those that are not metabolized or excreted accumulate in the fish's fatty tissues. The result is bioaccumulation of PCBs. Thus humans who consume certain fish may also accumulate PCBs. For these reasons, fish are monitored for PCB concentration in some waterways. Numerous states and federal regulatory bodies have established fish consumption guidelines, particularly for those fish known to accumulate a variety of chemicals, including mercury, PCBs, and other persistent contaminants.

The decline in environmental concentrations of PCB has resulted in a concomitant reduction in PCB concentration in fish throughout the country (Fenstershiem, 1993). Much attention has been focused on aquatic environments associated with the Hudson River because of the substantial release of PCBs over a 30-year period. The majority of fish samples that have been analyzed have shown detectable levels of PCBs, but researchers and the New York State Department of Environmental Conservation (NYSDEC) have

concluded that PCB concentrations in fish have been declining in the Hudson River since the 1970s (Fensterheim, 1993).

Studies conducted since 1980 have shown that on a national level PCB levels in freshwater fish have decreased significantly. In a 1986-89 EPA national assessment, fish from 97% of sites contained 10 ppm PCB or less, while one study showed fish from 74% of sites contained 1 ppm or less (Keuhl et al., 1994). Fish near some heavily contaminated industrial sites, however, contain much higher levels of PCBs. While data for shellfish are not as extensive as for fish in the U.S., there are ample data from a variety of locations including Boston Harbor, Florida, California, Washington, Delaware, North Carolina, and others, which all show declining PCB concentrations in mussels, oysters, and other bivalves (Fensterheim, 1993).

PCBs in foods are regulated by the U.S. Food and Drug Administration (FDA), which has established tolerances for PCBs in various food items ranging from commercial fish to infant/junior foods to paper and food-packaging materials intended for use with human food. According to FDA studies, the amount of PCBs consumed in the diet has decreased steadily (from almost 7 mg/day in 1971 to less than 0.1 mg/day in 1988 [Shank, 1991]). Even 10 years ago, the amount of PCBs in the human diet was less than 1% of the levels detected in the early 1970s, as measured by the Total Diet Study of the FDA.

It is evident that along with the declines in environmental concentrations of PCBs, a similar decline of PCB burden in those living organisms closest to

environmental sources, such as shellfish and fish, has also occurred. The most important aspect of this downward trend is what it has meant for human body burdens of PCBs over the years.

10. Decline in the Human Body Burden

PCBs in the environment and biota have dramatically and consistently declined over the years. From a public health perspective, trends in human body burden of PCBs are the most relevant data for assessment of human risk and potential for harm. Here, the picture is encouraging and not disputed.

The National Human Adipose Tissue Survey (NHATS), part of the National Human Monitoring Program (NHMP) established in 1967 by the U.S. Public Health Service, has shown several important trends in recent years. All of the samples analyzed have had PCB residues, but the trend in the percentage of the population with PCB levels in fatty tissue greater than 1 ppm has been dropping. The NHATS analysis in various subpopulations found no significant differences in trends between race groups, sex, or geographical region. Human blood sera have also shown similar declining trends in PCB concentration over the years, again reaffirming the fact that PCB exposure today is far lower than it was 25 years ago (Longnecker et al., 2003).

Recent studies and assessments of health in the Great Lakes and other regions continue to demonstrate the declining concentrations of PCBs in humans. Health Canada published a review (Health Canada, 1997) of

environmental contaminants and human health in the Great Lakes Basin. Over the past 25 years, levels of PCBs in breast milk have consistently declined.

A recently released report that has garnered the attention of many, including the media and advocacy/public interest groups, is the CDC's 2nd National Report on Human Exposure to Environmental Chemicals (CDC, 2003). Although this report is not a health risk assessment, it does provide an overview of human body burdens of 116 chemicals. While many PCB congeners were evaluated, the most frequently detected ones in the general population were PCBs 105, 118, and 156. Of these, PCB 118 was the most commonly detected (47%), and yet since 1988 the concentration in humans has declined approximately six-fold.

Levels of PCBs in the environment, fish and other food, and humans are significantly lower than they were years ago and continue to decline today. Any potential risk of health effect is less today than 25 years ago, a time when no study to date had demonstrated a causal link between PCB environmental exposure and adverse health effects in humans. In sum, there is no supportive evidence that adverse effects are occurring as a result of PCB exposure in today's environment (Longnecker, 2001).

11. Federal PCB-Related Regulations

Addressing the matter of PCBs in the environment over the past 25 years has entailed significant direct costs to business and indirect costs to the

public, borne through pass-along fees and the like, as well opportunity costs, including other public health intervention measures that had to be forgone.

Consider some of the federal regulations that deal with PCBs. Under the Toxic Substances Control Act (TSCA) of 1976, PCBs became the only chemical family fully regulated by the federal government. Any release of PCBs into the environment must be reported, and specific remedial actions are delineated, depending on the concentration of PCBs involved.

EPA regulations required that capacitors in areas where public exposure might occur (such as utility poles in backyards) be removed from service by 1988 (Fed. Reg., 1982a, b, 1985). Use of some transformers in protected environments, such as fenced utility grounds, is acceptable until the end of their useful life under the EPA PCB regulations. Under TSCA, owners and operators of these facilities are required to report regularly on safety, use, and disposal. EPA further restricted the use of PCB-containing transformers to deal with the possible hazards from fires in electrical equipment. After 1990, most regulations have focused on PCB waste tracking and disposal (EPA, 1991b, 1992a, b).

12. What Is Being Done to Clean Up PCBs?

The PCB environmental cleanup, combined with the ban on production and strictly regulated use, ensures that exposure to PCBs in the U.S. and overall PCB contamination will continue to decline in coming years. Throughout the country, the EPA oversees Superfund cleanup sites containing significant

levels of PCBs. Site decontamination includes soil excavation (and even soil incineration), with disposal in EPA-approved landfills. Complete remediation is sometimes not possible because of various factors, such as the lack of suitable technology (a particular problem for sediments) and local opposition to landfills or incinerators. Before actual remediation can occur, remedial investigation and feasibility studies (including cost analysis) must be performed.

Probably the single largest and most publicized example of environmental PCB remediation involved the dredging of sediments from a 40-mile stretch of the Hudson River in upstate New York. From 1947 to 1977, more than 500,000 pounds of PCBs were discharged, under permit, into the Hudson River from two General Electric (GE) capacitor-manufacturing plants at Fort Edward and Hudson Falls, New York. An initial study identified 40 Hudson River “hot spots” (defined as sediment containing 50 ppm or more PCBs).

The New York State Department of Environmental Conservation, along with GE, have together spent tens of millions of dollars and well over 10 years evaluating, debating, assessing and coming to some agreement over what needs to be done to “protect people and the environment from unacceptable risks due to PCB-contaminated sediments in the Upper Hudson River.” On February 1, 2002, the EPA issued a Record of Decision for the Hudson River Site that specifies that 2.65 million cubic yards of sediment be dredged from a 40-mile stretch of the river at a total estimated cost of about $500 million. However, along the way the project has been tainted with ill will and mistrust

among the EPA, GE, and homeowners along the Hudson. Lawsuits and debate over whether there is net harm in raising PCBs from contaminated sediments continue to this day.

Independent evaluators of the progress of this project have had the opportunity to solicit the views, concerns, and opinions of residents living along the Hudson, and many, while expressing concerns about the project, have also expressed distrust about the actions of local and federal government agencies that are in charge of the project. Clearly, this type of cleanup is not without controversy, and today there is little assurance that whatever dredging is done will result in a measurable net positive impact upon public health.

In Lake Michigan's Waukegan Harbor, where PCBs were discharged from several sources, more than one million pounds of contaminated sediment have been dredged from the harbor. This PCB-related remediation is now complete, and the site is operating under long-term operation and maintenance requirements. While the PCB remediation is done, there are residual PCB concentrations on-site, although these are below the required cleanup levels determined in the record of decision (EPA, 1999). The Waukegan Harbor story, however, is not complete. During PCB remediation, creosote contaminated soil was discovered, and today there are numerous additional contaminants under investigation, including arsenic, cyanide, benzene, and various PAHs, all of which have potentially far greater impact on human health than do PCBs.

It has been suggested that leaving contaminated rivers and waterbeds alone would allow for natural restoration and remediation of waters contaminated with PCBs. Bacterial degradation or biodegradation of PCBs in soil and sediment is a potentially useful technology that is receiving attention (Shannon, 1996; Brenner et al., 1994; Furukawa, 1994; Abramowicz, 1994; Anon., 1997). In fact, bacterial dechlorination of PCBs in buried sediments in the upper Hudson River has occurred (Brown et al., 1987), and currently there is increased interest and hopeful speculation that soon, microbial dechlorination of PCBs in aquatic sediments will present a viable additional alternative to physical dredging of sediments.

The sheer volume of sediments in this country that have “some” degree of PCB contamination simply will not allow manual dredging and removal, and alternative solutions must be developed if concerns over PCBs persist. It has been argued that natural remediation would be far cheaper and less disruptive to the ecosystem than remedial measures such as dredging, and there is growing support for the view that microbial PCB dechlorination, perhaps combined with aerobic degradation, holds much promise for the detoxification and remediation of PCBs in aquatic sediments (Bedard, 2001). Certainly, the dredging and subsequent transport and disposal or incineration of PCB-containing sediments involves cost, energy, and possible contamination of other media (e.g., roadways, railways, etc.), considerations that need to be weighed when deciding upon a remediation plan.

PCB remediation discussions frequently come down to the same scenario, one in which contaminated environmental compartments, typically soil or sediments, are the focus of remedial efforts, yet all too often there is little assessment of the actual, not perceived, health risks from these sources. For too long, the public has been presented with the perspective that PCBs are toxic environmental chemicals at low-dose levels, yet to date there is no known causal relationship between environmental PCB exposure and adverse impacts on public health.

13. What Is All This Costing?

The cost of replacing, monitoring, and disposing of PCBs has been substantial. (Early estimates provided in the 1980s totaled almost $970 million. [Fed. Reg., 1982a, 1985].) Remediation and cleanup costs alone are by now well into the billions of dollars, even though the net benefit to public health, if any exists, has never been demonstrated.

There have been other costs associated with replacement of PCB-containing power transformers and capacitors. For instance, those companies required to replace PCB-containing power capacitors in their systems invariably pass on fees to the customer. The cost to private industry of inspecting its equipment and replacing capacitors and transformers contributes to higher prices for goods. However, equipment replacement and maintenance costs pale in comparison to cleanup costs for PCB spills and PCB-containing waste sites. Since the 1990s, the majority of available funds

has gone toward the remediation of contaminated PCB sites such as parts of the Hudson River, New Bedford, MA, and Lake Michigan, among others. A decade ago, the EPA estimated the total quantity of PCB waste remaining to be remediated at 525 million tons (Agarwal, 1994). Assuming an average cost of $250 per ton for landfill and $500 per ton for incineration, the bill for disposing of all this PCB-laced waste would come to hundreds of billions of dollars. Many factors influence the cost of remediation and removal projects, including the location of PCBs (in water, soil, sediment, bedrock, etc.); aerobic (exposed to air) versus anaerobic conditions; specific PCBs present (highly chlorinated PCBs are generally more difficult to degrade than less-chlorinated PCBs); and specific methods of treatment, including developments in bioremediation (bacterial degradation) and other novel remediation techniques. However, the astronomical cost associated with remediating all PCB-contaminated sediment and soil (regardless of how low the contamination levels may be) is one that may well not be justified given current public health priorities.

PCBs, in a sense, have become the poster child for all environmental contaminants. Removal and eradication are the be all and end all, independent of any objective and science-based assessment of human exposure, health risk, or public health benefit. There are, of course, precedents – notably, asbestos and lead – where the philosophy of total removal sometimes backfired, with full-blown remediation resulting in increased blood lead levels or exposure of humans to airborne asbestos fibers. For PCBs, there is merit to weighing all options and their public health implications before deciding on a

path of action. There has already been too much emotion and too little objective analysis, although a weight-of-evidence evaluation suggests that removal of PCBs from the environment makes little sense.

14. Conclusion

Although PCBs can be found at trace levels in the environment, their levels in fish, other foods, and human tissues are on the decline, continuing a trend that has been going on for many years. No conclusive evidence exists to support the position that background levels in the general population, or even the much higher levels that occurred among some occupational groups, have resulted in carcinogenic or other chronic adverse health effects. In humans, the only effects correlated with chronic or high-level PCB exposure are skin and eye irritation. These effects, however, have not been observed in populations exposed to PCBs through fish consumption or from other environmental sources.

The U.S. ban on PCB production, along with replacement of PCB-containing equipment, has resulted in declining concentrations of PCBs in the environment and this, in turn, has significantly lowered human body burdens of PCBs. Based on available scientific data and evidence, PCB exposures from environmental sources today do not pose significant health risks.

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