Environmental Poisoning of Children — Lessons from the Past

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Children have physiologic and behavioral characteristics that make them vulnerable to damage from environmental chemicals. In the past, there have been episodes in which children became ill or died from environmental exposures that spared adults or affected them less severely. Among the characteristics leading to children’s sensitivity are their limited diets, dividing cells, differentiating organs and organ systems, slow or absent detoxification mechanisms, long life expectancy with the resulting ability to express damage with delayed consequences, and the severe metabolic demands of growth. There have been large outbreaks of poisonings involving children in Asia and Turkey, and some of the less obvious effects of chemicals have appeared in children in the United States. Although the United States has been spared a widespread outbreak of severe poisoning, such an incident is possible and would likely have greater consequences for children than adults. — Environ Health Perspect 103(Suppl 6):19–23 (1995)

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Introduction

This article is a compilation of incidents in which children have been affected by exposure to environmental chemicals. The results range from subtle developmental delay to death. They illustrate the ways in which differences between children and adults can make exposures that are safe or produce minimal toxicity in adults lead to detectable damage in children.

PCBs

In 1968, an epidemic of severe acne among residents of Kyushu province in Japan was traced to use of cooking oil that had been contaminated by PCBs during processing (1). Over 1000 people were eventually diagnosed as having “Yusho” (oil disease) by the local health authorities. Yusho was essentially chloracne, familiar to occupational physicians as “cable handler’s itch,” from the linear pruritic bands it produced at points of contact with insulated or impregnated cable. Chloracne was not specific to PCBs; in fact, it was produced by chlorinated naphthalenes, furans, or dioxins, all of which shared the substituted double-ring structure. Chloracne is similar to acne vulgaris except that cysts tend to be more severe and inflammation relatively less prominent. Healing is quite prolonged, and Yusho cases remained active for several years after exposure ceased, with, ultimately, substantial scarring among those that did resolve.

There are reports of 13 women who were pregnant at the time of exposure. One of the children was stillborn and was deeply and diffusely pigmented (2). Of the remaining children, 4 were small for dates, 8 had hyperbilirubinemia, 4 had pigmented gums, 9 had conjunctivitis with dilatation of the sebaceous glands of the eyelid, and 10 had diffuse skin pigmentation. PCBs appeared in the breast milk of cases, although levels were not remarkably different from those of controls. Breast-fed children had higher levels in their serum than bottle-fed children. Follow-up of children up to 9 years later, including some cases that were reported to have been produced by breast-milk transmission alone, showed apathy, lethargy, and soft neurological signs (3). The growth deficit apparent at birth resolved by about 4 years of age.

In 1979, there was an outbreak of acne among school children near a food-processing plant in Taichung province of Taiwan (4). Investigation proceeded much more rapidly than had been the case in Japan 11 years earlier, and contaminated rice oil was again the vector. Although the specific machine was not identified, residue under the site of a scrapped machine showed PCB-contaminated cooking oil. At this time, over 2000 patients are being followed by health authorities. The symptoms, signs, and laboratory findings are roughly similar to the Japanese outbreak, except that some of the milder cases have diffuse skin pigmentation without acne. Babies born to affected mothers show the same syndrome as in Japan, including several with natal teeth. We examined 117 such children in 1985 (5) and have followed them since. They have a variety of ectodermal defects, such as excess pigmentation, cataracts, teeth, poor nail formation, and short stature. They also have a developmental delay, on the average of about 5 to 8 points on standard IQ scales, which has both persisted as they have gotten older and is no better in children born up to 6 years after exposure than it is in those born in 1976 (6). The children also score higher on tests measuring poor conduct and hyperactive behavior (7).

Since PCBs accumulate in human beings, since they are of known toxic potential, and since they are present in a wide variety of goods and products, strict regulations concerning their disposal were developed as part of the Toxic Substances Control Act (TOSCA) of 1976. PCBs are no longer produced in the United States; however, large amounts have yet to be dealt with as the large transformers and other heavy machinery of which they are a part are out and must be discarded. Under TOSCA, wastes with greater than 50 ppm PCBs must be handled as hazardous substances and be disposed of in special landfills or subjected to high-temperature incineration. Both of these options are expensive. In addition, there are many old dump sites and abandoned
factories that afford poorly controlled or uncontrollable access to PCBs. Finally, there is a substantial fear that illegal dumping of substances will become more common as regulations are enforced. The experience of North Carolina, where about 200 miles of roadside were contaminated by dumped PCBs, would seem to bear this out (8). We had access to breast-milk samples of 11 women who told us that they lived on or near or had contact with this spill. We found that while their levels were no higher than expected, the heavily chlorinated congeners of the kind found in the spill occurred more frequently in the chromatograms of their milk. This indicated that absorption had likely taken place, but that the amount was small in relation to the existing body burden.

**Hexachlorobenzene**

A dermatologist at the government hospital at Diyarbakir in southeastern Turkey described an outbreak of porphyria cutanea tarda in 1957. The cause was the ingestion during periods of famine of seed wheat that had been treated with hexachlorobenzene as a fungicide. Until the government ban of treated wheat seed in 1959, almost 3000 cases were reported. The attack rate was highest among children 5 to 15 years of age. Patients had photosensitivity, skin pigmentation, bullae, hypertrichosis, and bright red urine. There was about a 10% case fatality rate (9).

Younger children tended not to develop porphyria. Rather, breast-fed children of cases had pembe yara (pink sore), which included weakness, convulsions, and an anular papular rash. This condition carried about a 95% case fatality rate, and cohorts of children died in some of the villages. The chemical was shown to be present in breast milk. Cripps and colleagues have followed 32 cases of porphyria turcica for 20 years (10) and still find elevated porphyrin excretion in urine among some and hexachlorobenzene still detectable in breast milk of one case. Attempts have been made to treat patients with chelating agents, with mixed success.

In the United States, there have been several accidental exposures, but no porphyria. Although residues have been reported in fat samples from those not occupationally exposed in Europe, Japan, and Canada, fat samples in the United States have not been shown to be generally contaminated (11). The major use of hexachlorobenzene is as a seed treatment to control wheat bunt; however, the chemical is a byproduct of several industrial synthetic processes and appears as a contaminant in waste flows. The chance for population exposure in the United States to treated seed is nil; exposure during transport or disposal of waste is a possibility.

**Endrin**

Endrin is a cyclodiene that was used in the United States only for control of birds in airplane hangars. Transport of endrin in the same trucks used for sugar transport was incriminated circumstantially in an outbreak in Pakistan (12). Nineteen of 194 affected persons died, most of an acute seizure disorder and respiratory arrest. Seventy percent of cases were children. Epidemiologic investigation showed that consumption of sugar that had been transported in trucks contaminated by the pesticide was associated with the illness, and the sugar was found to be contaminated with endrin. The authors stated that the levels found in the sugar were not high enough to be the obvious cause, but that there may have been foods high in sugar that children ate preferentially.

**Lead**

For a history of lead poisoning, see Lin-Fu (13). For a review of the effects of low-level exposure on cognition, see Needleman (14). White lead paint, which was an equal mixture of lead oxide and linseed oil, produced a durable, washable, white finish. It was used in the United States for interior surfaces in expensive housing and in high moisture or high abrasion areas, such as doors, windows, kitchens, and bathrooms, in all kinds of housing through the 1960s and 1970s. Lead had been known to be toxic from antiquity, and lead colic and wrist drop were known among painters and smelter workers. As the inner-city housing stock began to deteriorate in the 1940s and the process accelerated in the 1950s, interiors of homes painted with the expensive lead paints were not maintained, since families were unable or landlords were unwilling to do so. Lead paint chips, containing about 50% lead by weight, were ingested by children. Chips are poorly absorbed, but lead is poorly excreted, and children who had the habit of mouthing or swallowing paint chips could develop a cumulative dose leading to lead poisoning. Lead inhibits the formation of heme, the pigment in red blood cells that carries oxygen, and such children were anemic. They were constipated, further slowing the elimination of chips, which were visible in the intestines on X-ray. They stored lead in the metabolically active areas of their long bones, leading to lead lines visible on X-ray. In severe cases, they became unable to regulate the volume and pressure of the fluid surrounding their brains, leading to lead encephalopathy, coma, convulsions, and often death. Early clinicians believed that a child who recovered from a bout of lead encephalopathy did so without sequelae, but with improving medical supportive care and the introduction of chelating drugs that promoted the excretion of lead, more children survived, and follow-up showed that they were frequently mentally retarded or had disorders of behavior and conduct.

Clinicians were familiar with the idea that a disaster of the central nervous system, such as trauma or infection, could permanently compromise brain function; and the idea that lead encephalopathy could have that effect was accepted. However, lead encephalopathy was rare, and mental retardation and behavior disorders were common. Thus the question arose as to whether exposure to lead insufficient to produce encephalopathy might be sufficient to affect behavior and intelligence. These problems, when studied by finding children with retardation or disordered behavior and comparing their lead exposure with that of controls, were confounded by two questions. First, since lead exposure through deteriorated housing occurred in disadvantaged families, and since there appeared to be an independent effect of social class on IQ and behavior, might it be that the circumstances of the children's lives produced both the effects on their brains and their lead exposure? Second, since children got their lead from eating paint, and more active, less intelligent children were thought to exhibit more frequent and promiscuous mouthing behavior, might not the behavior have produced the lead exposure rather than the lead exposure the behavior?

Only in the 1980s, following studies in which children were identified at birth and followed, did the idea that lead exposure preceded and produced disordered behavior and deficits in intelligence achieve acceptance. In addition, the levels at which such effects were detectable turned out to be much lower than expected, and the duration of the effect, now documented to be well into school age, longer than expected. Children are now thought to be lead poisoned at levels that, a decade ago, were thought to be safe, and millions of children...
in the United States have what public health authorities have declared to be unacceptable lead exposure.

Lead paint was not meant to poison children, and it is easy to imagine that no one thought children would be exposed to it or that they would be as sensitive to it as they are. No amount of experience with adult toxicology would allow prediction of the young child’s response to lead. Perversely, two of the three major industrial uses of lead, in paint and in gasoline, inadvertently insured the widest possible exposure for children; in fact, it is difficult to think of two methods better suited to saturation exposure than putting it on the walls and vaporizing it into the air. Lead is now gone from gasoline, but another generation of children will grow up in housing painted with lead oxide and linseed oil. Advertisements from before World War II extoll the extreme durability of the paint, and the copy was right.

Mercury Poisoning in Minamata, Japan

Minamata is a fishing village in Japan and also the home, since 1907, of the Chisso chemical corporation. By tradition, the fisherman sold their best fish, took the second best for their families, and gave the worst fish to the cats. In 1952, the cats began walking stiff-legged about the docks and were reported to be “committing suicide”; in 1956, the first cases of disturbed speech, gait, and loss of fine motor skills occurred in the fishermen’s families. The cause was determined to be a heavy-metal poisoning in October of that year; however, controversy over the source remained. Chisso Corporation produced acetatedehyde by a process involving mercury, which was then discharged into the bay. Chisso, however, denied that the illness was due to the mercury from their plant. It was not until 1968 that the Japanese government declared that Chisso’s discharges were the cause of the illness. By January 1975, about 800 patients were confirmed, with another 2800 seeking indemnification.

Not all of the families who made their living from the bay were affected. However, by 1962, 17 cases of a cerebral palsylike illness developed in the children of women thought to be healthy but who had consumed fish. Some of these children died, and autopsy showed widespread lesions of the cerebral cortex attributable to mercury poisoning.

The Minamata episode led to the founding, in 1970, of the Central Pollution Board in Japan. The episode illustrates several aspects that have recurred at other times and places. First, a chemical etiology was not thought of or sought for the illness, and the victims were feared because they appeared to have a contagious disease. Second, children and fetuses were more susceptible because of their developmental stage, in the sense that doses that were insufficient to produce obvious illness in the pregnant women were sufficient to cripple the children.

Organic mercury remains a worldwide pollution problem, especially for cultures that depend on ocean fish for most of their protein. Mercury is still widely used in the photographic and battery industry and is a significant part of the waste stream. There is no evidence that the levels of either organic or inorganic mercury that occur commonly in the United States produce illness, but the margin of safety is likely not large. About half of the mercury exposure is organic, and probably comes from low-level food contamination. The other half is from metallic mercury used in restorative dentistry.

DDT

Dichlorodiphenyl trichloroethane (DDT) enjoyed extremely widespread use from its introduction in the 1930s to the withdrawal of its registration by the U.S. EPA in 1972. It is stable in the environment and remains active long after initial application. It was this stability, in fact, that led to its role as a widespread environmental pollutant. Once in the food chain, it resisted ultimate metabolism and was biocentered in predators. Among those predators was man, and during the 1960s evidence accrued that residue levels of DDT or its metabolites were detectable with very high prevalence in human fat tissue (as a consequence of their fat solubility) and human milk (as a consequence of its fat content).

Certain of the DDT family have some hormonal activity and are inducers of the mixed function oxidase system of enzymes. It was some combination of these properties that is hypothesized to have been the mechanism for the effect on sea bird reproduction of DDT. A-p-DDT is a weak estrogen, and the metabolism of estrogen is accomplished in part by the P-450 system. Thus, an estrogen imbalance is thought to have resulted in the thin shells and decreased hatching that was reported. DDT is a carcinogen in mice.

As part of a project in which we were looking for morbidity in children exposed to DDT (and PCBs) in breast milk, we collected breast-milk samples from about 800 women in North Carolina and followed their children. We had hypothesized that, rather than seeing a strong increase in illnesses in breast-fed children if DDT were toxic, we would see at most a mild increase and that women would then wean their children earlier, since it is clinical practice in the United States to provide supplemental feedings to children who are breast-fed but not doing well. As it turned out, we saw no evidence of increased illnesses among children even at the highest levels of DDT contamination. We did, however, see an unexpectedly large difference in lactation performance between women at the extremes of the DDT distribution; the women with the highest levels breast fed less than 40% as long as women with the lowest levels (20). We have preliminary data from a study we did in Mexico, where levels are higher, of a similar effect.

Breast feeding is the optimum way to feed a baby. Although there is at most a small difference in mortality between breast- and bottle-fed children in the United States, the differences in countries where there is not clean water or money for enough formula are huge. If this relationship between a broadcast pesticide and lactation performance is causal, it has implications for infant mortality. Further, if the mechanism is through the estrogenicity of DDT, then the problem is even greater. DDT use is declining throughout the world, but many pesticides have estrogen-like properties. To initiate lactation, women need a very low level of estrogen so that prolactin can act unopposed. This is another example of the vulnerability of children to physiologic disruption, this time because of their unique food supply, from levels of chemicals that result in no morbidity to adults.

Asbestos

Men who worked around uncontrolled asbestos dust developed severe pulmonary fibrosis and died young of right heart failure or pulmonary failure. As levels of exposure diminished, the workers lived longer, only to die of lung cancer, especially if they smoked cigarettes. As levels came down even more, workers who lived long enough developed mesothelioma, a tumor of the lining of the lung cavity or the abdominal cavity. Mesothelioma is uncommon, even among asbestos workers. It has a very long latency period, that is, the time between exposure and the development of illness.
can be decades. Many more workers might develop mesothelioma if they lived long enough, but the tumor's latency is longer than their life span. Asbestos dust, however, is tenacious. Anderson et al. (22) reported several cases of mesothelioma arising in the children of asbestos workers. Their source of exposure was dust brought home on their fathers' work clothes. Although this exposure was low, it took place very early in life, and the children had a long time during which to express disease. These tumors showed up when the offspring were in their 30s and 40s.

The first report of mesothelioma in association with asbestos exposure also resulted from an exposure that is peculiar to childhood. Wagner et al. (23) reported that children who played around the mine tailings in an asbestos-rich area of Cape Province in South Africa developed mesothelioma later in life.

Asbestos exposure is common, even though asbestos is now banned, and mesothelioma incidence has been rising. Some of this is due to the large number of workers who were employed short-term in shipyards during World War II (24). So far, disease in their offspring, who would now be in their 40s and 50s, has not been reported.

**Polybrominated Biphenyls**

In 1973, the Michigan Chemical Company was marketing two compounds with similar names and packaging—Nutrimaster, a magnesium oxide supplement for dairy cows, and Firemaster, a polybrominated biphenyl mixture used as a flame retardant. A mix-up occurred in shipment, and an unknown amount of PBB was mixed with feed. Cows developed a toxic syndrome, the cause of which was not readily apparent, since PBBs are not detected in routine analysis for pesticide residues; and other tests gave confusing or negative results. Eventually, a USDA chemist observed a very late-emerging peak on a feed sample chromatogram that had been left running inadvertently and identified the unusual pattern as PBB. By that time, however, cows, swine, and chickens that had eaten the food had been marketed.

Chemically, PBBs are similar to PCBs; they differ only in the halogen substitution. Since they were not intended as food additives or drugs, there had been very little toxicologic information gathered on them prior to marketing. There was no reason to believe that they would be any less toxic than PCBs, however, and a series of quarantines went into effect, resulting in the slaughter of thousands of livestock over the next few years. Little of the contaminated meat and dairy products made its way out of state. However, farm families both consumed their own goods and sold locally, and those who ate the food absorbed and stored the chemical. Like PCBs, PBBs are fat soluble and resist metabolism or excretion. By 1978, Michigan public health officials were able to show that more than 90% of a random sample of lower peninsula nursing women had PBBs in their milk; by inference, 90% of the population was detectably contaminated (25). The state eventually offered to test breast milk for PBBs and adopted advisory levels above which breast feeding was discouraged. In general, these were calculated to allow for the fact that the infant’s mass was increasing, and that intake below a certain level would still allow a net decrease in concentration. The impact of these programs is difficult to measure. There has not been a report of any specific illness occurring in a child attributable to PBB in his milk; thus, whether any morbidity was prevented by advisory activity is conjectural.

A variety of illnesses were ascribed to residents to the toxic effects of PBBs; and several state and federally sponsored surveys attempted to relate morbidity, mortality, or outcome of pregnancy to the degree of contamination. The “PBB syndrome”—asthenia, fatigue, short-term memory loss, dermatitis, and hypersomnia—did not seem to appear more frequently in those with higher blood levels of chemical, nor at all in Michigan Chemical Company workers, who had the highest levels. However, there were reports of T cell abnormalities (26), hepatosplenomegaly, and nonspecific liver function changes. No teratologic syndrome was reported in children born to farm families, and several studies of childhood morbidity did not show any consistent syndrome (27,28). However, when farm children were arrayed by their level of PBB as determined by analysis of fat biopsy specimens, there was a consistent tendency for those above the median to show lower developmental scores, as measured by the McCarthy Scales of Children’s Abilities (29). Overall, most children were in the normal range.

The long-term consequences, if any, for the health of Michigan children are unclear, although the passage of time has provided some reassurance. A cohort of over 4000 people, including children and reproductive-age women, is under prospective surveillance by the state. Conditions that have long latency periods may only develop after ten or more years of observation, but thus far no striking patterns of illness have emerged. Body burdens of the chemical, as measured by periodic screens and rescreens of serum samples, have remained remarkably stable, although there is some suggestion of a downward secular trend.

**Discussion**

It is intuitively plausible that children are more vulnerable to their environment than adults. Children are less able to identify substances as toxic; they are curious; they have a fascination with the beautiful, such as liquid mercury, and the gross, such as the slime on a waste site pool. They have higher intakes (per kilo) and metabolic rates than adults; the demands on their metabolism for growth are much higher; their detoxification systems are not mature. Diseases that take a long time to appear can result from exposures in childhood, while the consequences of the same exposure later in life might have appeared if death from some other cause had not supervened. Examples of environmental chemical exposures leading to greater morbidity in children illustrate this vulnerability. The selection of examples here is highly arbitrary and not exhaustive, and tends to be international. The United States has thus far been spared a severe outbreak of obvious chemical toxicity. This has a great deal to do with the scrutiny given food and drugs, but should not be interpreted as a guarantee. The food supply is complex and vulnerable to contamination at many levels. Household use of pesticides and other agents is universal. Proper disposal of chemical waste is an enormous problem only beginning to be solved. Experience implies that suspicion of a chemical etiology is a necessary first step, typically one taken by the patient, then by the clinician, then by authorities.

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