

CHILDREN'S EXPOSURE TO DIOXINS AND RELATED COMPOUNDS: A CALL FOR URGENCY FOR INTERNATIONAL ACTION ON THE GLOBAL POPS CONVENTION

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Introduction

Negotiations are underway for a global agreement on persistent organic pollutants (POPs). Dioxins, furans and PCBs will pose a particular challenge because of the difficulty in eliminating reservoirs in the environment and the costs associated with transition to alternative technologies and pollution controls. Children are often more susceptible to environmental pollutants.(1) They may be more sensitive because of their rapid growth and development and the vulnerability of developing organ systems, especially the nervous system. They may be more exposed because of different dietary constituents, because they breathe, eat and drink more per body weight than adults and because of their different behavioral patterns. Consideration of exposures to children, and the local and regional nature of sources to children, is important to informed decision making for the negotiations.

Materials and Methods

This paper examines the hazards of dioxins, furans and PCBs to children on a worldwide basis. A conventional risk assessment approach is used to identify potential hazards and exposures to children.(2) First, hazard identification is used to indicate the likely health hazards of concern to children. Second, exposure sources and pathways are examined, with particular emphasis on exposures that are likely to significantly contribute to children's risks. Exposures to adults and children, per body weight, are compared. The most significant exposure pathways are identified, with particular emphasis on significance to developing countries.

Results and Discussion

Hazard Identification

While there are significant concerns about both short and long-term health impacts of dioxins and related compounds on children, there are also considerable uncertainties. There is both direct evidence in humans that comes from studies of PCB exposures in highly exposed (due to accidental addition of PCBs into foods) and moderately exposed (via fish consumption) populations and indirect evidence derived from animal tests.(3) In human populations, studies of PCB exposures show that effects vary according to time of exposure and dose and are summarized in Table 1.(4, 5) In addition to these effects, which have been demonstrated in human populations, there are less certain but very serious concerns about low level exposures. In 1998, the International Agency for Research on Cancer (IARC) classified dioxin as a human carcinogen.(6) Recent studies indicate that dioxins and PCBs may alter thyroid hormone function of both pregnant women and infants.(7)

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Table 1: UNIQUE EFFECTS OF PCB AND DIOXIN EXPOSURES TO CHILDREN

Prenatal Exposure to Low Levels of PCBs

Newborns: decrease in birth weight (inconsistent)

Infants: motor delay detectable from newborn period to 2 years

7-month olds: defects in visual recognition memory

4-year olds: defects in visual recognition memory

11-year olds: delays in cognitive development

Prenatal exposure to high levels of PCBs and PCDFs

Newborns: low birth weight, conjunctivitis, natal teeth, and pigmentation

Infant through school age: delays on all cognitive domains tested: behavior disorders, growth retardation, abnormal development of hair, nails and teeth, pigmentation, and increased risk of bronchitis

Puberty: small penis in boys; growth delay in girls

Direct ingestion of high doses of PCBs and PCDFs

Any age: chloracne, keratoses, and hyperpigmentation; peripheral neuropathy; and gastritis

Dermal exposure to high levels of TCDD

Children: probably higher absorbed dose for a given exposure than adults; chloracne and liver function test abnormalities

Exposure Pathways

Prenatal transfer occurs via movement of contaminants across the placenta to the fetus. The greatest quantity is delivered during the period of rapid growth in the last trimester of pregnancy. Prenatal exposures to dioxins and PCBs have been documented to be associated with numerous adverse health effects at both low and high levels.

Lactation is a major source of exposure.(8) The World Health Organization recommends breast milk as the best food for infants due to its superiority from the standpoint of nutrition, immunologic properties, sanitation and promoting attachment between mother and child. Therefore, protection of the quality of breast milk is of paramount importance. Population studies indicate that the greatest dosage of dioxins over a lifetime of exposure is delivered to infants in breast milk. Highest exposures have been found in populations like the Inuit where the diet contains higher levels of dioxins.(9) Perinatal exposures to PCBs and dioxins have been found to be strongly associated with dietary intake of fish, chickens, eggs, dairy products and industrial oils.(10)

Dietary consumption is an important contributor. Because children eat more calories per body weight, foods like milk, eggs and fish with the identical level of contamination will give a higher dosage (exposure per body weight) for children than for adults. Of particular concern is consumption of fish from contaminated waters and of products of subsistence agriculture where there are local sources of dioxin contamination.

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In the U.S., subsistence farming in the proximity of pentachlorophenol wood preservative operations and poorly controlled industrial incinerators has led to higher levels of dioxins in eggs, chickens, cows and pork.(11, 12) Industrial accidents in which foods have been inadvertently contaminated have been unusual but serious sources of exposure. Recently in the U.S. a food additive for chicken and catfish feed was found to contain dioxin contamination.(13)

Inhalation is a minor source of exposure to the general population but could be significant in the presence of local sources of contamination and in the case of serious industrial accidents.

Soil ingestion is another possible route of exposure for children. Because children have normal behaviors that involve hand to mouth activity as well as mouthing toys and other objects, they consume more soil than do adults. The U.S. Environmental Protection Agency has long used 1ppb as an action level for dioxin TEQ in soil.(14) This is a much higher level than levels associated with contamination in food animals due to subsistence farming and very infrequently encountered except in heavily contaminated industrial sites and in spills.

Integration:

Children are susceptible to the effects of dioxins and PCBs and have unique susceptibilities in terms of prenatal and developmental hazards. Of particular concern on a population basis are effects on learning and neurological development. Children are also more exposed to dioxins and PCBs than adults, during the prenatal period, lactation, and the first few years of life. The most important routes of exposure are placental transfer, lactation and diet. Of most concern are populations with higher local sources of exposure. These include indigenous populations that are subsistence fishers and farmers. Sources may be near at hand (in the case of subsistence farmers) or far away (in the case of the Arctic Inuit). Higher exposures to children may occur wherever there are sources, not just in the polar regions but also in, for example, the hot desert region the Mojave desert in the U.S. when there are local emission sources. Because dioxins accumulate in the environment over time the concern for the health of children calls for a bold and far reaching approach to dioxins in the context of the global agreement on POPs.

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