International Journal of Occupational Medicine and Environmental Health, 2004; 17(1): 175—177

CHILDREN AS A VULNERABLE POPULATION

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Abstract. Children are not little adults. They have unique patterns of environmental exposure and developmentally determined susceptibilities that increase their risk of disease following toxic environmental exposure. Evidence is accumulating that children's exposure to toxic chemicals in the environment is contributing to changing patterns of pediatric disease.

Key words:
Environmental pediatrics, Vulnerable populations, Children's environmental health

INTRODUCTION

Patterns of illness have changed dramatically among children in the industrially developed nations of the world over the past century. The classic infectious diseases have been greatly reduced and many of them controlled. Infant mortality has been lowered. Life expectancy at birth has increased by more than 20 years. Today, the most serious diseases confronting children in the developed nations are a group of chronic, disabling, and sometimes life-threatening conditions termed the "new pediatric morbidity". Examples include asthma, for which incidence and mortality have more than doubled in the past decade; childhood cancer, for which incidence of certain types has increased substantially; neurodevelopmental and behavioral disorders; diseases caused by environmental tobacco smoke; and certain congenital defects of the reproductive organs such as hypospadias, for which incidence in the past two decades has doubled.

The causes of the chronic diseases and developmental disabilities that are so highly prevalent today among America's children are not well understood. An estimated 10–20% of these diseases appear to be of familial or genetic origin. Some are the consequence of obstetrical difficulties, infections, or trauma. But for the remainder, the causes are only beginning to be known.

In recent years, it has become increasingly recognized that children's exposures to toxic chemicals in the environment cause or contribute to the causation of certain of these diseases. The US National Academy of Sciences has estimated that toxic exposures in the environment contribute to the causation of as much as 28% of neurobehavioral disorders in children. Lead is now recognized as a significant cause of neurobehavioral impairment, producing measurable disability (loss of IQ) at very low levels in blood, as well as alteration of behavior. Exposures *in utero* to polychlorinated biphenyls (PCBs) and methyl mercury can also cause loss of intelligence. Certain pesticides appear to interfere with brain development and reproductive function.

Children are extensively exposed to synthetic chemicals in the environment. More than 80 000 new synthetic chemical compounds have been developed and disseminated in the environment over the past 50 years. Children are at special risk of exposure to the 2800 high-volume chemicals

Received: January 19, 2004. Accepted: January 30, 2004.
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that are produced in quantities greater than one million pounds per year and that are most widely dispersed in air, water, food crops, communities, waste sites, and homes. Fewer than half of these high-volume chemicals have been tested for their potential toxicity, and fewer still for their possible developmental toxicity to fetuses, infants, and children.

CHILDREN'S UNIQUE VULNERABILITY

Children are highly vulnerable to chemical toxins. They have disproportionately heavy exposures to environmental toxicants. Pound for pound of body weight, children drink more water, eat more food, and breathe more air than adults. Children in the first six months of life drink seven times as much water as the average American adult. One- to five-year-old children eat three to four times more food. The air intake of a resting infant is twice that of an adult. The implication of these findings for health is that children will have substantially heavier exposures than adults to any toxicants that are present in water, food, or air. Two additional characteristics of children further magnify their exposures: their hand-to-mouth behavior and their play close to the ground.

Children's metabolic pathways, especially in the first months after birth, are immature. Their ability to metabolize, detoxify, and excrete many toxicants differs from that of adults. In some instances, children are actually better able than adults to deal with environmental compounds such as polyaromatic hydrocarbons and estrogen. More commonly, however, they are less well able to deal with chemical toxins such as lead and organophosphate pesticides because they do not have the enzymes necessary to metabolize them and thus are more vulnerable to them.

Children undergo rapid growth and development, and their developmental processes are easily disrupted. Organ systems in infants and children undergo very rapid change prenatally, as well as in the first months and years after birth. These developing systems are very delicate and are not well able to repair damage that may be caused by environmental toxicants. Thus, if cells in an infant's brain are destroyed by chemicals such as lead, mercury, or solvents, or if false signals are sent to the developing reproductive organs, there is high risk that the resulting dysfunction will be permanent and irreversible.

Because children have more future years of life than most adults, they have more time to develop chronic diseases triggered by early exposures. Many diseases that are caused by toxicants in the environment require decades to develop. Many of those diseases, including cancer and neurodegenerative diseases, are now thought to arise through a series of stages that require years or even decades to evolve from earliest initiation to actual manifestation of disease. Carcinogenic and toxic exposures sustained early in life, including prenatal exposures, appear more likely to lead to disease than similar exposures encountered later.

CASE STUDIES

Understanding of children's vulnerability to environmental toxins traces its origins to early studies of major disease outbreaks of toxic origin in children exposed to high doses of chemical toxins. These analyses formed the basis for our current understanding that children are uniquely sensitive to many toxins in the environment. The following were among these reports:

- A report from Queensland, Australia, in 1904 describing an epidemic of lead poisoning in young children. Clinical and epidemiologic investigation traced the source of the outbreak to the ingestion of lead-based paint by children playing on verandas. This was the first report of lead poisoning in children, and it led to the banning of lead-based paint in many nations, although not in the US.
- Studies of an epidemic of leukemia in the 1950s among young children in Hiroshima and Nagasaki who were exposed to ionizing radiation in the atomic bombings. These and subsequent studies of fetuses exposed *in utero* established that infants and fetuses are more sensitive to leukemia following radiation than adults. Additionally, an increased risk of microcephaly was observed among infants exposed to radiation as fetuses in the first trimester of pregnancy during the atomic bombing.

■ A report from Minamata, Japan, in the 1960s describing an epidemic of cerebral palsy, mental retardation, and convulsions among children living in a fishing village on the Inland Sea. This epidemic was traced to ingestion of fish and shellfish contaminated with methyl mercury. The source of the mercury was found to be a plastics factory that had discharged metallic mercury into the sediments on the floor of Minamata Bay. The mercury was transformed by microorganisms into methyl mercury, and it then bioaccumulated as it moved up the marine food chain, eventually reaching children who ate fish and shellfish. The most devastating effects were seen among children exposed *in utero*.

SUB-CLINICAL TOXICITY

A critically important intellectual step in the development of understanding of children's special vulnerability was the recognition that environmental toxins can exert a range of adverse effects in children. Some of these effects are clinically evident, but others can be discerned only through special testing and are not evident on the standard examination, hence the term "sub-clinical toxicity". The underlying concept is that a dose-dependent continuum of toxic effects exists, in which clinically obvious effects have their sub-clinical counterparts.

The concept of sub-clinical toxicity traces its origins to pioneering studies of lead toxicity in clinically asymptomatic children undertaken by Herbert Needleman and colleagues. Needleman et al. [1] showed that children's exposure to lead could cause decreases in intelligence and alteration of behavior even in the absence of clinically visible symptoms of lead toxicity. The sub-clinical toxicity of lead in children has subsequently been confirmed in prospective epidemiologic studies. Similar sub-clinical neurotoxic effects have been documented in children exposed *in utero* to PCBs and to methyl mercury.

THE NEED FOR PREVENTION

The protection of children against environmental toxins is a major challenge to modern society. Hundreds of new chemicals are developed every year and are released into the environment. The majority of these chemicals are untested for their toxic effects on children. The challenge is to design policies that specifically protect children against environmental toxins. To meet this challenge, a new paradigm for environmental health policy needs to be developed that is centered on the sensitivities and exposures of children. The analysis begins with the child, his or her biology, exposure patterns, and developmental stage. The paradigm calls for a new way of thinking and a re-tooling of the risk assessment process so that it takes into account the increased vulnerability of children and that embodies the Precautionary Principle.

As we begin the 21st century, the issue of environmental exposure looms large globally. It is imperative that we develop prudent policies that will protect the health of our children now and in the future.

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