

# **Child Development and Environmental Toxins**



Why do some people get heart disease, diabetes, or cancer, while others remain relatively healthy throughout most of their lives? Research has shown that chemical exposures during child development may contribute to health problems that arise later in life.

Children's health research is a priority for the National Institute of Environmental Health Sciences (NIEHS). The environment plays a role in 85% of all diseases. New science is showing that the effects of exposure to chemicals at low doses, and in combination, can have an impact on human growth and development.

Some chemicals, pollutants, foods, and other behavioral changes that may have minimal adverse effects in adults, may impact a developing fetus and have long-lasting effects on a child's health even into adulthood. This is sometimes referred to as the *fetal basis of adult disease* or *windows* of susceptibility.

What follows is a compilation of some of the research findings we have on environmental toxins that might impact a child's development.

## **Air Pollution**

Investigators with the Columbia Center for Children's Environmental Health have found evidence of a link between early exposures to urban air pollutants that are formed by the combustion of gasoline and other fossil fuels, and children's cognitive development. Their results show that New York City children who



were prenatally exposed to high levels of these air pollutants scored more than 4 points lower on standardized intelligence tests at age 5 compared with the less-exposed children.<sup>2</sup> A 2011 study conducted by the Columbia Center researchers has linked prenatal exposure to these pollutants with higher scores on tests of anxiety, depression, and attention problems at age 5.<sup>3</sup>

#### Arsenic



Researchers at the University of California, Berkeley have found that Chilean children who were exposed to high levels of naturally occurring arsenic in their drinking water had a higher incidence of liver, lung, and kidney cancer as adults. Similar studies conducted in Japan have shown that infants fed arsenic-contaminated milk powder had higher mortality rates for skin and liver cancer.







## **Dioxins**

Dioxins, a group of environmentally persistent compounds that are by-products of some manufacturing and incineration processes, have been shown to produce a variety of effects in both animals and humans. People can be exposed to these compounds by eating meat, dairy products, and seafood that are contaminated with dioxins.

Data from the Dutch PCB/Dioxin Study conducted in 2000 found that children with higher exposures to umbilical cord blood and breast milk had a greater likelihood of developing recurrent ear infections and chicken pox.<sup>6</sup>

# **Endocrine Disruptors**

A growing body of evidence suggests that certain chemicals, known as endocrine disruptors, can mimic hormones or interfere with the function of the body's hormones. Endocrine disruptors, which usually mimic estrogen, are found in many of the everyday products we use, including some plastic bottles and containers, food can liners, detergents, flame retardants, toys, cosmetics, and pesticides. The compounds are of particular concern because they can alter the critical hormonal balances required for proper health and development.<sup>7</sup>

# ■ Bisphenol A (BPA)

Much of the concern about endocrine disruptors has focused on bisphenol A, a compound that is widely used in the manufacture of polycarbonate plastics and epoxy resins. Research with laboratory animals has shown that low-dose administration of BPA produces a wide spectrum of developmental and reproductive effects, including an increase in aggressive behavior, early onset of sexual maturation, changes in mammary gland development, and a decrease in testosterone levels and sperm production.<sup>8</sup>





## **■** Pesticides

Some endocrine disruptors were banned from commercial use more than 30 years ago, but they persist in the environment and in our bodies. These include DDT, a pesticide that was used to control the spread of mosquitoes and other insects. Scientists at the Center for Research on Women's and Children's Health at the Public Health Institute in Berkeley, Calif., found that girls who were exposed to DDT before the age of 14 had a five-fold increase in breast cancer risk as compared to those who were not exposed to the compound at this stage of life.<sup>9</sup>

## **■** Phthalates

Some endocrine disrupting compounds have the potential to stimulate androgen, a group of hormones that influence the growth and development of the male reproductive system. NIEHS-funded researchers at the University of Rochester are among the first to demonstrate an association between pregnant women's exposure to phthalates, compounds used in many consumer products, such as nail polish, hair spray, deodorant, and shampoo, and adverse effects on genital development in their male children. These investigators have also reported that prenatal exposure to phthalates can significantly reduce masculine behavior in boys. 10

Unexpectedly, NIEHS researchers have also found the use of lavender and tea tree oils, which are present in a number of commercial products, can be another source of estrogenic activity. A small study suggested that repeated topical use of products containing lavender or tea tree oil may cause male prepubertal gynecomastia, a rare condition resulting in enlarged breast tissue in boys prior to puberty.<sup>11</sup>



## Flame Retardants

Research conducted by NIEHS-funded scientists at Duke University suggests that babies are being exposed to at least eight different flame-retarding chemicals found in an array of products from car seats to changing table pads. These chemicals, which are added to polyurethane cushions to slow the spread of flames during a fire, can leak from the cushions and be inhaled or absorbed through a baby's skin.

Research with laboratory animals has shown that some of these chemicals can cause cancerous tumors, while others can alter hormones that are essential to reproductive and neurological development.<sup>12</sup>

#### Lead

Some environmental insults can permanently change the way the body works. For example, early life exposure to lead may change the hypothalamic-pituitary-adrenal axis, a complex system that controls many organ functions. That may explain why early lead exposure significantly increases the risk of hypertension, cardiovascular disease, diabetes, schizophrenia, and neurodegenerative changes later in life.<sup>13</sup>

# **Maternal Smoking**

Research shows that maternal smoking may play a significant role in childhood obesity. Data from the US Collaborative Perinatal Project, a study of 35,000 children born between 1959 and 1964, show that children of smokers had an increased risk of becoming overweight before the age of 8 compared with the offspring of nonsmokers. The link between maternal smoking and obesity was stronger in girls than in boys.<sup>14</sup>

# Mercury

There is increasing evidence that exposure to methylmercury before birth, primarily from maternal consumption of mercury-contaminated seafood, can cause disruptions in neurobehavioral and cognitive development in children. A study of Faroe Islands residents, funded in part by NIEHS, showed a positive relationship between mercury concentrations in the mothers' umbilical cord blood and developmental delays in their 7-year-old children. Scientists observed similar cognitive deficits in these children when tested at 14 years of age. 15



# **Early Puberty**

Results from studies conducted at the NIEHS-funded Breast Cancer and Environment Research Center in Cincinnati have added to widespread concern that girls are increasingly entering puberty at an earlier age. These investigators have found a positive association between early onset of puberty and increased risk of developing breast cancer.<sup>16</sup>

Research conducted by NIEHS/NTP scientists suggests that the mammary gland, the milk-producing structure in the breast, is uniquely sensitive to the effects of toxic chemicals. When pregnant mice were treated with perfluorooctanoic acid (PFOA), an industrial chemical used to make Teflon, the investigators noted delays in mammary gland development and impaired lactation in the offspring. The researchers also noted that chronic exposure of the mice to PFOA in their drinking water altered mammary gland development at concentrations found in contaminated human water supplies.<sup>17</sup>





### Children's Environmental Health Centers

NIEHS has partnered with the U.S. Environmental Protection Agency to support research centers devoted exclusively to children's environmental health and disease prevention. Known as the Centers for Children's Environmental Health and Disease Prevention Research, these centers utilize the expertise and resources of top universities and medical centers to focus on the important role that environmental toxicants play in the development of many childhood illnesses.

- <sup>1</sup> Irigaray P, et al. 2007. Lifestyle-related factors and environmental agents causing cancer: An overview. Biomed Pharmacother 61(10):640-658.
- <sup>2</sup> Perera FP, et al. 2009. Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. Pediatrics 124(2):e195-202.
- <sup>3</sup> Perera FP, et al. 2011. Polycyclic aromatic hydrocarbons-aromatic DNA adducts in cord blood and behavior scores in New York City children. Environ Health Perspect 119(8):1176-1181.
- <sup>4</sup> Liaw J, et al. 2008. Increased childhood liver cancer mortality and arsenic in drinking water in northern Chile. Cancer Epidemiol Biomarkers Prev 17(8):1982-1987.
- <sup>5</sup> Yorifuji T, et al. 2011. Cancer excess after arsenic exposure from contaminated milk powder. Environ Health Prev Med 16(3):164-170.
- <sup>6</sup> Weisglas-Kuperus N, et al. 2004. Immunological effects of environmental exposure to polychlorinated biphenyls and dioxins in Dutch school children. Toxicol Lett 1;149(1-3):281-285.
- Schug TT, et al. 2011. Endocrine Disrupting Chemicals and Disease Susceptibility. J Steroid Biochem Mol Biol; doi:10.1016/j.jsbmb.2011.08.007 [Online 28 August 2011].
- <sup>8</sup> Vom Saal FS, et al. 2005. An extensive new literature concerning low-dose effects of bisphenol A shows the need for a new risk assessment. Environ Health Perspect 113(8):926-933.
- 9 Cohn BA, et al. 2007. DDT and breast cancer in young women: New data on the significance of age at exposure. Environ Health Perspect 115(10):1406-1414.
- <sup>10</sup> Swan SH, et al. 2010. Prenatal phthalate exposure and reduced masculine play in boys. Int J Androl 33(2):259-269.
- <sup>11</sup> Henley DV, et al. 2007. Prepubertal gynecomastia linked to lavender and tea tree oils. N Engl J Med 356(5):479-485.
- <sup>12</sup> Stapleton HM, et al. 2011. Identification of flame retardants in polyurethane foam collected from baby products. Environ Sci Technol 45(12):5323-5331.
- 13 Cory-Slechta DA, et al. 2008. Lifetime consequences of combined maternal lead and stress. Basic Clin Pharmacol Toxicol 102(2):218-227.
- 14 Chen A, et al. 2006. Maternal smoking during pregnancy in relation to child overweight: Follow-up to age 8 years. Int J Epidemiol 35(1):121-130.
- <sup>15</sup> Yorifuji T, et al. 2011. Prenatal exposure to lead and cognitive deficit in 7- and 14-year-old children in the presence of concomitant exposure to similar molar concentration of methylmercury. Neurotoxicol Teratol 33(2):205-11.
- 16 Biro FM, et al. 2010. Pubertal assessment method and baseline characteristics in a mixed longitudinal study of girls. Pediatrics 126(3):e583-590.
- <sup>17</sup> White SS, et al. 2011. Gestational and chronic low-dose PFOA exposures and mammary gland growth and differentiation in three generations of CD-1 mice. Environ Health Perspect 119(8):1070-1076.