

Asthma among WTC Children Registry Yields First Child Health Report

The World Trade Center Health Registry (WTCHR), comprising persons most likely to have been heavily exposed to traumatic events and air pollution related to the World Trade Center attacks of 11 September 2001, includes 3,184 children under age 18 years who were living or attending school in lower Manhattan at the time, who were otherwise near the World Trade Center that morning, or who assisted in recovery efforts. In the first report of children enrolled in this registry, researchers observe that preschoolers exposed to smoke and dust from the collapsing towers had asthma rates twice the national average following the 9/11 attack, whereas asthma rates in exposed older children remained about average [*EHP* 116:1383–1390; Thomas et al.]. Children in certain ethnic groups also experienced disproportionate asthma rates, although the reasons for this are unclear.

Data were collected in 2003 and 2004 by telephone interviews with parents of younger children or the children themselves if they had turned 18 since the attacks. More than half the children reported having respiratory symptoms after the attacks, including cough and sinus problems. Nearly 6% of all children reported having asthma diagnosed after 9/11. At the time of the interviews, 16% of children then aged 2–4 years had been diagnosed with asthma, more than twice the average of 7% for children that age in the Northeast. Asthma rates in older subjects, however, were just slightly higher than the Northeast rate.

Childhood asthma normally develops in a child's first five years of life, often after exposure to an environmental irritant. Smoke and dust from the collapsing towers might have acted as such an early trigger in susceptible preschoolers. The researchers speculate that older youngsters could have had fewer new diagnoses because most susceptible children had been diagnosed before 9/11.

The researchers noted racial disparities in asthma rates. Black and Hispanic children in the WTCHR were twice as likely to be diagnosed with asthma as whites or Asians, both before and after the attacks. Reasons for the racial disparities are unclear, although prior studies on ethnic disparities in asthma suggest that both genetics and environment may play a role in etiology of the disease. Children of all ages and ethnicities were more likely to develop asthma if they were caught in the cloud of cement dust created by the collapsing towers, as pulverized cement dust is known to irritate mucous membranes.

The WTCHR data have several limitations, including lack of information on how long after 9/11 symptoms appeared and the presence of co-factors for asthma. Despite these limitations, findings from the WTCHR, which constitutes the largest collection of post-disaster data of children, could have broad impact, given that tens of thousands of New York City children may have been exposed to smoke and dust on 9/11. Researchers also expect their data to improve understanding of risks to children exposed to other polluting disasters, such as the California wildfires. —*Cynthia Washam*



Collapse of the World Trade Center towers, 11 September 2001

Sometimes Less Is More Perinatal Bacterial Exposure May Be More Important than Hepatitis for Liver Tumor Development

Helicobacter hepaticus, a bacterium discovered in 1994 and widespread in many experimental mouse populations, is associated with a high incidence of liver tumors in aging mice. A new mouse study shows that perinatal exposure to this pathogen, rather than development of hepatitis itself, may be the single most important factor in the development of liver tumors caused by *H. hepaticus* [*EHP* 116:1352–1356; Diwan et al.]. The results support evidence from other studies that progressive hepatitis and liver tumors in older mice may stem from early-life exposure.

The researchers isolated a strain of *H. hepaticus* from infected A/J mice. Female mice received injections with a single dose of the bacteria. Females testing positive for the bacteria were bred with uninfected males. The researchers assessed liver histopathologic findings and tumor growth in male offspring aged 2 weeks

to 2 years. Uninfected weanling males from another lab were injected at 3–4 weeks.

The results showed a significant incidence of liver tumors in the offspring after intraperitoneal maternal exposure to the bacteria: 33% developed liver tumors, usually multiple tumors, and 18% developed hepatocellular carcinoma. None of the mice injected with the bacteria as young adults developed any tumors.

Another striking result was that tumor outcome was not closely linked to severity of hepatitis; mice that contracted hepatitis did not necessarily develop hepatic tumors. Rather, it appeared that early exposure to the bacteria, not the hepatitis itself, was key to fostering tumor growth.

The type of additional perinatal event required to induce tumor development is unknown but could involve DNA damage at that vulnerable early stage, followed by subacute inflammation of the liver in response to *H. hepaticus* infection. The authors note that similar scenarios could apply to human infection with *Helicobacter pylori* and other pathogens linked with cancer. The results point to the need for further study of changes in perinatal tissues in response to such infections. —*David A. Taylor*

Reuters/Ray Subblebne

Developing Story

No Link Observed between Prenatal PFOA/PFOS Exposure and Milestone Achievement

Perfluorooctanoate (PFOA; also known as perfluorooctanoic acid) and perfluorooctanesulfonate (PFOS), chemicals used in waterproofing fabrics and greaseproofing fast-food containers, among other applications, have been detected at low concentrations in 98% of the U.S. population. These chemicals have half-lives of several years, and children tend to have higher serum concentrations than adults. Animal and human studies have hinted at a link between PFOA and PFOS and developmental effects, but human studies have been limited. A new human study suggests that maternal plasma levels of PFOA and PFOS may not be associated with delayed early development in babies [*EHP* 116:1391–1395; Fei et al.].

The research team randomly selected 1,400 mother–baby pairs from the Danish National Birth Cohort comprising 100,000 women recruited during early pregnancy between 1996 and 2002. The team measured PFOA and PFOS levels in maternal blood samples taken in the first trimester of pregnancy. Each newborn's Apgar score was obtained from Danish hospital records; this assessment of viability taken immediately after birth measures heart rate, respiratory effort, reflex irritability, muscle tone, and skin color.

When the children were 6 and 18 months old, their mothers provided data on gross and fine motor functioning, attention, cognitive function, language, and social-personal development via computer-assisted telephone interviews. The study was funded by the International Epidemiology Institute, which received money from the 3M Company, the original manufacturer of PFOA and PFOS, and the 3M Toxicology Laboratory performed the chemical analyses.

Earlier human and animal studies suggested that PFOA and PFOS might reduce fetal growth, delay learning, accelerate or delay sexual maturation, and produce other developmental effects. The current study found no significant association between maternal PFOA/PFOS levels and child achievement of early developmental milestones such as walking without support, taking off socks and shoes when asked, and turning a picture book right side up. However, a statistically nonsignificant association was observed between higher maternal PFOS levels and delay of a child's ability to sit without support.

PFOA and PFOS were significantly higher among first-time mothers than in women who had given birth previously. This may confound their results, because the presence of older siblings may accelerate the developmental progress of younger ones. Given animal studies showing potential adverse effects of PFOA and PFOS, and the limited data for humans, the authors write that additional studies should be conducted using more sensitive measures of early childhood development. —Valerie J. Brown

Clear Shot at Better Outcomes?

Closure of Coal-Burning Plants Could Improve Neurodevelopment

Coal burning, which provides up to 75% of China's electricity, is the main environmental source of polycyclic aromatic hydrocarbons (PAHs) in that country. Research in Europe, the United States, and Asia indicates that prenatal exposure to PAHs increases the risk of reduced fetal growth and adverse neurodevelopmental effects. Confirmation for these studies comes in new research in China, which suggests that reduction of prenatal exposure to PAHs was linked to improved developmental outcomes in a small group of Chinese children [*EHP* 116:1396–1400; Perera et al.].

The study took advantage of the opportunity to evaluate health-related effects of the prescheduled closure of a coal-fired power plant. Subjects included about 110 children in each of two parallel mother–infant cohorts in Tongliang, Chongqing Province. The first (2002) cohort was enrolled two years before the 2004 shutdown of the power plant; the second (2005) cohort was enrolled the year after the shutdown.

The infants were followed from birth through their second birthdays, at which time the investigators assessed the children's developmental attainment using the Gesell Developmental Schedules. Using high-performance liquid chromatography, they

analyzed PAH–DNA adduct levels in cord blood collected at delivery and also measured potential confounders for neurodevelopmental effects, including lead, mercury, and secondhand tobacco smoke. The relationships between PAH–DNA adduct levels and developmental outcomes in the two cohorts were evaluated through the use of multiple linear regression and logistic regression, adjusting for potential confounders. Cohort developmental outcomes, including frequency of developmental delay, also were compared.

The investigators found that the 2005 cohort had 40% lower PAH–DNA adduct levels in cord blood compared with the 2002 cohort. Earlier studies of the 2002 cohort showed significant associations between elevated adduct levels and lower average and motor development scores; however, these associations were not observed in the 2005 cohort. The frequency of developmental delay in the motor area was significantly reduced in the 2005 cohort compared with the 2002 cohort.

The results are limited by the lack of data on postnatal PAH–DNA adduct levels. However, the authors note that in 2002 the plant was the major source of PAH emissions in the study area (residential heating and cooking had been converted to natural gas). Thus, the results provide molecular epidemiologic evidence that developmental outcomes in infants were improved after the coal-burning power plant was shut down—a finding relevant to child development throughout China as well as other countries relying on coal and other fossil fuels for energy. —Tanya Tillett



Coal-fired power plants such as the Datong facility in the background provide 75% of China's electricity.