

Prenatal exposure to air pollution linked to childhood obesity

April 16 2012

Overall, 17% of children in the United States are obese, and in inner-city neighborhoods, the prevalence is as high as 25%. While poor diets and physical inactivity are the main culprits, there is new evidence that air pollution can play a role.

A study by Columbia University's Mailman School of Public Health finds that pregnant women in New York City exposed to higher concentrations of chemicals called [polycyclic aromatic hydrocarbons](#), or PAH, were more than twice as likely to have children who were obese by age 7 compared with women with lower levels of exposure. PAH, a common urban pollutant, are released into the air from the burning of coal, diesel, oil and gas, or other [organic substances](#) such as tobacco.

Results are published in the *American Journal of Epidemiology*.

"[Obesity](#) is a complex disease with multiple risk factors. It isn't just the result of individual choices like diet and exercise," says the study's lead author Andrew G. Rundle, Dr. P.H., a professor of epidemiology at Columbia's Mailman School of Public Health. "For many people who don't have the resources to buy healthy food or don't have the time to exercise, [prenatal exposure](#) to [air pollution](#) may tip the scales, making them even more susceptible to obesity."

Researchers recruited 702 non-smoking pregnant women through prenatal clinics at NewYork-Presbyterian Hospital and Harlem Hospital. The women were 18-35 years old, identified themselves as either

African-American or Dominican, and lived in areas in Northern Manhattan or the South Bronx that are predominantly low income. Over the course of two days during their [third trimester](#), they wore a small backpack equipped to continually sample the surrounding air; at night they placed it near their bed.

Children of women exposed to high levels of PAH during pregnancy were nearly twice as likely (1.79 times) to be obese at age 5, and more than twice as likely (2.26 times) to be obese at age 7, compared with children of mothers with lower levels of exposure. The 7-year-olds whose mothers were in the highest exposure group had, on average, 2.4 lbs. more fat mass than children of mothers with the least exposure.

"Not only was their body mass higher, but it was higher due to body fat rather than bone or muscle mass," says Dr. Rundle.

These findings fit with evidence from animal studies and tissue sample experiments. Mouse studies have shown that exposure to PAH causes gains in fat mass, while cell culture studies have shown that exposures to PAH prevent normal lipolysis, the process by which fat cells shed lipids and shrink in size.

Previous research at the Columbia Center for Children's Environmental Health (CCCEH) at the Mailman School found that prenatal exposure to PAH can negatively affect childhood IQs and is linked to anxiety, depression and attention problems in young children. PAH also disrupt the body's endocrine system and are known carcinogens.

Fortunately, there are ways to reduce PAH exposure. Certain fuels release more of the chemicals than others, explains Dr. Rundle, and efforts in New York City to take diesel buses off the streets and retrofit oil furnaces so they burn cleaner fuel is already starting to help.

Despite known linkages between socioeconomic status and obesity levels, the researchers found the impact of [PAH](#) on risk of obesity was not influenced by household income or neighborhood poverty. They also ruled out the influence of cigarette smoke in the household and proximity to highly trafficked roads.

Robin Whyatt, DrPH, the paper's senior author, notes that the study is one of the first to present evidence that chemicals in the environment can contribute to obesity in human beings. Future research will focus on identifying other examples of these "obesogens" and ways to reduce them, says Dr. Whyatt, who is deputy director at CCCEH and professor of clinical environmental health sciences at the Mailman School.

Provided by Columbia University

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