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Research suggests pollution-related asthma may start in the womb

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Children born in areas with increased traffic-related pollution may be at greater risk of developing asthma due to genetic changes acquired in the womb, according to new research from the University of Cincinnati (UC) and Columbia University Mailman School of Public Health. The team reports its findings in the Feb. 16, 2009, issue of *PLoS ONE*.

In a study of umbilical cord blood from New York City children, researchers have discovered evidence of a possible new biomarker—an epigenetic alteration in the gene ACSL3—associated with prenatal exposure to polycyclic aromatic hydrocarbons (PAHs). These chemical compounds are created as byproducts of incomplete combustion from carbon-containing fuels, resulting in high levels in heavy-traffic areas. Exposure to PAHs has been linked to diseases such as cancer and childhood asthma.

Researchers say this finding provides a potential clue for predicting environmentally related asthma in children—particularly those born to mothers who live in high-traffic areas like Northern Manhattan and South Bronx when pregnant.

This is the first study to examine the effects of prenatal ambient air pollutant exposure on epigenetic changes linked to asthma. Epigenetic changes may disrupt the normal functioning of genes by affecting their expression but do not cause structural changes or mutations in the genes.

For this study, UC researchers teamed with Columbia's Mailman School

of Public Health to study the relationship between prenatal PAH exposure and childhood asthma, hypothesizing that transplacental exposure to PAHs could "reprogram" fetal genes and lead to airway inflammation or asthma during childhood. Epigenetic reprogramming is the result of an organism's genes interacting with the environment.

"Our data support the concept that environmental exposures can interact with genes during key developmental periods to trigger disease onset later in life, and that tissues are being reprogrammed to become abnormal later," says Shuk-mei Ho, PhD, senior author of the paper, chair of UC's Department of Environmental Health and the director of the Center for Environmental Genetics.

"This research is aimed at detecting early signs of asthma risk so that we can better prevent this chronic disease that affects as many as 25 percent of children in Northern Manhattan and elsewhere," adds Frederica Perera, DrPH, professor of environmental health sciences and director of the Columbia Center for Children's Environmental Health (CCCEH) at the Mailman School of Public Health and co- first author on the paper.

Using biological specimens from the CCCEH birth cohort of mothers and children living in Northern Manhattan and the South Bronx, UC scientists analyzed umbilical cord white blood cell samples from 56 children for epigenetic alterations related to prenatal PAH exposure. (The mothers' exposure to PAHs was monitored during pregnancy using backpack air monitors).

The researchers found a significant association between changes in ACSL3 methylation—a gene expressed in the lung—and maternal PAH exposure. ACSL3 also was associated with a parental report of asthma symptoms in the children prior to age 5.

With confirmation in further studies, researchers say changes in the ACSL3 gene could serve as a novel biomarker for early diagnosis of pollution-related asthma.

"This study provides a blueprint for the discovery of epigenetic biomarkers relevant to other investigations of exposure-disease relationships in birth cohorts," says Wan-yee Tang, PhD, a UC research scientist and a co-first author on the paper.

"Understanding early predictors of asthma is an important area of investigation," adds Rachel Miller, MD, director of the CCCEH asthma project and study co-author, "because they represent potential clinical targets for intervention."

Source: University of Cincinnati