PRENATAL EXPOSURE TO AIR POLLUTANTS: LINKS TO ATTENTION DEFICIT HYPERACTIVITY DISORDER

PHAs — short for polycyclic aromatic hydrocarbons — are bad actors: they’re toxic, ubiquitous pollutants that readily cross the placenta, causing damage to the fetal brain. Now, results from a new study show that PHA-induced fetal brain damage may lead to severe behavioral problems during early childhood, including aggression and attention deficit hyperactivity disorder (ADHD).

The deleterious effects of air pollution — greater risk of stroke, heart attacks and cognitive deterioration — are widely recognized. However, the new study assessed prenatal exposure and identified specific physical damage in the brain. The researchers used magnetic resonance imaging to measure the brains of 40 children from a cohort consisting of more than 600 mother-baby pairs.
The mothers were either Latina (Dominican) or African American nonsmoking women from minority communities in New York City, aged 18 to 35 years. During the third trimester of pregnancy, the women carried personal backpack monitors that measured exposure to eight common PAHs over 48 hours. Such exposure occurred by breathing contaminated air.

PHAs — common components of air pollution — are often found together in groups of two or more and persist in the environment for long periods of time. They’re generated by motor vehicles, waste incineration, wildfires and agricultural burning, and oil and coal burning for heat and electricity. Cooking (especially charred foods), tobacco smoke, and space heaters are indoor sources of PHAs. Low-income, urban, and minority communities are disproportionately exposed to these air pollutants.

The researchers had previously demonstrated that exposure of the pregnant women from the same cohort to airborne PAHs was associated with multiple neuro-developmental disturbances. Results form the new study indicate that such disturbances have a biological root in the altered architecture of the brain. Specifically, PHA exposure was linked to reductions of the white matter surface in later childhood. These reductions were confined almost exclusively to the left hemisphere of the brain, and involved almost its entire surface.
The researchers don’t know why the left side seemed to be affected more, but they suspect the compounds interfere with an early biochemical process that helps the fetal brain divide into slightly asymmetrical hemispheres. Results from the study also show that postnatal PAH exposure correlates with white matter surface measures in other regions of the brain, the dorsal prefrontal regions. Thus, the children involved in the study were exposed to “a double hit”, first as developing fetuses, and then at an early age. Indeed, Bradley Peterson, lead author of the study, told the Los Angeles Times: “It’s a double hit. They have the abnormality from prenatal life throughout the left hemisphere and then on top of that they have this bilateral frontal hit from exposures around age 5.”

In other words, **prenatal PAH exposure** disrupts the development of left hemisphere white matter, whereas **postnatal PAH exposure** contributes to additional disturbances in the development of white matter in dorsal prefrontal regions, which is associated with concentration, reasoning, judgment, and problem-solving ability. Peterson said in a press release: “Our findings raise important concerns about the effects of air pollutants on brain development in children, and the consequences of those brain effects on cognition and behavior. If confirmed, our findings have important public health implications, given the ubiquity of PAHs in air pollutants in the general population.”
The researchers recognize that the numbers of children involved in the study was limited. Thus, a much larger study is underway to confirm and extend these findings.

Peterson believes that “Health providers should educate prospective parents, especially early in pregnancy, about these risks and urge them to avoid, to the extent possible and for the health of their baby, exposure to smoke, exhaust, and other sources of PAH.”