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In Utero Exposure to Carbon Monoxide Increases Infants' Risk of Poor Lung Function

ATS 2018, San Diego, CA –Exposure to elevated levels of carbon monoxide (CO) in utero increases infants' risk of poor lung function at one month after birth, according to new research conducted as part of the Ghana Randomized Air Pollution and Health Study (GRAPHS). While household air pollution from solid fuel stoves has previously been associated with child mortality, this is the first study to investigate the association between household air pollution and lung development beginning in utero and to identify the most harmful times during pregnancy for exposure to this pollution. The study was presented at the 2018 American Thoracic Society International Conference.

Approximately 40 percent of the world's population burn solid fuels, such as coal and wood, to meet their cooking and heating needs. These fuels are typically used in combustion cookstoves that burn their fuel inefficiently, leading to the release of numerous airborne pollutants.

"Our research suggests that children, especially girls, born to mothers with increased household air pollution exposures during pregnancy have impaired lung function measurable at birth," said lead author Alison Lee, MD, MS, of the Icahn School of Medicine at Mount Sinai, New York, New York. "Exposures during the second and third trimesters appear to have the largest impact. These findings have implications for future respiratory health."

The GRAPHS study group recruited Ghanaian women during early pregnancy, and randomized them to one of three groups: two cookstove interventions and one control group. The control group used a three-stone fire, the traditional means of cooking in Ghana; intervention group one was given BioLite Stoves, improved combustion efficiency biomass stoves; and intervention group two was given LPG gas stoves, similar to those used in the U.S.

"Households were given the intervention stove, but in some cases preferentially or simultaneously used their three-stone fire," Dr. Lee said. "Because our analyses looked at the effect of in utero CO exposures and not the source, this was not a problem, and we were still able to gather the data we needed."

Four 72-hour CO measurements were taken for each study participant, and calculated to estimate weekly CO exposures. Four hundred of the women's infant children were then given lung function tests one month after their birth. The researchers applied statistical models to estimate the association between average lung function and in utero CO exposure. These models were adjusted statistically to rule out variables that may have affected results such as birth weight and gestational age at birth.

Dr. Lee and colleagues found a significant association between increased prenatal CO exposure and decreased lung function. Infants whose mothers were exposed to CO in the second and third trimester were at highest risk for reduced lung function when they were tested at the age of one month.

"Women are commonly the primary cooks and continue to cook while pregnant, so a child's exposure to household air pollution begins in utero," said Dr. Lee. "Importantly, we know that lung function development progresses rapidly over gestation and alterations in lung development secondary to toxic maternal exposures impair lung development with lasting effects. Lung function at birth has been shown to predict lung function through adulthood and increase risk for future respiratory symptoms and disease."

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Title: In Utero Household Air Pollution Exposure Is Associated with Reduced Pulmonary Function in Infants

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Rationale: Household air pollution (HAP) exposure secondary to the burning of solid fuels is an independent predictor of childhood mortality, largely due to childhood pneumonia. HAP may affect lung development *in utero* and increase susceptibility to future respiratory disease. No study has investigated the association between HAP and lung development beginning *in utero* nor identified critical windows of exposure.
Methods: The Ghana Randomized Air Pollution and Health Study (GRAPHS) recruited women during early pregnancy and randomized them to one of three groups (two cookstove interventions and control). Four, seventy-two hour carbon monoxide (CO) personal air pollution exposure measurements were performed during pregnancy and interpolated to estimate weekly prenatal CO exposure. A subset of study children (n=400) performed infant lung function testing per ATS protocol at age one month. The primary lung function outcome was the ratio of time to peak expiratory flow to expiratory time (TPEF:TE); tidal volume (TV, mL), respiratory rate (RR, breath/min), minute ventilation (MV, mL/min), and passive respiratory system compliance (Crs, mL/cm H2O) were also measured. Multivariable linear regression models estimated the association between average prenatal CO exposure and lung function. Distributed lag models (DLMs) estimated the time-varying association between lung function variables and weekly CO exposures. Models were adjusted for child sex, birthweight, gestational age (GA) at birth, weight, height and age at lung function test, socioeconomic status, and maternal education.
Results: Mothers mostly had no (46%) or <12 years (51%) education and were all never smokers. Infants were term (GA 39.7 weeks, IQR 39-40.7) and 30 days old (IQR 27-30.3) at lung function testing. Median prenatal CO exposure was 1.1ppm (0.6-1.9). Multivariable linear regression models demonstrated a significant association between increasing prenatal CO exposure and TPEF:TE (PE = -0.002, p=0.03), RR (PE = 0.29, p=0.01), and MV (PE = 7.47, p=0.04) per 1ppm increase in CO. DLMs identified a significant critical window of prenatal CO exposure and reduced TPEF:TE [Fig 1(12-23 weeks)], increased RR (13-29 weeks), and reduced Crs (18-24 weeks).
Conclusions: Our data demonstrate that children born to mothers exposed to increased CO during pregnancy, specifically during the pseudoglandular (5-17 weeks) and canalicular (17-26 weeks) phases of lung development, were at increased risk for reduced lung function at age one month. Understanding temporal effects of HAP on lung development may elucidate underlying mechanisms, critical for the development of preventative strategies.

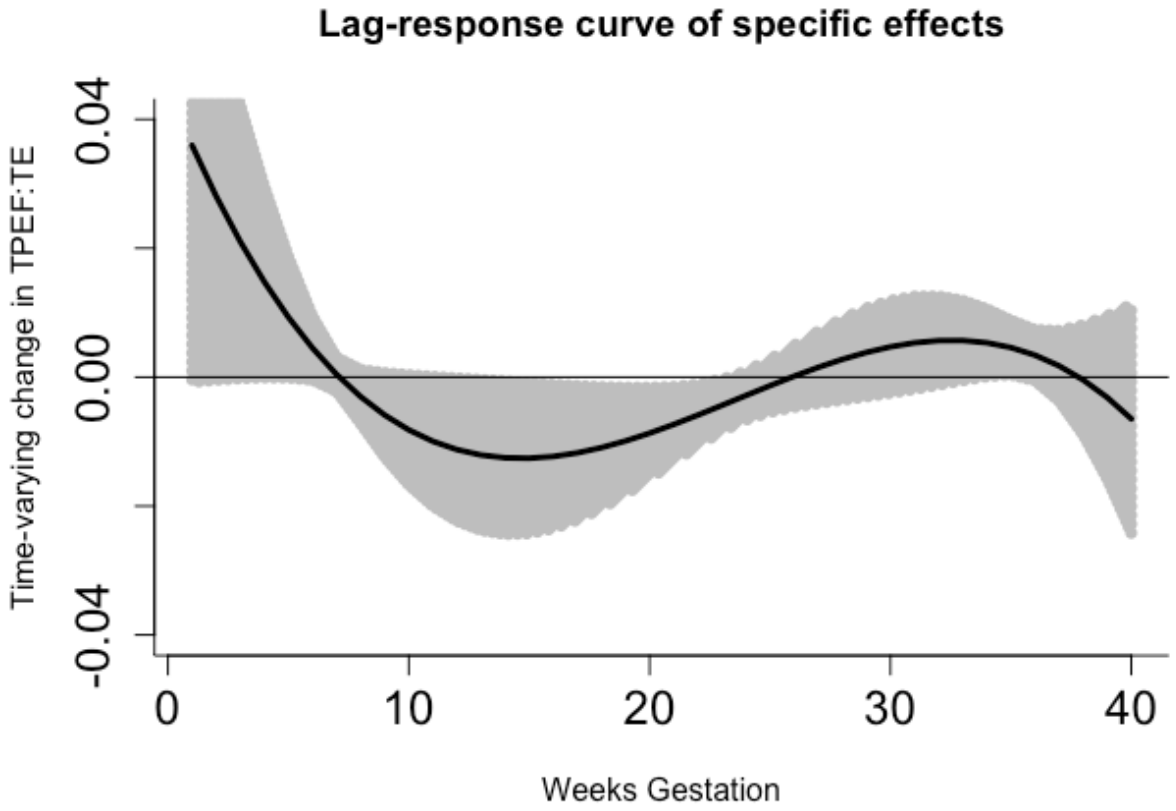

Figure 1. Association between weekly carbon monoxide (CO) exposure over gestation and infant lung function, ratio of time to peak expiratory flow to expiratory time (TPEF:TE), adjusting for child sex, birth weight, gestational age at delivery, weight, height, age, socioeconomic status, and maternal education. The Y-axis shows the change in TPEF:TE in relation to a 1ppm increase in prenatal CO exposure; the X-axis shows gestational age in weeks. The solid line shows the predicted parameter estimate and the gray shaded area demonstrates the 95% confidence interval. A sensitive window is identified when the estimated pointwise 95% confidence interval does not include zero.