EFFECTS OF PRENATAL AIR POLLUTION EXPOSURE ON CHILDHOOD BLOOD PRESSURE AND CAROTID INTIMA-MEDIA THICKNESS

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Background and Aims: Air pollution exposure is a risk factor for atherosclerosis in adults but whether this association exists at a younger age remains unclear. We hypothesized that prenatal and lifetime exposures to air pollutants would be associated with increased blood pressure and carotid intima-media thickness (CIMT) in children.

Methods: We investigated these hypotheses in a subset of 568 children from the Children's Health study on whom we measured blood pressure and right common carotid IMT and calculated prenatal and lifetime air pollution exposures. Average pollutant concentrations for each trimester and total lifetime exposure were computed using a weighted average of calendar month averages based on central site monitoring data and residential history. Caline dispersion model estimates of PM_{b5} and NO_v based on traffic data within 5 km of each residence were calculated. Linear regression models were used in all analyses.

Results: Prenatal exposures to NO2, PM25, and CO were associated with increased childhood systolic blood pressure (SBP) in univariate analyses. After adjustment for sex, age, ethnicity, BMI percentile, income, town, and parental history of high blood pressure and high cholesterol, only prenatal exposures to NO₂ (β= 0.09 mm Hg per 1ppb, p= 0.06) and PM₁₀ (β=0.05 mm Hg per 1 µg/m³, p=0.06) during the third trimester remained marginal predictors of childhood SBP. Second trimester exposure to elemental carbon (EC) was significantly associated with a 0.01 mm increased CIMT (p=0.04) per µg/m³ change in a smaller subset of children with EC data (n=403). However, this association was reduced by half after adjustment for covariates. None of the lfetime exposures and Caline-derived estimates were associated with childhood SBP or CIMT.

Conclusions: Prenatal exposure to air pollutants may have deleterious effects on childhood cardiovascular risk factors.