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Prenatal exposure to ambient air pollution and traffic and indicators of adiposity in early childhood: The Healthy Start study

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Abstract

Background: Prenatal exposure to ambient air pollution and traffic have been related to a lower birth weight and may be associated with greater adiposity in childhood. We aimed to examine associations of maternal exposure to ambient air pollution and traffic during pregnancy with indicators of adiposity in early childhood.

Methods: We included 738 participants of the Colorado-based Healthy Start study whose height, weight, waist circumference and/or fat mass were measured at age 4–6 years. We estimated residential exposure to ambient concentrations of fine particulate matter ($PM_{2.5}$) and ozone (O_3) averaged by trimester and throughout pregnancy via inverse distance-weighted interpolation of central site monitoring data. We assessed the distance to the nearest major roadway and traffic density in multiple buffers surrounding the participants' homes. Associations of prenatal exposure

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Author contributions

APS and LDB designed the research question. LDB performed the statistical analyses, and LDB and APS interpreted the results. LDB drafted the first version of the manuscript. All authors reviewed and critically revised the manuscript. All authors read and approved the final version of the manuscript for submission. LDB takes responsibility for the contents of the manuscript.

Competing Interests Statement

The authors declare that they have no actual or potential competing financial interests.

to air pollution and traffic with overweight, waist circumference, percent fat mass and fat mass index (FMI) were assessed by logistic and linear regression.

Results: Associations of exposure to $PM_{2.5}$ and O_3 at the residential address during pregnancy with percent fat mass and FMI at age 4–6 years were inconsistent across trimesters. For example, second trimester $PM_{2.5}$ was associated with a higher percent fat mass (adjusted difference 0.70% [95%CI 0.05, 1.35%] per interquartile range (IQR; 1.3μg/m^3) increase), while third trimester $PM_{2.5}$ was associated with a lower percent fat mass (adjusted difference −1.17% [95%CI −1.84, −0.50%] per IQR (1.3μg/m^3) increase). Residential proximity to a highway during pregnancy was associated with higher odds of being overweight at age 4–6 years. We observed no associations of prenatal exposure to $PM_{2.5}$ and O_3 with overweight and waist circumference.

Conclusions: We found limited evidence of associations of prenatal exposure to ambient $PM_{2.5}$ and O_3 with indicators of adiposity at age 4–6 years. Suggestive relationships between residential proximity to a highway during pregnancy and greater adiposity merit further investigation.

Introduction

The prenatal period is one of the critical periods for the development of childhood obesity (1, 2). Multiple studies have shown the potential for long lasting effects of the prenatal environment on the development of obesity and adverse fat distribution (1). Recent epidemiological studies suggest that prenatal exposure to environmental toxicants, including ambient air pollution, increase the risk of childhood obesity (3, 4). Higher exposure to ambient air pollution during pregnancy has been associated with a lower birth weight (5–9). Accelerated postnatal growth is a common compensatory mechanism for low birth weight and has been related to higher adiposity and an increased risk of obesity in childhood (10–12). Potential biological mechanisms that explain associations of prenatal exposure to air pollution with lower birth weight (and potentially subsequent higher adiposity in childhood) include alterations in endothelial function, systemic oxidative stress and inflammation (5, 13).

Findings of studies examining associations of prenatal exposure to ambient air pollution or traffic with childhood body composition or growth trajectories have been inconsistent (14–24).

For example, a study among 1418 participants of a Boston-area birth cohort did not find associations between third trimester fine particulate matter ($PM_{2.5}$) exposure and body mass index (BMI) z-scores in early (median age 3.3 years) and mid- (median age 7.7 years) childhood (14). In contrast, third trimester $PM_{2.5}$ exposure was related to an increased risk of overweight or obesity between ages two and nine years in 1446 children from another birth cohort in Boston (15). Three studies from the United States have examined relationships between prenatal exposure to ambient air pollution and childhood growth trajectories. One study found that exposure to $PM_{2.5}$ across the full pregnancy was related to weight trajectories from birth to age six years in 4797 children (23), while the two other studies did not find associations of prenatal exposure to air pollution with childhood BMI trajectories (21, 22).

The current evidence base contains important gaps. Most prior studies have only included body weight or BMI (15, 17, 18, 20–23), which is a crude proxy for body composition and does not differentiate between fat and fat-free mass. BMI has a low sensitivity to detect excess adiposity in children and fails to identify more than 25% of children with excess body fat percentage (25). Furthermore, excess fat located in the upper abdominal region appears to be more strongly related to adverse cardiometabolic outcomes than fat located in other areas in children and adults (26–28). It is therefore important to examine associations of prenatal exposure to air pollution and traffic with indicators of adiposity other than BMI, such as waist circumference (a measure of central obesity), percent fat mass and fat mass index (FMI), as was done in the present study.

Additionally, most previous studies have been conducted in areas with relatively high concentrations of ambient air pollutants, such as Hong Kong and the northeast United States. No studies have been performed in the Denver metropolitan area, where ozone (O_3) levels exceed national regulatory standards (29), but particulate matter concentrations are relatively low compared to other large US cites. The chemical composition of $PM_{2.5}$ varies regionally, indicating that studies in diverse geographic areas are needed (30).

The aim of this study is therefore to examine the associations of prenatal exposure to ambient air pollution and traffic with indicators of adiposity at age 4–6 years in participants of the Healthy Start study, an ongoing longitudinal birth cohort based in Denver, Colorado.

Subjects and Methods

Study design and population

Pregnant women were recruited from outpatient obstetrics clinics at the University of Colorado Hospital from 2009 to 2014. Women were eligible to participate in the Healthy Start study if they were ≥16 years of age, were <24 weeks gestation at the time of enrolment and expecting a singleton birth, had no history of stillbirth or extremely preterm birth (<25 weeks of gestation) and had no preexisting diabetes, asthma, cancer or psychiatric illness. A total of 1410 pregnant women were enrolled, of which 11 withdrew or were lost to follow-up prior to delivery. Participants were invited to complete in-person study visits in early pregnancy (median 17 weeks of gestation), mid-to late pregnancy (median 27 weeks of gestation), at delivery (median one day after birth), in infancy (median five months) and early childhood (age 4–6 years; median five years). The study protocol has been approved by the Colorado Multiple Institutional Review Board and all participating women provided written informed consent. In this study, we included 738 mother-child pairs who had data on prenatal exposure to ambient air pollution and traffic and data on at least one indicator of adiposity at age 4–6 years (Figure S1).

Exposure assessment

We estimated exposure to ambient $PM_{2.5}$ and O_3 and traffic at the maternal residential addresses during pregnancy. Participants reported their home address via questionnaire at the time of enrollment in the Healthy Start study. A detailed description of the exposure assessment has been published previously (31).

We used stationary monitors in the United States Environmental Protection Agency (US EPA) Air Quality System (AQS) to estimate outdoor concentrations of $PM_{2.5}$ and O_3. Ten stationary monitors measuring 24-h average $PM_{2.5}$ concentrations and 19 stationary monitors measuring hourly O_3 concentrations during the study period and located within 50km of at least one residential address were identified. For each monitor, we averaged daily $PM_{2.5}$ concentrations over each trimester and over the full pregnancy. Hourly O_3 concentrations were averaged over each 8-h interval during a 24-h period for each monitor and daily 8-h maximum values were averaged over each trimester and across the full pregnancy. We used all monitors located within 50km of the residence and for which at least 75% of expected concentrations were non-missing to determine inverse-distance weighted average $PM_{2.5}$ and O_3 exposure for each participant. Trimester and full pregnancy averages from each monitor were weighted according to the following formula: 1/distance-squared.

We included multiple measures of traffic exposure: 1) the distance to the nearest highway, 2) the distance to the nearest non-highway major roadway and 3) traffic density on major roads (highways or other major and minor arterial and connector roads) within buffers of 150m, 250m, 500m and 1000m surrounding the residential addresses. We used data from the Colorado Department of Transportation to assess the Euclidean distance from the residences to the nearest major roadway, calculated separately for interstates and limited access highways and for other major and minor arterial and connector roads. We additionally estimated traffic density on major roads in buffers of 150m, 250m, 500m and 1000m around the participants' homes. For each major roadway segment in a buffer, the contribution to traffic density was calculated as the annual average daily vehicle count, obtained from the Colorado Department of Transportation, multiplied by the length of the segment within the buffer. The total traffic density in a buffer (in vehicles/day x kilometers of roadway) was calculated as the sum of the contributions from each intersecting road segment. Since the distance to the nearest major roadway and traffic density variables are based on annual average data, we were only able to assess exposure to traffic for the year of the child's birth.

Indicators of adiposity

Children's height, weight, waist circumference and percent fat mass were measured by trained research staff during the early childhood study visit (i.e., at age 4–6 years). Height (to the nearest 0.1cm), weight (to the nearest 0.1kg) and waist circumference were measured without shoes and heavy clothes. We calculated BMI (weight (kg)/height $(m)^2$) from the weight and height measurements and we defined overweight (including obesity) as BMI at or above the 85^{th} percentile for children of the same age and sex, using the Centers for Disease Control and Prevention (CDC) growth curves (32). Waist circumference was measured twice. If the two measurements differed by >1 cm, a third measurement was obtained. The average of the two closest measurements was used in the analyses. Percent fat mass was assessed with the BOD POD device with Pediatric Option (COSMED, Rome, Italy). The BOD POD uses air displacement plethysmography and is accurate, precise and reliable in estimating percent fat mass in children aged 2–6 years (33). Trained staff took two measurements, with a third measurement obtained when the first two measurements differed by >2%. We used the average of the two closest measurements in the analyses. Persons will differ in percent fat mass either if they have identical fat-free mass but different

fat mass or if they have identical fat mass but different fat-free mass. To resolve this issue, fat mass can be normalized for height (34, 35). We therefore additionally calculated FMI using the following formula: fat mass/height^2.

Covariates

Pregnant women reported their age, race/ethnicity, level of education and number of previous pregnancies via questionnaire at the early pregnancy study visit. We defined parity as the number of previous live births and created a binary variable: no previous live births (primiparous) and ≥1 previous live birth (multiparous). Maternal smoking during pregnancy was self-reported in early pregnancy, mid- to late pregnancy and shortly after delivery. We dichotomized maternal smoking during pregnancy as mothers who reported smoking during the past three months at any of the three study visits and those who did not report recent smoking at any visit. Maternal height was measured during the early pregnancy visit and pre-pregnancy weight was obtained from medical records or self-reports, if medical records were unavailable. We defined pre-pregnancy BMI categories as follows: normal weight/underweight (BMI <25 kg/m^2), overweight (BMI 25–30 kg/m^2) and obesity (BMI ≥30 kg/m^2). Since only 19 women (2.6%) were underweight (BMI <18.5 kg/m^2) prior to their pregnancy, we included women whose BMI falls within the underweight or healthy weight range in one category. Finally, we used the median income in the Census tract of the mother's residence, obtained from the 2012–2016 American Community Survey, as an indicator of area-level socioeconomic status (SES).

Statistical methods

First, we visually examined whether the relationships between the ambient air pollutants and indicators of adiposity (overweight, waist circumference, percent fat mass and FMI) were linear by generalized additive models with integrated smoothness estimation and a logit or identity link (R package 'mgcv', GAM function) (36). The distance to the nearest major roadway and traffic density measures were highly skewed. We therefore categorized these variables into quartiles to reduce the influence of outliers. For traffic density in a buffer of 150m around the residential address, we combined quartiles 1 and 2 due to the large number of zero values. We then examined associations of prenatal exposure to ambient air pollution and traffic with indicators of adiposity at age 4–6 years by multiple logistic or linear regression analyses.

Covariates were selected via the construction of a directed acyclic graph (Figure S2). We specified three models with increasing covariate adjustment. Model 1 was the unadjusted model. Model 2 was adjusted for child's sex, child's age (years), maternal age at delivery (years), pre-pregnancy BMI (normal weight/underweight, overweight, obesity), parity (any vs. no previous live births), race/ethnicity (non-Hispanic white, Hispanic, non-Hispanic black, other), maternal level of education (less than 12th grade, some college, four years of college, graduate degree), maternal smoking during pregnancy (yes, no), season of birth (spring, summer, fall, winter) and median income in the Census tract (continuous). Model 3 (only for associations with $PM_{2.5}$ and O_3) was additionally adjusted for the other air pollutant in the same trimester or across the full pregnancy. We evaluated effect modification

by child's sex by including a product interaction term between sex and the ambient air pollutants in adjusted models.

Finally, we performed several sensitivity analyses. We examined associations of prenatal exposure to ambient air pollution and traffic with obesity (yes, no) and sex- and age-specific BMI z-scores (continuous) at age 4–6 years, using the CDC growth curves. We defined obesity as BMI at or above the 95th percentile for children of the same age and sex (32). Additionally, we repeated the analyses in the subgroup of children whose mothers have not smoked during pregnancy (n = 692).

Analyses were conducted in SAS version 9.4 (SAS Institute Inc., Cary, NC, USA), except the analyses of the linearity of the associations, which we conducted in R version 3.6.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

The median age of the children during the early childhood study visit was 4.6 years (range 4.0–8.3 years, Table 1) and 93.2% of the children were aged 4 or 5 years. Participants were racially and ethnically diverse, with 55.0% non-Hispanic white, 24.3% Hispanic, 15.5% non-Hispanic black and 5.3% of participants from all other races and ethnic groups combined. Our study population comprised 104 (14.2%) children with overweight or obesity. The median (interquartile range; IQR) waist circumference was 50.9 (4.5) cm and the median (IQR) percent fat mass was 19.7 (8.5)%. Children aged ≥5 years had a higher waist circumference (median [IQR] 52.9 [5.1] cm) and lower percent fat mass (median [IQR] 15.8 [10.0]) than children aged <5 years (median [IQR] 50.5 [4.4] cm and 20.5 [7.7]). Our study participants were generally representative of the baseline Healthy Start population, but mothers in our study population had slightly higher educational attainment (Table S1).

Pearson correlations between BMI and waist circumference, percent fat mass and FMI ranged from 0.44 to 0.79 (Table 2). Waist circumference was moderately positively correlated with percent fat mass (r_p=0.33) and FMI (r_p=0.53). Higher maternal level of education and higher median income in the Census tract of the mother's residence during pregnancy were related to a lower prevalence of overweight at age 4–6 years (Table S2). We did, however, not observe differences in waist circumference, percent fat mass, FMI or ambient $PM_{2.5}$ and 8-hr maximum O_3 concentrations throughout pregnancy by maternal level of education or quartiles of median income in the Census tract.

The median (IQR) ambient $PM_{2.5}$ and 8-hr maximum O_3 concentrations throughout pregnancy were 7.5 (0.9) μg/m^3 and 44.0 (5.8) ppb, respectively (Table 3). The median (IQR) distance from the residential address during pregnancy to the nearest highway was 866 (1285) m. Spearman correlations between average $PM_{2.5}$ and O_3 concentrations in the same trimesters were negative (r_s from −0.32 to −0.23, Figure S3), reflecting the different seasonal patterns of the two air pollutants. $PM_{2.5}$ and O_3 concentrations by trimester and across the full pregnancy were weakly correlated with the distance to the nearest roadway and traffic density measures (r_s from −0.16 to 0.15 for $PM_{2.5}$; r_s from −0.16 to 0.17 for O_3).

The associations between prenatal exposure to air pollution and indicators of adiposity at age 4–6 years were linear or almost linear, so we included the air pollutants as continuous exposures in our analyses and expressed the associations per IQR increase in exposure. Since we did not observe interactions between child's sex and exposure to ambient air pollution at the residential address during pregnancy (all p-values for interaction ≥0.35), we did not present results stratified by sex. In unadjusted models, first trimester average 8-h maximum O_3 was associated with lower odds of being overweight at age 4–6 years (odds ratio (OR) 0.65 [95% confidence interval (CI) 0.44, 0.97] per IQR increase), while third trimester O_3 was related to higher odds of being overweight (OR 1.62 [95%CI 1.09, 2.42] per IQR increase) (Table S3). However, we observed no relationships between exposure to $PM_{2.5}$ and O_3 at the residential address during pregnancy and overweight and waist circumference at age 4–6 years after covariate adjustment (Table S4). Associations changed minimally after additional adjustment for the other air pollutant in the same trimester or across the full pregnancy (Table 4).

Associations of $PM_{2.5}$ and O_3 with percent fat mass and FMI were inconsistent across trimesters (Table 4). Second trimester $PM_{2.5}$ was associated with a higher percent fat mass (adjusted difference 0.70% [95%CI 0.05, 1.35%] per IQR increase) and FMI (adjusted difference 0.13 [95%CI 0.01, 0.25] per IQR increase). In contrast, third trimester $PM_{2.5}$ was associated with a lower percent fat mass (adjusted difference −1.17% [95%CI −1.84, −0.50%] per IQR increase) and FMI (adjusted difference −0.18 [95%CI −0.31, −0.06] per IQR increase). First trimester average 8-h maximum O_3 was related to a lower percent fat mass at age 4–6 years, while third trimester O_3 was related to a higher percent fat mass. We found no associations of exposure to $PM_{2.5}$ and O_3 averaged across the full pregnancy with percent fat mass or FMI at age 4–6 years.

Residential proximity to a highway was associated with higher odds of being overweight and a (non-statistically significant) higher waist circumference, percent fat mass and FMI at age 4–6 years (Tables 5 and S5), although these relationships were not strictly linear. For example, children whose mothers lived <404m from a highway during pregnancy (i.e., the highest exposure) had higher odds of being overweight (adjusted OR 2.26 [95%CI 1.11, 4.62]) than children whose mothers lived >1689m from a highway (i.e., the lowest exposure). Traffic density in multiple buffers surrounding the residential addresses during pregnancy was not associated with indicators of adiposity at age 4–6 years.

We did not observe associations of prenatal exposure to $PM_{2.5}$ and O_3 with obesity age 4–6 years (Table S6). Second trimester $PM_{2.5}$ was associated with a higher BMI z-score (adjusted difference 0.11 [95%CI 0.00, 0.21] per IQR increase). Children whose mothers lived closer to a highway during pregnancy had a higher BMI z-score compared to those who lived further from a highway (Table S7), which is consistent with the results from the main analyses. We also found associations of residential proximity to a highway during pregnancy with higher odds of obesity at age 4–6 years (Table S7). However, confidence intervals were very wide, because of the small number of children with obesity. Finally, excluding children whose mothers have smoked during pregnancy did not substantially influence the results (Tables S8 and S9). However, we observed an association of O_3

averaged across the full pregnancy with higher odds of being overweight at age 4–6 years in this subgroup (adjusted OR 1.85 [95%CI 1.06, 3.22] per IQR increase).

Discussion

In this racially and ethnically diverse population of mother-child pairs residing in Denver, Colorado, we found limited evidence of relationships between prenatal exposure to ambient air pollution and traffic and indicators of adiposity at age 4–6 years. We observed no associations of ambient concentrations of $PM_{2.5}$ and O_3 at the residential address during pregnancy with overweight and waist circumference, while associations with percent fat mass and FMI were inconsistent across trimesters. Children whose mothers lived closer to a highway during pregnancy had higher odds of being overweight and a (non-statistically significant) higher waist circumference, percent fat mass and FMI at age 4–6 years.

We found associations of prenatal exposure to $PM_{2.5}$ and O_3 with percent fat mass and FMI at age 4–6 years, which were inconsistent across trimesters. For example, first trimester average 8-h maximum O_3 was associated with a lower percent fat mass at age 4–6 years, while third trimester O_3 was associated with a higher percent fat mass. Ambient concentrations of $PM_{2.5}$ and O_3 at the residential address during pregnancy were, however, not related to BMI z-scores, overweight or obesity at age 4–6 years. BMI is a crude proxy for body composition and has a low sensitivity to detect excess adiposity in children (25), which may explain why we were not able to detect associations with BMI or overweight in early childhood.

It is not apparent why associations of $PM_{2.5}$ and O_3 at the residential address during pregnancy with percent fat mass and FMI were inconsistent across trimesters. Previous epidemiological studies have also shown that relationships between exposure to ambient air pollution and birth outcomes and postnatal growth may differ across various stages of gestation (37, 38). This suggests that there may be specific windows of pregnancy during which adiposity development is more sensitive to air pollution exposure. The majority of fetal growth occurs in late pregnancy (39), which may explain why we observed associations of third trimester $PM_{2.5}$ and O_3 with percent fat mass and FMI at age 4–6 years. This does, however, not explain why we found both positive and negative associations of $PM_{2.5}$ and O_3 in different trimesters.

To the best of our knowledge, this is the first study that has assessed relationships between maternal exposure to ambient O_3 during pregnancy and indicators of adiposity in childhood. A previous study in the Healthy Start cohort has examined associations of prenatal exposure to ambient air pollution and traffic with infant weight and adiposity (31). That study observed that third trimester average 8-h maximum O_3 was associated with greater fat mass and adiposity (defined as percent fat mass) at age five months and a greater rate of change in fat mass and adiposity from birth to age five months, which is consistent with the results from the present study (31). Additional studies are needed that examine relationships between prenatal exposure to ambient O_3 and indicators of adiposity in childhood.

We observed a strong seasonal pattern of ambient O_3 concentrations. Exposure to O_3 at the residential address during pregnancy was considerably higher in summer than in winter. Previous studies have shown that season of birth is related to birth weight (40, 41). In our study, season of birth was associated with indicators of adiposity at age 4–6 years. For example, children born in summer had a higher waist circumference, percent fat mass and FMI at age 4–6 years than children born in winter. We have therefore adjusted our analyses for season of birth as a four-level categorical variable. It is, however, possible that our observed associations of prenatal exposure to O_3 with percent fat mass partly reflect seasonal differences in indicators of adiposity (for reasons other than air pollution, such as ambient temperature and vitamin D status). In other words: there may be residual confounding due to other seasonal patterns. Because of the limited sample size in each stratum, we did not stratify our analyses by season of birth. Moreover, stratifying by season of birth could lead to over-adjustment, resulting in limited O_3 exposure variation in each stratum. Future studies are needed to identify the underlying pathways through which season of birth may be associated with birth weight and indicators of adiposity in childhood, including seasonal differences in ambient air pollution concentrations.

Findings of previous studies examining associations of maternal exposure to ambient $PM_{2.5}$ during pregnancy with indicators of adiposity in childhood have been inconsistent. In contrast to the results from our study, Mao et al. found associations of prenatal exposure to ambient $PM_{2.5}$ with an increased risk of overweight or obesity between ages two and nine years in 1446 mother-child pairs from Boston (15). These associations were consistent across all exposure periods examined, i.e., preconception (defined as the 90 days before pregnancy), the first, second and third trimesters and full pregnancy (15). Another study among 239 children aged three to five years from Boston observed that prenatal $PM_{2.5}$ exposure was related to higher BMI z-scores and fat mass in boys and increased waist-to-hip ratio in girls (16). No associations were found between maternal exposure to $PM_{2.5}$ during pregnancy and body composition in early (median age 3.3 years) and mid- (median age 7.7 years) childhood in 1418 participants of another Boston-area birth cohort (14). Finally, one study found that prenatal exposure to ambient air pollution was associated with impaired growth in early childhood (24). This study by Fossati et al. showed that higher $PM_{2.5}$ exposure in the first trimester of pregnancy was associated with decreased z-scores of weight and BMI at age four years among 1724 mother-child pairs in Spain (24). The discrepancy between the findings from our study and findings from previous studies may be explained by differences in the chemical composition of $PM_{2.5}$ across regions or by differences in study design and the age groups and outcomes studied. Alternatively, our relatively small sample size and the low concentrations and low variability of $PM_{2.5}$ in our study area may have prevented us from finding associations with indicators of adiposity at age 4–6 years.

We observed associations of residential proximity to a highway during pregnancy with higher odds of being overweight and a (non-statistically significant) higher waist circumference, percent fat mass and FMI at age 4–6 years. In other words: maternal residence closer to the nearest highway was positively associated with the different indicators of adiposity at age 4–6 years. However, not all relationships reached statistical significance, which is likely due to lower statistical power of our analyses with quartiles of exposure. Associations of residential proximity to a highway during pregnancy were

non-linear, suggesting that distance to the nearest highway may only be associated with adiposity at age 4–6 years within certain exposure ranges. Alternatively, these non-linear associations could be due to spatial or other unmeasured confounders not correlated equally with the exposure across the quartiles.

Only two previous studies conducted within Project Viva, a Boston-area pre-birth cohort, have assessed relationships between exposure to traffic during pregnancy and childhood body composition or growth trajectories (14, 22). In line with the results from our study, Fleisch et al. observed that children whose mothers lived close to a major roadway at the time of delivery had a higher BMI z-score and waist circumference in early childhood and greater fat mass in mid-childhood (14). The other study found that residential proximity to a major roadway during pregnancy was associated with greater gains in BMI from birth to age ten years, but effect sizes were small and confidence intervals crossed the null (22).

The correlations between the distance to the nearest highway and ambient concentrations of $PM_{2.5}$ and O_3 in our study were low (Figure S3). This could suggest that the distance from the mother's residence to the nearest highway may be a better approximation of residential exposure to traffic-related air pollution than inverse-distance-weighted average $PM_{2.5}$ and O_3 concentrations. Alternatively, neighborhood features distinct from $PM_{2.5}$ and O_3 concentrations (which we have not included in our analyses) may have driven our observed associations between residential proximity to a highway and greater adiposity at age 4–6 years. For example, road traffic noise, ambient concentrations of nitrogen oxides (NO_x) and lower neighborhood walkability may be related to adiposity in children (42–44). Finally, our observed associations between residential proximity to a highway during pregnancy and greater adiposity at age 4–6 years could be explained by sociodemographic rather than environmental factors. Previous research suggests that socioeconomic and racial/ethnic disparities exist in the United States with respect to lower-income and minority populations living near high traffic and road density areas (45). We included maternal level of education in our analyses as an indicator of individual SES, but we lacked complete information on other possible SES proxy variables, such as household income and parental occupation. Since we used this imperfect indicator of individual-level SES for model adjustment, residual confounding could have biased our findings.

Our study has several strengths. This is the first study that has examined associations of maternal exposure to air pollution and traffic during pregnancy with indicators of adiposity in early childhood in the Mountain West region of the United States. We included multiple measures of adiposity that have been measured by trained research staff, i.e., BMI, waist circumference, percent fat mass and FMI. Moreover, we used a racially and ethnically diverse population with detailed information on maternal and child characteristics from direct measurements, medical records and questionnaires. In addition to individual characteristics, we adjusted our models for area-level SES by including the median income in the Census tract. Several studies in the United States have shown the importance of accounting for area-level SES when examining the health effects of air pollution, since a lower area-level SES has been associated with both increased susceptibility to poor health and higher ambient air pollution concentrations (46).

Our study also has some limitations. We estimated residential exposure to ambient air pollution via inverse-distance weighted interpolation of stationary monitoring data. Therefore, we were unable to capture finer scale variability in air pollution concentrations due to residential proximity to major roads and other sources. Additionally, the relatively sparse monitoring network in the Denver metropolitan area may have led to misclassification of ambient air pollution exposure, which may differ for different air pollutants and seasons. A previous analysis in a subset of participants of the Healthy Start cohort has shown a moderate to high agreement among the three closest monitors, suggesting that most of the variability in $PM_{2.5}$ and O_3 concentrations in our study is due to seasonal (temporal) changes in air pollution concentrations (31). We therefore expect that misclassification of exposure due to the interpolation procedure is a relatively minor source of error.

No data on maternal time-activity patterns during pregnancy were available in this study and exposure estimates were based on residential addresses only. We were not able to take into account indoor air pollution levels and exposure to ambient air pollution and traffic at other locations (e.g., at work and during commuting). A study in Canada has shown that pregnant women tend to spend more time at home during the latter stages of pregnancy, independent of family income, employment status and parity (47). This suggests that the differences between ambient air pollution concentrations at the home addresses and personal air pollution exposure may be lower in the third as compared to the first and second trimesters of pregnancy. This would, however, not explain why we observed both positive and negative associations of ambient concentrations of $PM_{2.5}$ and O_3 in different trimesters with percent fat mass and FMI at age 4–6 years.

We assigned exposures to the first reported maternal residential address during pregnancy, which was collected via questionnaire at the time of enrollment in the Healthy Start study. We did not have information on the timing of changes in residence during pregnancy. Two studies in New York and Texas found that the likelihood of misclassification of air pollution exposure due to residential mobility during pregnancy is small (48, 49). We do not know whether this also applies to participants of the Healthy Start study, but the evidence from these previous studies suggests that the effect of residential mobility during pregnancy is limited.

Furthermore, we were only able to include 738 mother-child pairs in this study (53.4% of the full potentially eligible Healthy Start study population). This may have led to selection bias, which represents a threat to the internal validity of estimates derived from cohort studies. Our study participants were, however, generally representative of the baseline study population in measured characteristics (Table S1). We therefore assume that the associations of prenatal exposure to ambient air pollution and traffic with indicators of adiposity at age 4–6 years would not be different in the baseline Healthy Start study population.

Finally, we have included the median income in the Census tract as an indicator of the social and physical environment, which may affect health independent of individual-level SES (50). It is possible that the inclusion of area-level SES may have introduced bias due to spatial autocorrelation. However, adjustment for median income in the Census tract did not meaningfully change our results, indicating that any bias introduced may be negligible.

In conclusion, we found limited evidence of relationships between exposure to $PM_{2.5}$ and O_3 at the residential address during pregnancy and indicators of adiposity at age 4–6 years. Suggestive associations of residential proximity to a highway during pregnancy with greater adiposity merit further investigation. Continued follow-up of the Healthy Start cohort will allow us to study associations of exposure to air pollution and traffic during pregnancy and childhood with indicators of adiposity and related metabolic conditions in middle childhood and adolescence.

Supplementary Material

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Funding Statement

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Table 1.

Characteristics of the study population (n = 738).

Characteristic	n (%) or median (25th – 75th percentiles)
Overweight (including obesity) [a]	104 (14.2)
Obesity [b]	46 (6.3)
Waist circumference (cm)	50.9 (49.0 – 53.5)
Percent fat mass [c]	19.7 (15.3 – 23.8)
FMI (kg/m^2) [c]	3.0 (2.3 – 3.7)
Boys	389 (52.7)
Age (years)	4.6 (4.4 – 4.9)
Season of birth	
Spring	179 (24.3)
Summer	224 (30.4)
Fall	169 (22.9)
Winter	166 (22.5)
Maternal age at delivery (years)	29.0 (23.0 – 33.0)
Pre-pregnancy BMI (kg/m^2)	
Normal weight/underweight (<25.0)	382 (51.8)
Overweight (25.0 – 30.0)	203 (27.5)
Obesity (≥30.0)	153 (20.7)
Parity	
Primiparous	353 (47.8)
Multiparous	385 (52.2)
Race/ethnicity	
Non-Hispanic white	406 (55.0)
Hispanic	179 (24.3)
Non-Hispanic black	114 (15.5)
Other	39 (5.3)
Maternal level of education	
Less than 12th grade or high school degree	219 (29.7)
Some college	166 (22.5)
Four years of college	168 (22.8)
Graduate degree	185 (25.1)
Maternal smoking during pregnancy (yes)	46 (6.2)

Characteristic	n (%) or median (25th – 75th percentiles)
Median income in the Census tract ($)	56749.0 (43182.0 – 83085.0)

Abbreviations: FMI = fat mass index; BMI = body mass index.

[a] Overweight (including obesity) was defined as BMI ≥85th percentile for children of the same age and sex, using the CDC growth curves.

[b] Obesity was defined as BMI ≥95th percentile for children of the same age and sex, using the CDC growth curves.

[c] For 86 children (11.7%), percent fat mass has not been determined.

Table 2.

Pearson correlations between BMI, waist circumference, percent fat mass and FMI.

	BMI	Waist circumference	Percent fat mass	FMI
BMI		0.79	0.44	0.67
Waist circumference			0.33	0.53
Percent fat mass				0.95
FMI				

Abbreviations: BMI = body mass index; FMI = fat mass index.

Sample sizes differ due to missing anthropometry data: BMI, n = 733; waist circumference, n = 738; percent fat mass and FMI, n = 652.

Table 3.

Exposure to ambient $PM_{2.5}$ and O_3 and traffic at the residential address during pregnancy.

Exposure	n	median (25^{th} – 75^{th} percentiles)
Average $PM_{2.5}$ ($\mu g/m^3$) [a]		
Trimester 1	671	7.5 (6.9 – 8.2)
Trimester 2	663	7.4 (6.8 – 8.0)
Trimester 3	695	7.3 (6.8 – 8.1)
Full pregnancy	711	7.5 (7.0 – 7.9)
Average 8-hr max O_3 (ppb) [a]		
Trimester 1	718	41.8 (32.7 – 53.2)
Trimester 2	718	42.7 (34.1 – 52.2)
Trimester 3	717	46.2 (35.3 – 54.0)
Full pregnancy	718	44.0 (41.0 – 46.8)
Distance to nearest highway (m)	738	866 (404 – 1689)
Distance to nearest non-highway major roadway (m)	738	153 (68 – 308)
Traffic density on major roads (vehicles*km/day)		
150m buffer	725	437 (0 – 2846)
250m buffer	725	3276 (0 – 9494)
500m buffer	725	23059 (9478 – 42551)
1000m buffer	725	111625 (64368 – 160575)

Abbreviations: $PM_{2.5}$ = fine particulate matter; O_3 = ozone.

[a]Sample sizes differ due to missing monitoring data. Averages from a given monitor were included if at least 75% of the daily air pollution concentrations throughout the specified pregnancy period were non-missing.

Table 4.

Associations of exposure to $PM_{2.5}$ and O_3 at the residential address during pregnancy with indicators of adiposity at age 4–6 years - model 3.

Exposure (increment)	Overweight OR (95% CI)	Waist circumference β (95% CI)	Percent fat mass β (95% CI)	FMI β (95% CI)
Trimester 1 average $PM_{2.5}$ (1.3 μg/m^3)	0.95 (0.69, 1.30)	−0.08 (−0.48, 0.32)	−0.28 (−0.94, 0.38)	−0.06 (−0.18, 0.06)
Trimester 2 average $PM_{2.5}$ (1.3 μg/m^3)	1.06 (0.78, 1.43)	0.32 (−0.07, 0.72)	**0.70 (0.05, 1.35)**	**0.13 (0.01, 0.25)**
Trimester 3 average $PM_{2.5}$ (1.3 μg/m^3)	1.03 (0.74, 1.43)	−0.25 (−0.66, 0.16)	**−1.17 (−1.84, −0.50)**	**−0.18 (−0.31, −0.06)**
Full pregnancy average $PM_{2.5}$ (0.9 μg/m^3)	1.10 (0.77, 1.57)	−0.05 (−0.51, 0.40)	−0.35 (−1.12, 0.42)	−0.05 (−0.19, 0.09)
Trimester 1 average 8-hr max O_3 (20.4 ppb)	0.95 (0.37, 2.43)	−0.14 (−1.27, 1.00)	**−2.49 (−4.39, −0.58)**	−0.35 (−0.70, 0.01)
Trimester 2 average 8-hr max O_3 (18.0 ppb)	1.19 (0.54, 2.62)	−0.04 (−1.03, 0.96)	0.04 (−1.59, 1.67)	0.03 (−0.28, 0.33)
Trimester 3 average 8-hr max O_3 (18.7 ppb)	1.12 (0.50, 2.51)	−0.98 (−2.03, 0.07)	**1.89 (0.15, 3.63)**	0.25 (−0.08, 0.57)
Full pregnancy average 8-hr max O_3 (5.8 ppb)	1.32 (0.79, 2.20)	−0.37 (−1.01, 0.27)	−0.45 (−1.54, 0.64)	−0.06 (−0.26, 0.14)

Abbreviations: $PM_{2.5}$ = fine particulate matter; O_3 = ozone; FMI = fat mass index. Statistically significant results are highlighted in bold ($p < 0.05$).

Associations are shown for an interquartile range increase in exposure. Associations are adjusted for child's sex, child's age, maternal age at delivery, pre-pregnancy BMI, parity, race/ethnicity, maternal level of education, maternal smoking during pregnancy, season of birth and median income in the Census tract. Associations with $PM_{2.5}$ are additionally adjusted for the average 8-hour maximum O_3 concentration during the specified pregnancy period. Associations with O_3 are additionally adjusted for the average daily $PM_{2.5}$ concentration during the specified pregnancy period.

Table 5.

Adjusted associations of exposure to traffic at the residential address during pregnancy with indicators of adiposity at age 4–6 years.

Exposure	Overweight	Waist circumference	Percent fat mass	FMI
	OR (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)
Distance to highway (m)				
Quartile 1 (<404.24)	**2.26 (1.11, 4.62)**	**0.87 (0.02, 1.73)**	0.39 (−1.04, 1.83)	0.22 (−0.04, 0.49)
Quartile 2 (404.24 – 866.43)	1.30 (0.61, 2.77)	0.69 (−0.16, 1.54)	0.55 (−0.88, 1.98)	0.21 (−0.06, 0.48)
Quartile 3 (866.43 – 1688.56)	**2.41 (1.20, 4.81)**	0.78 (−0.03, 1.60)	0.42 (−0.97, 1.81)	0.18 (−0.08, 0.44)
Quartile 4 (≥1688.56)	ref	ref	ref	ref
Distance to other major roadway (m)				
Quartile 1 (<68.01)	0.98 (0.52, 1.87)	0.26 (−0.58, 1.10)	0.32 (−1.09, 1.72)	0.07 (−0.19, 0.33)
Quartile 2 (68.01 – 152.89)	0.97 (0.51, 1.85)	0.03 (−0.80, 0.85)	0.87 (−0.51, 2.24)	0.13 (−0.13, 0.38)
Quartile 3 (152.89 – 307.58)	0.85 (0.45, 1.62)	−0.06 (−0.89, 0.76)	0.85 (−0.55, 2.24)	0.13 (−0.13, 0.39)
Quartile 4 (≥307.58)	ref	ref	ref	ref
Traffic density within 150m (vehicles*km/day) [a]				
Quartiles 1 and 2 combined (0.00 – 437.04)	ref	ref	ref	ref
Quartile 3 (437.04 – 2846.08)	1.03 (0.59, 1.79)	0.32 (−0.40, 1.04)	0.23 (−0.96, 1.43)	0.04 (−0.18, 0.26)
Quartile 4 (≥2846.08)	1.22 (0.70, 2.11)	0.06 (−0.66, 0.79)	0.03 (−1.19, 1.25)	0.00 (−0.22, 0.23)
Traffic density within 250m (vehicles*km/day)				
Quartile 1 (0.00)	ref	ref	ref	ref
Quartile 2 (0.00 – 3276.04)	0.96 (0.51, 1.81)	0.51 (−0.32, 1.35)	−0.02 (−1.42, 1.37)	0.02 (−0.24, 0.28)
Quartile 3 (3276.04 – 9493.52)	0.63 (0.33, 1.22)	−0.15 (−0.96, 0.67)	−1.00 (−2.37, 0.37)	−0.21 (−0.47, 0.04)
Quartile 4 (≥9493.52)	1.16 (0.63, 2.14)	0.04 (−0.79, 0.87)	0.27 (−1.10, 1.64)	0.06 (−0.20, 0.31)
Traffic density within 500m (vehicles*km/day)				
Quartile 1 (<9478.13)	ref	ref	ref	ref
Quartile 2 (9478.13 – 23058.84)	0.60 (0.31, 1.16)	−0.05 (−0.88, 0.78)	−0.20 (−1.59, 1.20)	−0.04 (−0.30, 0.22)
Quartile 3 (23058.84 – 42550.53)	0.62 (0.32, 1.19)	−0.40 (−1.24, 0.45)	0.02 (−1.39, 1.44)	−0.01 (−0.28, 0.25)
Quartile 4 (≥42550.53)	1.08 (0.58, 2.01)	0.35 (−0.50, 1.21)	0.50 (−0.92, 1.92)	0.14 (−0.12, 0.41)

Exposure	Overweight	Waist circumference	Percent fat mass	FMI
	OR (95% CI)	**β (95% CI)**	**β (95% CI)**	**β (95% CI)**
Traffic density within 1000m (vehicles*km/day)				
Quartile 1 (<64368.33)	ref	ref	ref	ref
Quartile 2 (64368.33 – 111625.29)	1.22 (0.61, 2.42)	0.34 (−0.51, 1.20)	**1.50 (0.06, 2.94)**	**0.28 (0.01, 0.54)**
Quartile 3 (111625.29 – 160574.81)	1.56 (0.79, 3.05)	0.44 (−0.44, 1.32)	1.14 (−0.34, 2.62)	0.25 (−0.03, 0.53)
Quartile 4 (≥160574.81)	1.12 (0.56, 2.27)	0.49 (−0.40, 1.38)	0.81 (−0.68, 2.31)	0.24 (−0.04, 0.51)

Abbreviations: FMI = fat mass index. Statistically significant results are highlighted in bold (p <0.05).

Associations are adjusted for child's sex, child's age, maternal age at delivery, pre-pregnancy BMI, parity, race/ethnicity, maternal level of education, maternal smoking during pregnancy, season of birth and median income in the Census tract.

[a]For traffic density in a buffer of 150m around the home address, quartiles 1 and 2 were combined due to the large number of zero values.