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**ABSTRACT:**

There is little known about the health effects from ambient levels of hazardous air pollutants (HAPs), particularly in regard to maternal health. We help fill this gap by investigating whether pregnant women from diverse sociodemographic backgrounds face systematic differences in HAPs exposure, assess the impact of ambient HAP levels on gestational hypertension (GH) and gestational diabetes mellitus (GDM), and determine whether historically underserved groups experience disproportionately more severe health effects at comparable exposure levels. We compile data on the population of pregnant women and singleton births in North Carolina from 2002-2016, and combine these data with a comprehensive, spatially explicit measure of ambient HAP concentrations. We estimate multivariate regression models that employ high-resolution fixed effects and an instrumental variables approach based on reported fugitive emissions to identify the plausibly causal effects of ambient HAP levels on maternal health. The results demonstrate that pregnant women from historically underserved subsets of the population face higher baseline risks of GH and GDM, and are exposed to greater levels of HAPs during pregnancy. We find evidence that a one percent increase in HAPs leads to a roughly 0.03% increase in the risks of GH and GDM, and examine heterogeneity in these health effects based on race, ethnicity, and proxies for income. Considering the average 31% reduction in ambient HAP concentrations during our study period, these results suggest a reduction of 61 to 68 statistical cases of GH in North Carolina each year, and a comparable decrease of 57 to 59 statistical cases of GDM. Our quantified estimates of the maternal health effects can be used to inform future policy decisions and demonstrate how HAPs exacerbate maternal health disparities.

**Keywords:** air pollution; environmental justice; hazardous air pollution; HAP; maternal health; gestational hypertension; gestational diabetes mellitus

**JEL Codes:** I14, I18, Q53, Q56

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## 1. INTRODUCTION

It is well-established that exposure to criteria air pollutants (e.g., fine particulates, nitrogen dioxide, ozone) adversely affects maternal and newborn health (e.g., Currie et al. 2011, Bravo et al. 2024, Ha et al. 2001, Hu et al. 2014, Robledo et al. 2015, Zou et al. 2021). Yet few studies have examined the effects of toxic or hazardous air pollutants (HAPs) on the health of these sensitive populations (Currie et al. 2015, Currie and Schmieder 2009, Ogneva-Himmelberger et al. 2015, Walker et al. 2022). Chemicals designated as a HAP are regulated separately under the US Clean Air Act (US EPA 2024a), often have more heterogeneous (and unknown) properties, and can be more toxic, persistent, and bioaccumulative.

Understanding the health effects of HAPs on maternal populations is particularly pressing in the US, where maternal mortality rates are generally rising (Fleszar et al., 2023). Such broader risk increases are disproportionately experienced by racial and ethnic minority groups, with non-Hispanic Black women experiencing a maternal mortality rate 2.6 times greater than non-Hispanic White women (Hoyert, 2023). Similar disparities are evident among American Indian and Hispanic mothers compared to White mothers, even after adjusting for maternal comorbidities and education (Leonard et al., 2019; Fleszar et al., 2023). These disparities highlight the need to address systemic inequities within a broader framework of maternal health inequity that considers the multifaceted and interconnected drivers of these outcomes, including environmental toxicants.

Our specific research objectives are to (i) examine if pregnant women from different sociodemographic backgrounds are systematically exposed to different levels of HAPs; (ii) identify the effect of ambient HAP levels on two key maternal health outcomes – gestational hypertension (GH) and gestational diabetes mellitus (GDM); and (iii) analyze whether, conditional on the same level of exposure, pregnant women from historically underserved groups experience greater increases in adverse health risks. To fulfill our research objectives, we utilize the Risk-Screening Environmental Indicators Geographic Microdata (RSEI) – a dataset of HAPs from the US Environmental Protection Agency (EPA) that reports location-specific ambient HAP concentrations based on emissions from all stationary air pollution sources reporting to the Toxic Release Inventory (TRI). Industries subject to TRI reporting include manufacturing facilities, metal mines, electric power generators, chemical manufacturers, and hazardous waste treatment facilities (US EPA 2025). We spatially and temporally link the RSEI data to a dataset of over 1.4 million births in North Carolina (NC) from 2002 to 2016, and estimate a series of regression models.

The results confirm that compared to White pregnant women, pregnant women who are Black, Hispanic, or of another race or ethnicity are exposed to higher levels of ambient HAPs. We also find that women living in more urban, metropolitan areas tend to be exposed to higher ambient HAP concentrations. In line with much of the environmental justice literature (Cain et al. 2024), we identify and quantify these disparities, but do not attempt to explain the underlying causal mechanisms leading to higher exposure.

In contrast, we do try to identify the causal effect of HAPs on maternal health (i.e., GH and GDM). The identification strategies employed include census tract fixed effects, mother fixed effects, and an instrumental variable approach that utilizes exogenous variation based on more intermittent, fugitive emissions. Across these numerous approaches, we find fairly robust evidence that

increases in ambient HAPs lead to increased risks of GH and GDM. At the same time, we find that women who are Black, Hispanic, or of another racial or ethnic group, as well as those receiving income-based government aid (e.g., Medicaid or supplemental nutrition programs), experience higher baseline risks of GH and GDM, on average, compared to White pregnant women. The higher exposure to HAPs among these groups, along with the corresponding increases in adverse health effects, demonstrate that HAPs further exacerbate maternal health disparities.

The tools for air quality benefits assessment of federal regulations largely focus on criteria air pollutants, resulting in incomplete benefit-cost and distributional analyses to inform regulatory actions and environmental programs. By quantifying the adverse maternal health effects of HAPs, this study provides results that government agencies and other organizations can use to inform policy decisions.

## 2. BACKGROUND AND LITERATURE

Previous research has shown that facilities that report HAP emissions to the US EPA's Toxic Release Inventory (TRI) are disproportionately located in minority and low-income communities, and that this proximity may impact human health (e.g., Charette et al., 2022; Currie 2011). There are currently 799 listed chemicals under the TRI program. Facilities that manufacture, process, or otherwise use these chemicals in amounts above the established thresholds must submit annual emissions reports for each chemical. Industries subject to TRI reporting include typically larger manufacturing facilities, metal mines, electric power generators, chemical manufacturers, and hazardous waste treatment facilities (US EPA 2025). Focusing on facilities regulated under the US EPA's Risk Management Plan (RMP) program, Guignet et al. (2023, 2024) find that those using highly hazardous chemicals in their industrial processes, and where there is a greater frequency of chemical accidents and releases of HAPs, tend to be located near communities with larger proportions of Black, Hispanic, and Asian populations, and that have higher poverty rates. Pavan et al. (2023) go beyond just relying on proximity and utilize the same modelled ambient HAP concentration data that we use in our study. Focusing on Michigan, they find that Black and Hispanic populations, as well as households living in poverty, are more likely to live in Census tracts with the highest ambient HAP levels. Our study contributes to this literature by examining the environmental equity implications of ambient HAP concentrations in another study area, North Carolina in the southeast U.S.

We then investigate whether higher ambient HAP concentrations translate to changes in maternal health risks. Much of the literature on air pollutants and health risks focus on what are commonly referred to as criteria air pollutants. The US Clean Air Act (CAA) distinguishes between criteria air pollutants versus HAPs, with different regulations applying to each category (US EPA 2024a). The US EPA established National Ambient Air Quality Standards (NAAQS) for the six criteria air pollutants: carbon monoxide (CO), lead (Pb), ground-level ozone ($O_3$), particulate matter ($PM_{2.5}$, $PM_{10}$), nitrogen dioxide ($NO_2$), and sulfur dioxide ($SO_2$) (US EPA 2024a). Although there are some mixed results (e.g., Padula et al. 2018), growing evidence from meta-analyses of the literature support that exposure to criteria air pollutants during pregnancy is associated with increased risks of pregnancy complications, including hypertensive pregnancy disorders and GDM (Hu et al. 2014; Pedersen et al. 2014; Bai et al., 2020).

The literature on the maternal health effects associated with HAPs is far less abundant. There is a small, related literature that finds HAP exposure during pregnancy leads to increases in newborn mortality and morbidity (Currie and Schmieder 2009; Agarwal et al. 2010; Currie et al. 2015; Ogneva-Himmelberger et al. 2015; White et al. 2019; Clay et al. 2025). Others find that the adverse impacts of perinatal HAP exposure may be long-lasting, resulting in intellectual disabilities (Grineski et al. 2022) and reduced educational performance in later childhood (Jacqz 2022). Focusing specifically on maternal health, Walker et al. (2022) conducted a cross-sectional analysis of pregnant women in Kentucky. They find that higher ambient concentrations of lead and chromium are associated with a greater frequency of hypertensive pregnancy disorders. Focusing specifically on polycyclic aromatic hydrocarbons and volatile organic compounds, Zhu et al. (2017) find that high exposure before conception and during the first trimester is associated with a greater risk of GH.

Our study contributes to this literature in three primary ways. First, whereas much of the literature has focused on the maternal health effects of criteria air pollutants, we add to a smaller body of literature on the maternal health effects associated with greater HAP exposure. Second, much of the literature identifies statistical associations and provides suggestive evidence, but our study employs a variety of econometric identification strategies to estimate the plausibly causal relationships between ambient HAP levels and maternal health. As discussed by Brewer et al. (2023), virtually all the dose-response estimates that are currently used to inform impact analyses of air pollution regulations more broadly rely on correlational epidemiological studies. An economics literature has emerged that primarily focuses on identifying the causal health impacts of air pollution (Brewer et al. 2023), and we contribute to this literature by being the first to identify causal relationships between aggregate ambient HAP concentrations and maternal health. Third, we examine whether the maternal health implications of HAPs are heterogeneous across socioeconomic groups. Even when living in areas with similar ambient HAP concentrations, pregnant women may have different levels of awareness, information, and resources available to take mitigative and aversive behaviors. Underlying baseline health conditions could also yield differences in the marginal health effects of an increase in HAPs.

## 3. METHODOLOGY

We investigate three questions. First, does exposure to HAPs vary across socioeconomic groups? Second, does HAP exposure affect maternal health? Third, conditional on HAP exposure, do maternal health effects vary across mothers with different socioeconomic characteristics? Below, we discuss the models used to investigate each of these questions.

### *3.1. Model of HAP Exposure*

We first examine whether mothers with different individual or neighborhood-level characteristics tend to be exposed to relatively higher HAP levels. In contrast to our later two research questions, we do not attempt to identify a causal relationship here. We merely set out to assess whether some groups of pregnant women are systematically exposed to higher levels of HAPs. The formal model to be estimated is:

$$IHS(HAP_{imjst}) = \alpha_0 + \boldsymbol{X}_{imjst}\boldsymbol{\alpha}_1 + \boldsymbol{\omega}_s + \boldsymbol{\rho}_t + u_{imjst} \quad (1)$$

where the vector $\boldsymbol{X}_{imjst}$ denotes characteristics of the mother and her place of residence. The parameters to be estimated are $\alpha_0$, $\boldsymbol{\alpha}_1$, and conception month and year fixed effects, $\boldsymbol{\omega}_s$ and $\boldsymbol{\rho}_t$, respectively. These temporal fixed effects are included to control for broader trends and seasonal effects.

The dependent variable is the ambient concentration of HAPs experienced by mother *m* during the birth of her $i^{th}$ child. The remaining subscripts denote that mother *m* lived in Census tract *j*, and that the baby was conceived in month *s* of year *t.* Given the non-normal and highly right-skewed distribution of HAPs, we transform $HAP_{imjst}$ before it enters equation (1). Usually, a natural log transformation is applied, but given the non-trivial number of zero HAP observations, we instead apply the inverse hyperbolic sine (IHS) transformation for our primary models (Bellemare and Wichman 2020), as shown:

$$IHS(HAP_{imjst}) = ln\left(HAP_{imjst} + \sqrt{HAP_{imjst}^2 + 1}\right) \quad (2)$$

The transformed variable $IHS(HAP_{imjst})$ is the dependent variable in equation (1). The corresponding coefficient vector $\boldsymbol{\alpha}_1$ is of primary interest, as it reflects the associations between HAPs and different maternal and location characteristics, including the mother's race and ethnicity, how urban or rural the mother's place of residence is, and individual- or neighborhood-level proxies for wealth.

### *3.2. Model of Maternal Health Effects*

To answer our second research question—whether exposure to HAPs increases the risk of adverse health effects—we estimate a series of linear probability models (Wooldridge 2010, pp. 562). Given our desire to incorporate high-dimensional fixed effects to control for potentially omitted variables, we pursue a simple linear probability model (LPM) instead of common nonlinear binary models (e.g., probit or logit). Wooldridge (2010, pp. 563) describes how the linear probability model provides a reasonable approximation when the objective is to estimate the partial effects of the explanatory variables on the probability of the event of interest, as is the case here. Additionally, the LPM accommodates a mix of continuous and categorical variables and offers easily interpretable marginal effects (Angrist and Pischke 2009). We then use the estimated coefficients from the LPMs to express how ambient HAP levels affect maternal health in terms of semi-elasticities and elasticities.

Consider the basic LPM where $Y_{imjst}$ is a binary indicator equal to one if an adverse health condition was diagnosed during the pregnancy (i.e., GH or GDM), and zero otherwise. The independent variable of primary interest is the continuous measure of ambient HAP concentrations, $IHS(HAP_{imjst})$. The vector $\boldsymbol{X}_{imjst}$ again entails characteristics of the mother, and in some models, of the neighborhood and location where the mother lived. The formal model is:

$$Y_{imjst} = \beta_1 IHS\left(HAP_{imjst}\right) + \boldsymbol{X}_{imjst}\boldsymbol{\beta}_2 + \boldsymbol{\eta}_s + \boldsymbol{\tau}_t + \varepsilon_{imjst} \quad (3)$$

where $\varepsilon_{imjst}$ is an assumed normally distributed disturbance term that is allowed to be correlated within counties. The parameters to be estimated are $\beta_1$ and $\boldsymbol{\beta}_2$, and $\boldsymbol{\eta}_s$ and $\boldsymbol{\tau}_t$ are conception month and year fixed effects, respectively.

To provide a more intuitive interpretation, we express the relationship between HAPs and maternal health as semi-elasticities and elasticities. For notational ease, we drop the subscripts when deriving the semi-elasticity and elasticity formulas. A useful intermediate result is that the derivative of $IHS(HAP)$ is $\frac{\partial IHS(HAP)}{\partial HAP} = \frac{1}{\sqrt{HAP^2+1}}$.

The corresponding semi-elasticity $(\nu)$ can be calculated by taking the partial derivative of equation (3) with respect to $HAP$, and then multiplying both by sides by $HAP$. More formally:

$$\nu = \partial Y \frac{HAP}{\partial HAP} = \frac{\beta_1 HAP}{\sqrt{HAP^2+1}} \quad (4)$$

Semi-elasticities of this form would normally be interpreted as a one percent change in HAPs leads to a $\nu/100$ unit change in $Y$. However, $Y$ is binary in our application, and so the correct interpretation is that a one percent change in HAPs is associated with a $\nu$ percentage point change in the risk of experiencing the adverse health outcome (i.e., the probability that $Y = 1$).[1]

Dividing both sides of equation (4) by $Y$ yields the corresponding elasticity, as shown in equation (5).[2] This elasticity is interpreted as that a one percent change in HAPs leads to a $\xi$ percent change in $Y$.

$$\xi = \frac{\partial Y}{Y}\frac{HAP}{\partial HAP} = \frac{\beta_1 HAP}{Y\sqrt{HAP^2+1}} \quad (5)$$

When calculating the mean elasticities and semi-elasticities as per equations (4) and (5), we use the sample mean values for $Y$ and $HAP$.

A key objective is to estimate the causal effect of ambient HAPs on maternal health. Ambient air pollution concentrations, however, are not random and can be correlated with individual and community socioeconomic characteristics. For example, pregnant women who experience high levels of HAPs may also be more likely to experience other environmental, financial, and social stressors. In fact, the environmental justice literature suggests that this is often the case (e.g., Charette et al. 2022; Currie 2011; Pavan et al. 2023; Guignet et al. 2023, 2024). If such factors are correlated with maternal health outcomes but are not fully controlled for in equation (3), then

[1] In other words, to interpret this zero to one indicator as a zero to 100 percentage, we multiply by 100. Doing so cancels out the aforementioned division by 100 that is common when interpreting semi-elasticities from a linear-log model and similar specifications, like that in equation (3).

[2] See Bellemare and Wichman (2020) for an in-depth discussion of elasticities when using the inverse hyperbolic sine transformation.

estimates of $\beta_1$ and the resulting estimates in equations (4) and (5) will suffer from an omitted variable bias.

We attempt to minimize the potential for such bias in several ways. First, a comprehensive set of individual-level variables are included in the vector $\boldsymbol{X}_{imjst}$, including demographic (e.g., age, race/ethnicity, marital status), socioeconomic (e.g., WIC, Medicaid), educational (e.g., college education), and behavioral characteristics (e.g., smoking) of the mother; and characteristics of the surrounding community (e.g., urbanicity/rurality, and the percent of the population that is Black, Hispanic, and living in poverty), including other environmental stressors (e.g., criteria air pollutants, extreme heat) (see Table 1 for details). Conception month and year fixed effects $\boldsymbol{\eta}_s$ and $\boldsymbol{\tau}_t$ are included to flexibly control for broader temporal trends and seasonal effects associated with maternal health. Second, we employ three alternative techniques to further control for otherwise potentially confounding factors, and ultimately to estimate a plausibly causal relationship between HAPs and maternal health—neighborhood-level spatial fixed effects, individual-level maternal fixed effects, and an instrumental variables approach. Each of these approaches has advantages and disadvantages.

We define neighborhoods based on the U.S. Census Bureau's 2010 tract definitions and include tract-level spatial fixed effects $\theta_j$, as shown:

$$Y_{imjst} = \beta_1 IHS\left(HAP_{imjst}\right) + \boldsymbol{X}_{imjst}\boldsymbol{\beta}_2 + \boldsymbol{\eta}_s + \boldsymbol{\tau}_t + \theta_j + \varepsilon_{imjst} \quad (6)$$

The fixed effects $\theta_j$ account for all time-invariant factors associated with maternal health that are constant within neighborhood *j*, which could include the general character of the neighborhood, crime, environmental conditions, how urban or rural a location is, proximity to healthcare providers, and other location-specific factors. The coefficient $\beta_1$ is then identified based on temporal and within-tract variation in HAPs and maternal health.

An initial concern with the neighborhood fixed effects approach is whether there is sufficient temporal variation in HAPs to estimate $\beta_1$. If not, $\theta_j$ could inadvertently absorb HAP-related health effects of interest (Abbott and Klaiber 2011).[3] This concern, however, is at least partially alleviated based on the observed temporal variation in the HAPs data (see Figures 1 and 2).

In the same spirit as the spatial fixed effects, we also estimate models that include higher resolution individual-level maternal fixed effects $\psi_m$, as shown in equation (7). These maternal fixed effects account for all time-invariant factors associated with each individual mother, which may include wealth and other available resources, general health, behavioral factors, etc. Under the maternal fixed effects model in equation (7), $\beta_1$ is identified solely from within-mother variation in the maternal health outcomes and HAPs. Such variation in HAPs stems from temporal changes in the pollution levels, as well as if a mother moves residences in between pregnancies.[4]

[3] This is a potential concern in our application. Regression models of $IHS\left(HAP_{imjst}\right)$ on just Census tract fixed effects yield R-squares of 0.677 to 0.908, suggesting that the tract fixed effects explain a large portion of the variation in HAPs.

[4] Of the 325,777 mothers in our data where two or more births are observed, 169,660 (52%) moved to another census tract at least once in between births.

$$Y_{imjst} = \beta_1 IHS\left(HAP_{imjst}\right) + \boldsymbol{X}_{imjst}\boldsymbol{\beta}_2 + \boldsymbol{\eta}_s + \boldsymbol{\tau}_t + \psi_m + \varepsilon_{imjst} \quad (7)$$

While controlling for potentially confounding mother-specific factors is an attractive feature, there are some disadvantages. First, the sample size is significantly limited because all mothers who gave birth to only one child in North Carolina during our 2002-2016 study period are disregarded.[5] Second, reliance on only mothers who give birth to two or more children could lead to a selection bias, which may diminish the generalizability of our results to the broader population of interest. Mothers who have multiple children could be systematically different than those who have one child. Of particular concern for this analysis is that mothers who experienced adverse maternal health outcomes in the past may be less likely to have additional children. Both of these points are investigated (see Section 5.2 and Appendix B). Third, as with the spatial fixed effects model, mother fixed effects could absorb key variation in maternal health and HAPs that we are interested in estimating.

As an alternative to the spatial and mother fixed effects models, we pursue an instrumental variables (IV) approach to address otherwise potentially confounding factors (see, for example, Greenstone and Gayer (2009) and Wooldridge (2010)). The first stage equation is:

$$IHS\left(HAP_{imjst}\right) = \boldsymbol{X}_{imjst}\boldsymbol{\alpha}_1 + \alpha_2 Z_{imjst} + \boldsymbol{\omega}_s + \boldsymbol{\rho}_t + u_{imjst} \quad (8)$$

where $u_{imjst}$ is an assumed normally distributed disturbance term, and $\boldsymbol{\alpha}_1$ and $\alpha_2$ are parameters to be estimated, as are the corresponding month and year fixed effects, $\boldsymbol{\omega}_s$ and $\boldsymbol{\rho}_t$. The variable $Z_{imjst}$ denotes the inverse hyperbolic sine of ambient HAP concentrations based solely on fugitive emissions. The predicted values for $IHS\left(HAP_{imjst}\right)$ from equation (8) are then plugged into the second stage equation, as shown:

$$Y_{imjst} = \beta_1 IHS\left(\widehat{HAP}_{imjst}\right) + \boldsymbol{X}_{imjst}\boldsymbol{\beta}_2 + \boldsymbol{\eta}_s + \boldsymbol{\tau}_t + \varepsilon_{imjst} \quad (9)$$

The variable $Z_{imjst}$ serves as the assumed exogenous instrument used for identification under our IV approach, and therefore must meet two conditions. First, $Z_{imjst}$ must be a strong predictor of ambient HAP concentrations. Second, $Z_{imjst}$ must only affect the corresponding health outcome $Y_{imjst}$ indirectly through $IHS\left(\widehat{HAP}_{imjst}\right)$, meaning that $Z_{imjst}$ is uncorrelated with $\varepsilon_{imjst}$ in equation (9). Our broader ambient HAP measure is based on emissions from surrounding industrial facilities, including two types of emissions – stack and fugitive emissions. Stack emissions are those reported as point-source emissions, usually from smokestacks and other confined air streams (e.g., vents, ducts, and pipes). In contrast, fugitive emissions in our data are described as all non-stack (or nonpoint) releases, such as equipment leaks from valves, pump seals, and flanges; releases from building ventilation systems; fugitive dust, and evaporative losses from impoundments and spills. Although stack emissions often routinely occur, fugitive emissions are more intermittent (Clay et al. 2025). Subject facilities must report both routine and accidental

[5] About 62% of the mothers in our sample gave birth to only one child during our study period, and are thus excluded when estimating the maternal fixed effects models.

releases, including releases resulting from catastrophic or other one-time events (US EPA 2023). Such accidental releases may be more likely reported as fugitive emissions. The US EPA's RSEI data provides the information needed to derive an aggregate measure of ambient HAP concentrations based solely on fugitive emissions, and we use this measure as our instrument $Z_{imjst}$. In their analysis of airborne lead emissions, Clay et al. (2025) proposed a similar IV approach, where they instrument based on an interaction term between fugitive lead emissions and wind speed. Our measure of fugitive-only ambient HAPs is modelled by the US EPA based on fugitive emissions, wind, and other factors (US EPA 2023). We demonstrate that our fugitive-only ambient HAP concentration measure satisfies the first condition needed to be a valid instrument; and to the extent that fugitive emissions reflect "random" variation in toxic spills, accidents, and intermittent and one-time release events, this instrument satisfies the second necessary condition.[6]

Key advantages of the IV approach are that if the above two conditions hold then estimates of $\beta_1$ will be stripped of any confounding biases and thus provide a plausibly causal estimate of how ambient HAP concentrations affect maternal health. Additionally, in contrast to the spatial and maternal fixed effect specifications, we do not have to be concerned about the fixed effects absorbing variation of interest, nor about potential biases due to sample selection. The disadvantage of the IV approach is that a causal interpretation hinges on the assumed exogeneity of the fugitive emissions instrument. We also estimate variants of our IV models that also employ tract-level spatial fixed effects ($\theta_j$). A combined tract fixed effects and IV model is advantageous because by conditioning on tract fixed effects, we need only assume that temporal and highly local within-tract variation in our assumed fugitive-based ambient HAP instrument is exogenous. This helps guard against concerns that, for example, fugitive emissions may be correlated with unobserved factors associated with more disadvantaged neighborhoods.

Overall, none of the proposed identification strategies are perfect, and so we examine the robustness of our findings across all approaches and estimate numerous variants of the models shown in equations (3), and (6) through (9).

### *3.3. Heterogeneity in Maternal Health Effects*

To examine our last question about heterogeneity in health effects across pregnant women with different characteristics, we add interaction terms between $IHS(HAP_{imjst})$ and key variables from the vector $\boldsymbol{X}_{imjst}$. More specifically, we include whether a pregnant woman is Nonwhite (i.e.,

[6] A series of regression models were estimated where ambient concentrations based only on smokestack emissions is the dependent variable, and the independent variables are fugitive-based ambient concentrations; mother and neighborhood characteristics; conception month and year fixed effects, and tract or mother fixed effects. If fugitive-based ambient HAP concentrations are uncorrelated with more routine stack-based concentrations, then this lends support to our assumption that fugitive-based HAPs is as good as random. Linear models of smokestack and fugitive emissions-based ambient HAP concentrations suggest negligible or perhaps even a small negative relationship, lending support to the claim that fugitive emissions are quasi-random and that our fugitive-only ambient HAP measure serves as a valid instrument. Some models where both stack- and fugitive-based ambient HAP concentrations enter following the inverse hyperbolic sine transformation do suggest a positive correlation, with positive coefficient estimates as high as 0.375 (when no other covariates are controlled for). Notably, however, when neighborhood specific factors are controlled for through the inclusion of Census tract fixed effects, we see a small and statistically insignificant 0.016 coefficient estimate, again lending support to our use of the fugitive-based ambient HAP concentration measure as an instrument. See Tables C1 and C2 in Appendix C for details.

Black, Hispanic, or identifies as another racial or ethnic group); has no college degree; or is on Medicaid. The latter two variables are intended to act as individual-level proxies for income. Let $x^0_{imjst}$ denote one of these variables, and $H_{imjst} = IHS(HAP_{imjst}) \times x^0_{imjst}$ is the corresponding interaction term. We focus on the spatial fixed effects and IV models for this examination of health effect heterogeneity. The spatial fixed effects model variant is straightforward, where we simply add the interaction term to equation (6), as shown:

$$Y_{imjst} = \beta_1 IHS(HAP_{imjst}) + \boldsymbol{X}_{imjst}\boldsymbol{\beta}_2 + \beta_3 H_{imjst} + \boldsymbol{\eta}_s + \boldsymbol{\tau}_t + \theta_j + \varepsilon_{imjst} \quad (10)$$

The coefficient $\beta_3$ will reflect any heterogeneity in the maternal health effects associated with HAPs.

A similar variant of the IV approach is pursued, where for statistical identification two first-stage equations must be estimated. The first is for the original endogenous variable $IHS(HAP_{imjst})$, and the second is for the corresponding interaction term $H_{imjst}$, which is also considered endogenous. For this system of equations, we add an interaction term between our instrument $Z_{imjst}$ and $x^0_{imjst}$ for the second exclusion restriction, as follows:

$$IHS(HAP_{imjst}) = \boldsymbol{X}_{imjst}\boldsymbol{\alpha}_1 + \alpha_2 Z_{imjst} + \alpha_3\{Z_{imjst} \times x^0_{imjst}\} + \boldsymbol{\omega}^a_s + \boldsymbol{\rho}^a_t + u^a_{imjst} \quad (11a)$$

$$H_{imjst} = \boldsymbol{X}_{imjst}\boldsymbol{\gamma}_1 + \lambda_2 Z_{imjst} + \lambda_3\{Z_{imjst} \times x^0_{imjst}\} + \boldsymbol{\omega}^b_s + \boldsymbol{\rho}^b_t + u^b_{imjst} \quad (11b)$$

where $\alpha_1$,…, $\alpha_3$, and $\lambda_1$, …, $\lambda_3$ are coefficients to be estimated, and $\boldsymbol{\omega}^k_s$ and $\boldsymbol{\rho}^k_t$, are the corresponding seasonal and annual fixed effects, for $k = a, b$; and $u^k_{imjst}$ denotes the corresponding disturbance terms.

The second stage equation is then estimated as shown, where the predicted values for $IHS(HAP_{imjst})$ and $H_{imjst}$ from equations (11a) and (11b), respectively, are substituted into the right-hand side:

$$Y_{imjst} = \beta_1 IHS(\widehat{HAP}_{imjst}) + \boldsymbol{X}_{imjst}\boldsymbol{\beta}_2 + \beta_3 \widehat{H}_{imjst} + \boldsymbol{\eta}_s + \boldsymbol{\tau}_t + \varepsilon_{imjst} \quad (12)$$

If estimates of $\beta_3$ from equations (10) and (12) are statistically insignificant, then it suggests that pregnant women in different socioeconomic groups experience the same increase in health risks from an increase in ambient HAPs. There is reason to suspect, however, that even when living in locations with the same ambient HAP concentrations, some women may experience greater marginal increases in adverse health risks due to differences in awareness and information, resources for taking mitigative and aversive actions, and underlying baseline health conditions.

## 4. DATA

A comprehensive dataset of maternal health outcomes and ambient HAP concentrations is compiled for all of North Carolina, spanning the population of singleton births from 2002-2016.[7] We next describe the various data sources and processing, and then discuss the final dataset for analysis.

### *4.1. Data sources*

#### *4.1.1. Maternal Health Data*

The maternal health data for this study were obtained from the Vital Statistics Department of North Carolina State Center for Health Statistics (NCSCHS). This research is conducted under an agreement with the Children's Environmental Health Initiative (CEHI) at the University of Illinois-Chicago. The data include detailed records for all live births in North Carolina, including information on maternal health and obstetrics history.

We analyze two maternal health outcomes – gestational hypertension (GH) and gestational diabetes mellitus (GDM). Information on GH is available for most of the 2002-2016 study period but was not reported for 2010 due to a transition in data reporting procedures by NCSCHS. Data on GDM was not recorded until after this transition, so information on GDM is only available for 2011-2016.

The North Carolina birth records data provide information on a mother's race and ethnicity, education, age, smoking status, birth order, etc. From 2011 through 2016, the birth records data also contain information on whether the mother participated in the US Department of Agriculture's Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) or paid for medical visits through Medicaid. Both are income-based government assistance programs, and are included as a proxy for income. Most notably, the data include the estimated date of conception, the date of birth, and the coordinates of the mother's place of residence at the time of the child's birth. The individual-specific coordinates and the temporal window of pregnancy are used to link each mother-birth record to census tract sociodemographic data and to ambient HAPs and other location-specific environmental stressors.

#### *4.1.2. Hazardous Air Pollution (HAP) Data*

Our measure of hazardous air pollutants (HAPs) is based on the US EPA's Risk Screening Environmental Indicators Geographic Microdata (RSEI). The EPA provides chemically aggregated RSEI data for each year from 2001 to 2016 at various geographic scales. We use the data reported at the 2010 Census block group level. These data are based on modeled chemical air concentrations derived from the American Meteorological Society/EPA Regulatory Model (AERMOD). AERMOD uses pollutant emissions, wind speed and direction, ambient temperature, and observed cloud cover to determine chemical transport and final ground-level air concentrations

[7] Observations pertaining to births in 2010 are excluded. This year was a transition year in how the Vital Statistics Department of the North Carolina State Center for Health Statistics (NCSCHS) recorded birth records, and key information pertaining to maternal health was missing for this year.

(EPA 2023). The annual ambient air concentrations for each block group are based on the HAP emissions reported to the EPA's Toxic Release Inventory (TRI) by all facilities within 50 miles of a block group. Ambient air pollutant concentrations are modelled for each hazardous chemical reported to the TRI. For this analysis we focus on the aggregate toxicity-weighted annual ambient concentration calculated by EPA, which provides a comprehensive and spatially detailed measure of toxic chemicals in the air, accounting for numerous chemicals and across many polluting facilities.

Subsequent data processing uses the chemical-specific RSEI data, which is disaggregated by individual chemicals and types of emissions. These data are used to create an ambient HAP concentration measure that is based solely on fugitive emissions, which we argue is a plausible instrument for statistical identification under our IV modeling approach (see Section 3.2).

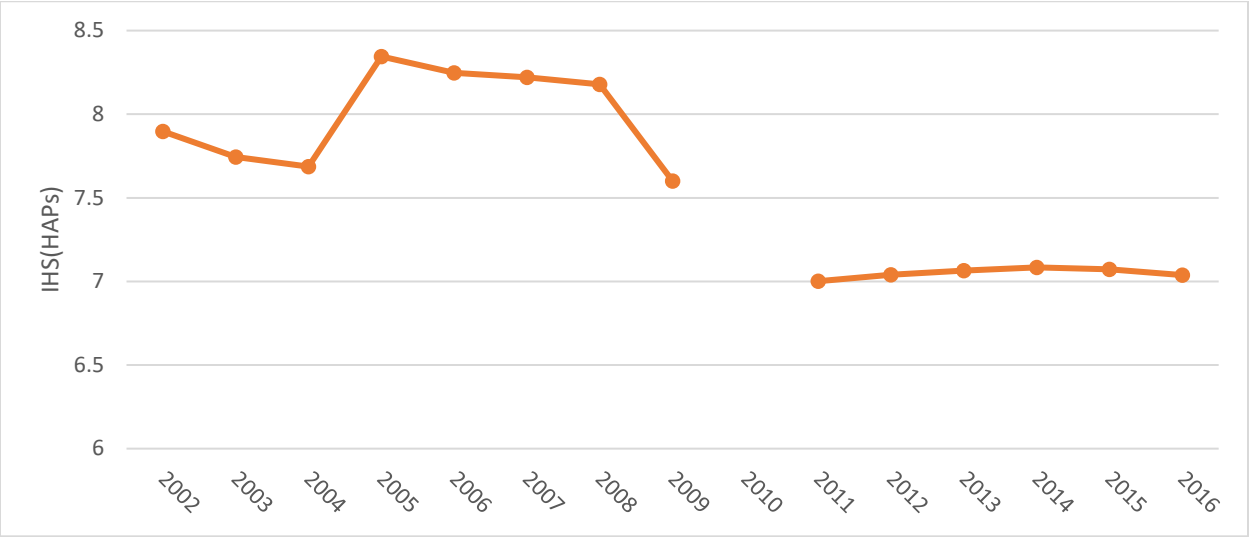
Although the primary analysis focuses on the toxicity-weighted aggregated measure of ambient HAP concentrations, it is worth noting that chemicals like methanol, toluene, ammonia, and lead and lead compounds are among the most commonly reported HAPs in North Carolina. These chemicals are used as industrial solvents, in paints and paint thinners, in fuel and cleaning products, fertilizer, and in the production of batteries and a variety of other industrial and agricultural processes. These chemicals are also associated with numerous adverse health effects. For example, chronic or repeated exposure to methanol can cause neurological issues, and lead to birth defects among newborns (US HHS 2011). Chronic and repeated exposure to toluene can lead to neurological issues and even brain damage (ATSDR n.d.). Newborns exposed to large amounts of toluene during gestation due to purposeful inhalation (i.e., pregnant women "huffing" to get high) face an increased risk of birth defects, including reduced mental abilities and growth (ATSDR n.d.). Ammonia can irritate the upper respiratory system and skin, and acute exposure to higher concentrations can lead to death (ATSDR n.d.). There is likely causal evidence that prenatal lead exposure is associated with adverse birth outcomes (US EPA 2024b; Bui et al. 2022). Lead is also associated with increased risks of cancer, kidney and cardiovascular issues, and mortality in adults (US EPA 2024b; US HHS 2020; Klemick et al. 2022), and causes cognitive deficits, attention disorders, and behavioral problems in children (US EPA 2024b; Lanphear et al. 2005; Miranda et al. 2007; Aizer et al. 2018; US HHS 2020; Shadbegian et al. 2019, 2024).

Each birth record in our data is linked to the corresponding annual toxicity-weighted average ambient HAP concentration based on the census block group where a woman resided during pregnancy. If the entire gestation period occurred within the year of birth, then the corresponding annual toxicity-weighted ambient HAP concentration for that year was assumed. In many cases, however, the pregnancy overlapped two calendar years, and so a weighted average is taken across the two years based on the proportion of the gestation period overlapping each year, yielding a measure of HAPs that varies based on the date of conception.[8]

As demonstrated in Figures 1 and 2, there is noticeable spatial and temporal variation in the toxicity-weighted average annual HAP concentrations. Such variation strengthens our identification strategy, especially in models that include Census tract or mother fixed effects.

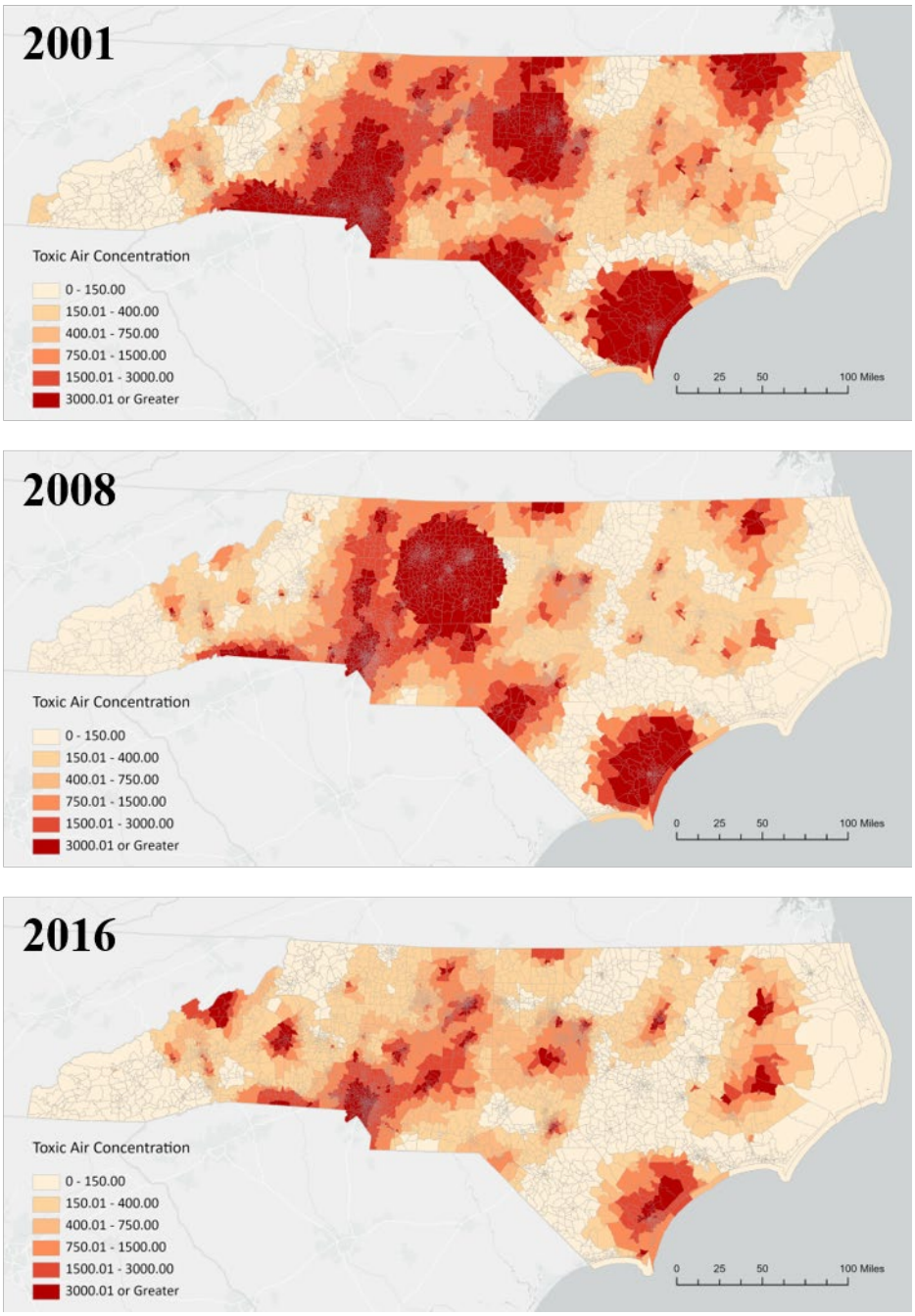
[8] Although we focus on pregnant women who gave birth between 2002-2016, the data on HAPs (and other environmental stressors discussed later, i.e., criteria air pollutants and high temperature extremes) include earlier years, and thus allow us to account for exposures during the entire gestation period for all observations, including cases where conception occurred before 2002.

Figure 1. Annual average of toxicity-weighted mean HAPs during pregnancy.




Note: The y-axis displays the transformed ambient HAP concentration, based on the inverse hyperbolic sine (IHS) transformation (see section 3.1 for details). Data for 2010 are omitted due to changes in how the NCSCHS records birth observations and the resulting missing information for key maternal health variables.

Figure 2. Toxicity-weighted HAP concentrations by 2010 Census Block Group for 2001 (top panel), 2008 (middle panel), and 2016 (bottom panel).




*4.1.3. Other Environmental Stressors and Location Characteristics*

We attempt to account for a comprehensive set of location-specific factors that may affect maternal health and could otherwise confound our estimates of the effects of HAPs. Such factors include characteristics of the neighborhood where an individual lives, as well as exposure to other

environmental stressors, namely extreme heat and criteria air pollutants. These variables are merged to each maternal birth record based on their residential coordinates and the timing of conception.

Ambient HAP levels may be positively correlated with other criteria air pollutants that have been shown to affect maternal and newborn health (see section 2). To account for these otherwise potentially confounding effects, we include the average concentration of $PM_{2.5}$, $O_3$, and $NO_2$ during pregnancy. These birth-specific measures were derived by CEHI using daily concentration data from the Socioeconomic Data and Applications Center (SEDAC).[9]

Similarly, extreme heat has been shown to affect maternal health (e.g., Shashar et al. 2020; Part et al. 2022; Kim et al. 2021; Baharav et al., 2023; Ulrich et al. 2025). We control for extremely high temperatures by calculating the percent of days during each pregnancy where the daily average temperature exceeded the 95$^{th}$ percentile for spring and summer months (i.e., May to September). Daily temperature data were collected at the census tract level for average, maximum, and minimum temperatures for the contiguous United States using the National Center for Environmental Information's (NCEI) nClimdGrid-Daily. These data are derived from observations from the Global Historical Climatology Network daily dataset, with additional processing for spatial and temporal variations (Durre et al., 2022). To account for local differences in acclimatization, the 95$^{th}$ percentile thresholds are calculated based on the temperature distribution for spring and summer months from 2001-2019, and this is done separately for each of North Carolina's eight climate divisions.[10]

Additional location-specific factors are included to account for sociodemographic characteristics of a neighborhood and how urban or rural an area is. We account for the percent of the local population that is living in poverty; Black; and of Hispanic, Latino or Spanish descent. These variables are derived at the census tract level based on the 2010 American Community Survey (ACS) (5-year estimates, 2006-2010). We rely on urban versus rural designations based on the Rural-Urban-Commuting Area (RUCA) codes for 2010 provided by the United States Department of Agriculture (USDA). RUCA codes classify census tracts into different levels of urbanicity or rurality based on the size of urban centers and the extent of commuting flows between rural and urban areas. RUCA designations were linked to each birth record based on the corresponding 2010 census tract. We designated census tracts as being either a metropolitan area (RUCA=1, 2, or 3), micropolitan area (RUCA=4, 5, or 6), small town (RUCA=7, 8, or 9) or rural (RUCA=10).

### *4.2. Data Processing and Descriptive Statistics*

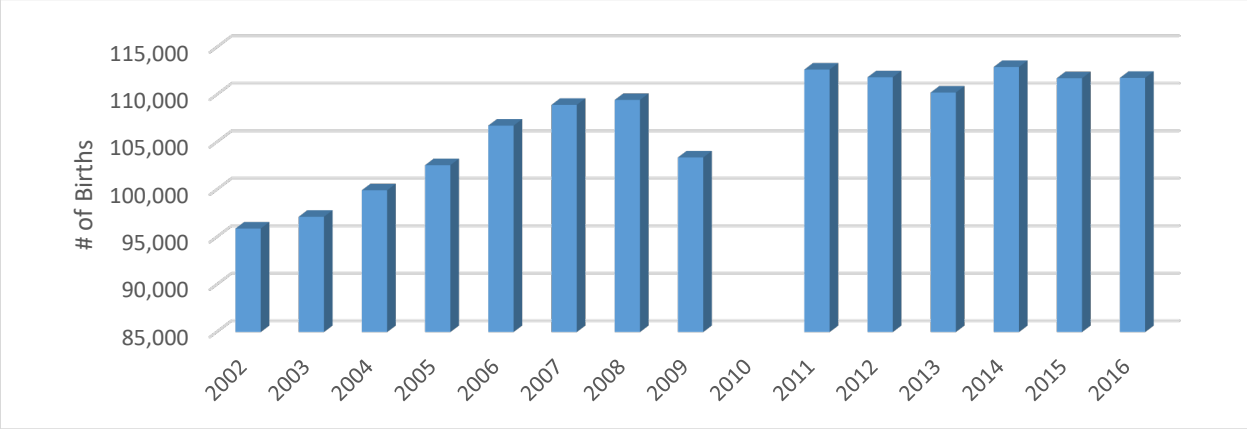
The original dataset started with the population of over 2.4 million live births in NC from 2001 through 2019. Focus was then drawn to singleton birth events between 2002-2016. We started the study period in 2002 because the relevant data of ambient HAPs was only available starting in

[9] Socioeconomic Data and Applications Center (SEDAC). 2021. Daily and Annual PM2.5 Concentrations for the Contiguous United States, 1-km Grids, v1 (2001-2016). https://beta.sedac.ciesin.columbia.edu/data/set/aqdh-pm2-5-concentrations-contiguous-us-1-km-2000-2016, accessed 14 April 2024.

[10] The climate divisions are delineated by the National Oceanic and Atmospheric Administration (NOAA) based on area-weighted daily temperature and precipitation values (Karl and Koss, 1984).

2001, and we wanted to make sure we had a measure of HAPs during the entire pregnancy period leading up to the date of birth. We ended the study period with births in 2016 because the processed data on criteria air pollutants were only available up until this date, and we believe that these criteria air pollutants are important to control for when trying to identify the effect of HAPs on maternal health. We then excluded observations where the geographic coordinates for the mother's place of residence were not available. Lastly, we dropped 108,139 birth observations that occurred in 2010, because information on maternal health was not available that year due to NCSCHS's transition in data recording procedures. This left a final dataset of 1,495,331 singleton births in NC from 2002 through 2016. Figure 2 shows the number of births by year in our final dataset for analysis.

Figure 3. Number of Births by Year.




Note: Data for 2010 are omitted due to changes in how the NCSCHS records birth observations and the resulting missing information for other key variables.

Descriptive statistics for our final sample are displayed in Table 1. First focusing on our adverse maternal health outcomes, we see GH occurs in about 5.5% of the mother-by-birth observations, and we see about the same rate for GDM.

Our NC births data includes individual-level sociodemographic data for pregnant women. The majority of pregnant women are White (56%), followed by Black (23%), Hispanic or Latino (15%), or another race or ethnic group (almost 6%). For less than 0.1% of the observations, race and ethnicity were not reported. The greatest proportion of the mother-by-birth observations pertain to a woman's first birth (41%), and this proportion decreases for the second birth (33%), third birth (16%), etc. We divided maternal age into three categories, with the later omitted category being pregnant women between the ages of 25 and 35 years. About 35% of the study population are 15 to 24 years old at the time of the child's birth, and 13% are aged 35-44 years. Among most pregnant women who reported smoking behavior, we see about 10% smoked during their pregnancy.

Although we do not observe individual-level household income, we control for several proxy variables that are likely correlated with household income, including being unmarried at the time of birth (39%), or having at least a bachelor's degree (28%). From 2011 onward, the data include information on whether an individual participated in the USDA's WIC supplemental food

program, or was enrolled in Medicaid. When such information is reported, we see that 47% of our sample participated in WIC, and about 44% were enrolled in Medicaid. Although the descriptive statistics for these variables exclude observations with missing values, in our later regression models we code missing values as zero and include a companion missing value indicator. This applies for all variables reported in Table 1 where a companion missing value indicator is presented.

As can be seen in Table 1, most of the sample lived in metropolitan (80%) and micropolitan (14%) areas, followed by small towns (4%) and rural areas (less than 2%). On average, a mother lives in a Census tract where about 16% of households are living in poverty, 24% of the population is Black, and 9% of the population is Hispanic or Latino.

Table 1. Births and Maternal Data Descriptive Statistics.

| Variable | Obs | Mean | Std. Dev. | Min | Max |
|---|---|---|---|---|---|
| Gestational hypertension (GH) | 1,495,331 | 0.055 | 0.228 | 0 | 1 |
| Gestational diabetes mellitus (GDM) | 671,201 | 0.055 | 0.227 | 0 | 1 |
| Ambient HAPs | 1,495,331 | 57,265 | 1,330,412 | 0 | 1.85E+08 |
| IHS(Ambient HAPs) | 1,495,331 | 7.572 | 2.130 | 0 | 19.730 |
| % days with temp ≥ 95th pct | 1,495,331 | 2.88 | 2.74 | 0 | 46.03 |
| $PM_{2.5}$ ($\mu g/m^3$) | 1,495,331 | 11.04 | 2.40 | 2.53 | 19.47 |
| Ozone (ppb) | 1,495,331 | 41.03 | 3.27 | 27.49 | 60.00 |
| $NO_2$ (ppb) | 1,495,331 | 17.03 | 7.40 | 0.17 | 61.82 |
| White | 1,495,331 | 0.561 | 0.496 | 0 | 1 |
| Black | 1,495,331 | 0.228 | 0.420 | 0 | 1 |
| Hispanic | 1,495,331 | 0.153 | 0.360 | 0 | 1 |
| Other race/ethnicity | 1,495,331 | 0.057 | 0.232 | 0 | 1 |
| Race/ethnicity not reported | 1,495,331 | 0.001 | 0.026 | 0 | 1 |
| First birth | 1,495,331 | 0.413 | 0.492 | 0 | 1 |
| Second birth | 1,495,331 | 0.327 | 0.469 | 0 | 1 |
| Third birth | 1,495,331 | 0.162 | 0.368 | 0 | 1 |
| Fourth birth | 1,495,331 | 0.062 | 0.241 | 0 | 1 |
| Fifth or greater birth | 1,495,331 | 0.037 | 0.188 | 0 | 1 |
| Maternal age 15-24 years | 1,495,331 | 0.352 | 0.478 | 0 | 1 |
| Maternal age 35-44 years | 1,495,331 | 0.131 | 0.337 | 0 | 1 |
| Not married | 1,495,331 | 0.393 | 0.488 | 0 | 1 |
| Smoked | 1,494,353 | 0.106 | 0.308 | 0 | 1 |
| Missing: Smoked | 1,495,331 | 0.001 | 0.026 | 0 | 1 |
| Maternal education: college | 1,492,738 | 0.282 | 0.450 | 0 | 1 |
| Missing: Maternal education | 1,495,331 | 0.002 | 0.042 | 0 | 1 |
| WIC | 670,008 | 0.474 | 0.499 | 0 | 1 |
| Missing: WIC | 1,495,331 | 0.552 | 0.497 | 0 | 1 |
| Medicaid | 670,578 | 0.443 | 0.497 | 0 | 1 |
| Missing: Medicaid | 1,495,331 | 0.552 | 0.497 | 0 | 1 |

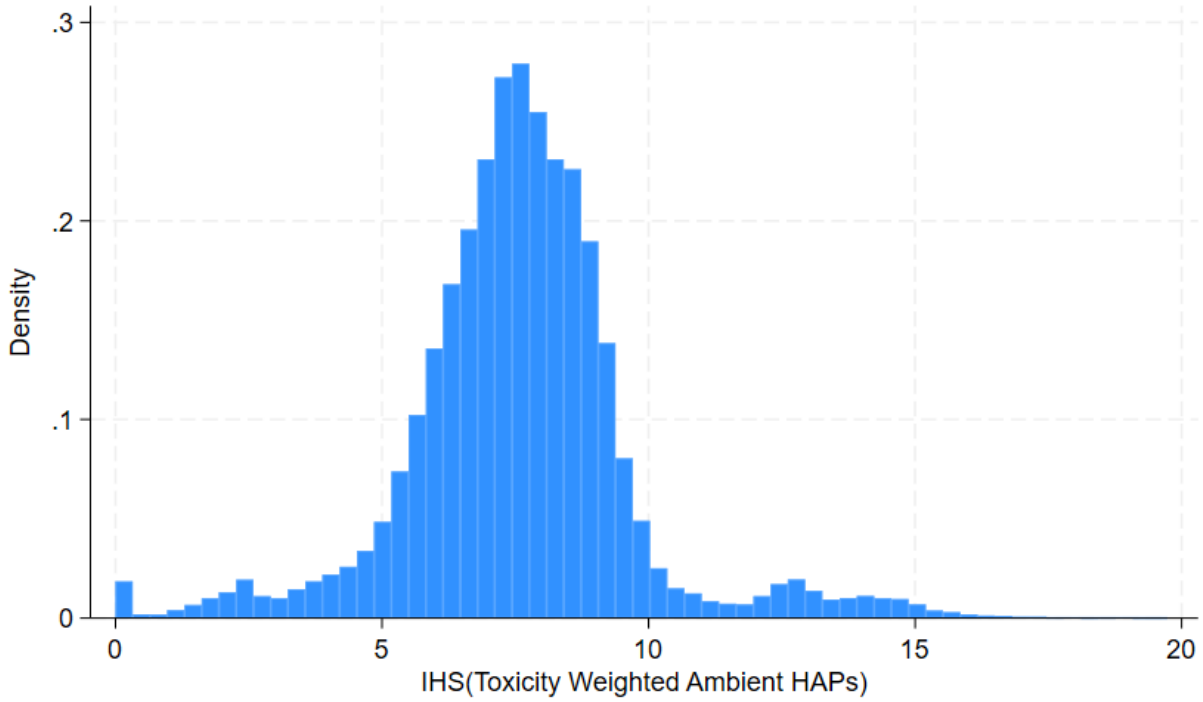
| | | | | | |
|---|---|---|---|---|---|
| Metropolitan area | 1,495,331 | 0.804 | 0.397 | 0 | 1 |
| Micropolitan area | 1,495,331 | 0.138 | 0.345 | 0 | 1 |
| Small town | 1,495,331 | 0.040 | 0.196 | 0 | 1 |
| Rural area | 1,495,331 | 0.018 | 0.132 | 0 | 1 |
| Rural/urban not reported | 1,495,331 | 3.34E-06 | 0.0018 | 0 | 1 |
| % Poverty in Tract | 1,495,273 | 16.36 | 11.13 | 0 | 96.28 |
| % Black in Tract | 1,495,302 | 24.00 | 21.26 | 0 | 100 |
| % Hispanic in Tract | 1,495,302 | 9.38 | 9.29 | 0 | 71.39 |
| Missing: % Poverty in Tract | 1,495,331 | 3.88E-05 | 0.006 | 0 | 1 |
| Missing: % Black/Hispanic in Tract | 1,495,331 | 1.94E-05 | 0.004 | 0 | 1 |

Of primary interest for this study is our measure of HAPs. The average toxicity-weighted ambient HAP concentration that a woman is exposed to during pregnancy is 57,265, but there is substantial variation in this toxicity-weighted average, ranging from zero to over 185 million. This ambient HAP concentration measure does not have units per se because it is a toxicity-weighted average across numerous toxic chemicals. As such, we consider it to be more of an ambient HAP index.[11] As discussed in Section 3, the HAPs distribution is highly right-skewed, and contains a large number of zero HAP observations. As such we apply the inverse hyperbolic sine transformation (Bellemare and Wichman 2020). As shown in Figure 4, the transformed HAP variable more closely resembles a normal distribution. Although we do note the slight multi-modal shape of the distribution, with a notable spike at zero.

To identify a causal relationship between ambient HAP levels and maternal health, we must control for other environmental stressors that are potentially correlated with HAPs. More specifically, we control for measures of three criteria air pollutants, and exposure to extreme high temperatures. As shown in Table 1, the average woman is exposed to about 11 $\mu g/m^3$ of fine particulate matter ($PM_{2.5}$) during her pregnancy, and about 41 ppb of ozone and 17 ppb of $NO_2$. On average, just under 3% of the pregnancy period is during an extreme high-temperature day, where the daily average temperature exceeded the $95^{th}$ percentile for warmer months in the climate region where the pregnant women lived. There is noticeable variation in this exposure to high-temperature extremes, ranging from zero to almost half (46%) of the pregnancy period.

[11] See EPA's Risk-Screening Environmental Indicators (RSEI) Methodology (2023) for details.

Figure 4. Distribution of Maternal Exposure to IHS(Toxicity-Weighted Ambient HAPs).




## 5. RESULTS

Next, we present the regression model results for each of our three research questions. First, a series of regression models estimating the association between maternal and neighborhood characteristics and toxicity-weighted ambient HAP concentrations is presented. Then, we present the results of models that attempt to identify the causal effect of HAPs on maternal health, followed by a series of regression models that investigate potential heterogeneous maternal health effects from HAPs.

### *5.1. HAP Exposure and Maternal Characteristics*

Following equation (1), we first assess maternal and locational characteristics associated with exposure to higher ambient HAP concentrations. Models 1 through 6 in Table 2 include conception month and year fixed effects to control for broader trends and seasonal patterns, but differ in terms of what maternal characteristics are included as independent variables. Due to correlations across different types of variables (e.g., race and ethnicity and proxies for income), and our interest in simply identifying statistical associations, we include different categories of variables across the models in Table 2, rather than including all variables in a single model.

Model 1 only includes binary indicators denoting a pregnant women's race and ethnicity. These variables are coded as being mutually exclusive in the births data, and White women is the omitted category. Relative to pregnant White women, Black, Hispanic, and women identifying as another race or ethnicity tend to live in areas with greater ambient HAP levels. Interpreting the magnitude of the estimated associations for binary indicators like those in Model 1 is not straightforward

when the outcome variable is transformed following the inverse hyperbolic sine transformation, but as described by Bellemare and Wichman (2020), the conventional calculations put forth by Halvorsen and Palmquist (1980) and Kennedy (1981) for the natural logarithmic transformation often provide a reasonable approximation. The calculation of the percent change in HAPs ($\%\Delta HAP$) following Halvorsen and Palmquist (1980) is simply $\%\Delta HAP = \left(exp\left(\hat{\alpha}_{1[k]}\right) - 1\right) \times 100$, where $\hat{\alpha}_{1[k]}$ is the corresponding coefficient in the vector $\boldsymbol{\alpha}_1$ from equation (1). Kennedy (1981) proposed a small-bias correction to this derivation, as follows: $\%\Delta HAP = \left(exp\left(\hat{\alpha}_{1[k]} - 0.5\widehat{Var}\left(\alpha_{1[k]}\right)\right) - 1\right) \times 100$, where $\widehat{Var}\left(\alpha_{1[k]}\right)$ is the variance of $\hat{\alpha}_{1[k]}$. Depending on which calculation is used, Model 1 suggests that pregnant Black women are exposed to 38.5% to 41.3% higher ambient HAP levels compared to White pregnant women. Similarly, Hispanic women are exposed to HAP levels that are 26.3% to 27.4% higher, and women identifying as another racial or ethnic group experience levels 39.2% to 41.2% greater, again, relative to White pregnant women.

These clear exposure disparities with respect to race and ethnicity hold even after controlling for how urban or rural a neighborhood is in Model 2. The omitted category is metropolitan areas, and so the results suggest that pregnant women in the most urban, metropolitan areas are exposed to the highest levels of HAPs. The point estimates suggest that HAP levels then monotonically decrease as we move towards more rural areas; suggesting HAP levels that are roughly 75%, 86%, and 93% less, relative to the most urban, metropolitan areas.

The data do not contain information on individual household income or wealth, and so Models 3 through 6 in Table 2 rely on various proxies – maternal education, participation in Medicaid or WIC supplemental programs, and the percent of households in their census tract that are living below the poverty line. Note that companion missing value dummies are included so that, despite missing values (see Table 1) we can retain the full sample of pregnant women and birth events. Models 3 through 6 suggest no statistically significant heterogeneity in HAP exposure based on these proxies for income.

Table 2. Models of Toxicity-Weighted Ambient HAP Exposure.

| | (1) | (2) | (3) | (4) | (5) | (6) |
|---|---|---|---|---|---|---|
| Black | 0.3458* | 0.3429* | | | | |
| | (0.2005) | (0.1834) | | | | |
| Hispanic | 0.2421* | 0.2142* | | | | |
| | (0.1286) | (0.1111) | | | | |
| Other race/ethnicity | 0.3448** | 0.2601** | | | | |
| | (0.1648) | (0.1293) | | | | |
| Race/ethnicity not reported | -0.1258 | -0.2093 | | | | |
| | (0.1795) | (0.1639) | | | | |
| Micropolitan area | | -1.3503*** | -1.3351*** | -1.3570*** | -1.3475*** | -1.4131*** |
| | | (0.4380) | (0.4460) | (0.4412) | (0.4425) | (0.4478) |
| Small town | | -1.9446*** | -1.9115*** | -1.9358*** | -1.9241*** | -1.9924*** |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | (0.4422) | (0.4489) | (0.4441) | (0.4451) | (0.4512) |
| Rural area | | -2.6194*** | -2.6311*** | -2.6554*** | -2.6424*** | -2.7126*** |
| | | (0.4225) | (0.4351) | (0.4304) | (0.4318) | (0.4384) |
| Rural/urban not reported | | 0.8454* | 1.0748** | 1.0339** | 1.0381** | -0.3682 |
| | | (0.4910) | (0.4731) | (0.4528) | (0.4546) | (0.5194) |
| Maternal education: college | | | 0.1286 | | | |
| | | | (0.1227) | | | |
| Missing: Maternal education: college | | | -0.3887** | | | |
| | | | (0.1647) | | | |
| Medicaid | | | | 0.0372 | | |
| | | | | (0.0964) | | |
| Missing: Medicaid | | | | 0.5908** | | |
| | | | | (0.2956) | | |
| WIC | | | | | -0.0890 | |
| | | | | | (0.0892) | |
| Missing: WIC | | | | | -0.2545* | |
| | | | | | (0.1283) | |
| % Poverty in Tract | | | | | | 0.0112 |
| | | | | | | (0.0071) |
| Missing: % Poverty in Tract | | | | | | 1.5754*** |
| | | | | | | (0.3184) |
| Constant | 7.4364*** | 7.7575*** | 7.8445*** | 7.5513*** | 8.0419*** | 7.7122*** |
| | (0.2529) | (0.2827) | (0.3225) | (0.3057) | (0.3440) | (0.3125) |
| Month FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Observations | 1,495,331 | 1,495,331 | 1,495,331 | 1,495,331 | 1,495,331 | 1,495,331 |
| Adjusted R-squared | 0.065 | 0.159 | 0.155 | 0.155 | 0.155 | 0.158 |

Note: Dependent variable is the inverse hyperbolic sine of the toxicity-weighted ambient HAP concentrations. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

### *5.2. Effect of HAPs on Maternal Health*

Now that we have established that Black, Hispanic, and other racial and ethnic groups, as well as pregnant women living in urban areas, tend to be exposed to greater HAP levels, the next question is whether exposure to HAPs translates to adverse effects on maternal health.

First focusing on GH, we estimate the regression models depicted in equations (3), and (6) through (9). The full results are displayed in Table A1 in Appendix A. Model 1 includes no spatial or maternal fixed effects, nor the proposed IV approach, to control for any remaining confounders, but does include the full set of covariates. Models 2 and 3 include tract and maternal fixed effects, respectively; and Model 4 uses the proposed IV approach. Model 5 includes tract fixed effects and

the IV approach. As seen in Table A1, the coefficient estimates for some variables vary across specifications, but a few robust patterns are worth highlighting. First, all else constant and irrespective of HAP exposures, Black pregnant women experience about a one percentage point greater risk of GH compared to White pregnant women (the omitted category). In contrast, Hispanic pregnant women and those identifying as another race or ethnicity experience lower GH risks, all else constant. GH risks seem highest among those giving birth to their first child (the omitted category), and among older women (aged 35-44 years). Although we do not observe an individual's income directly, there is evidence suggesting that lower income populations experience higher baseline risks of GH. All else constant, pregnant women with a college degree experience a lower risk of GH. Participation in USDA's WIC program, and possibly in Medicaid, is associated with a small, roughly 0.2 percentage point increase in GH risk. Living in a Census tract with higher poverty rates is also associated with a greater risk of GH. Lastly, the positive and significant coefficients corresponding to the micropolitan, small town, and rural area indicators suggest that those in more rural areas experience higher baseline risks of GH (compared to the omitted metropolitan area category). We emphasize that these statistical associations should not necessarily be interpreted as causal, but do shed light on who experiences higher baseline risks of GH, irrespective of HAP exposure.

We now turn to the estimated effect of HAPs on GH. The coefficient estimates are presented in Table A1 in Appendix A, but the discussion here focuses on the semi-elasticity and elasticity estimates presented in Table 3, which are calculated following equations (4) and (5), respectively. The base model semi-elasticity estimate suggests that a one percent increase in ambient HAP concentrations is associated with a 0.0026 percentage point increase in the risk of GH, which as shown by the estimated elasticity, corresponds to a 0.048% increase in risk. Both estimates are statistically significant at conventional levels, with $p < 0.01$. Similar results are found in the tract fixed effects specification (Model 2), suggesting that a one percent increase in HAPs corresponds to a 0.0018 percentage point (or 0.033%) increase in the risk of GH.

These results are not robust to the inclusion of maternal fixed effects, but we have concerns regarding the smaller sample, whether there is sufficient within-mother variation in HAPs and GH, and sample selection biases. Estimation of the maternal fixed effects models relies solely on women who experienced two or more births during our study period, and therefore roughly half the sample (50.9%) is disregarded. Additionally, the finding shown in Table A1 that GH risks are highest among first births suggests that pregnant women who experienced GH may be less likely to give birth to additional children in the future. The maternal fixed effects models may suffer from selection bias if they are less likely to include pregnant women who are more vulnerable to GH. In fact, we find that pregnant women included in the maternal fixed effects models are less likely to have experienced GH and GDM. We also find statistically significant differences among pregnant women in the maternal fixed effects models in terms of race, ethnicity, maternal age, marriage and college status, rurality of where they live, and the sociodemographic characteristics of their neighborhood (see Appendix B for details).

Our results are robust to the IV procedure, as demonstrated in Model 4 of Table 3, which suggests that a one percent increase in HAPs leads to a 0.0020 percentage point or 0.036% increase in GH risk. The first stage model results (following equation (8)) are presented in Table A2 in Appendix A. The overall predictive power of our first stage models is reasonable, as suggested by the adjusted R-squares ranging from 0.757 to 0.955. Most importantly, our fugitive emissions-based

instrument is a positive and statistically significant predictor of ambient HAPs concentrations. The corresponding F-statistic is 698.37, suggesting that fugitive-based ambient HAPs is a strong instrument (Stock and Yogo 2005). A Hausman-like test of the endogeneity of our ambient HAPs variable confirms that it is, in fact, endogenous (p = 0.037) and thus supports our use of the IV approach.[12]

As a final robustness check, we estimate a variant of the IV model that includes the Census tract-level spatial fixed effects ($\theta_j$) in both the first and second stages (Model 5 in Table 3). Recall that such a model is advantageous because by conditioning on tract fixed effects, it relaxes some of the assumptions needed for our fugitive-based ambient HAP instrument and a causal interpretation. Put plainly, we need only assume that the timing of ambient HAP increases from fugitive emissions is as good as random, and not necessarily the location. This mitigates concerns that, for example, fugitive emissions may be correlated with unobserved factors associated with more disadvantaged neighborhoods. In contrast to the previous IV model, a Hausman-like test here fails to reject the null hypothesis of exogeneity (p = 0.434), suggesting that the more efficient tract fixed effects model without instrumenting (Model 2) is preferable. Nonetheless, the results or primary interest are robust, suggesting that a one percent increase in HAPs leads to a 0.0020 percentage point or 0.036% increase in the risk of GH. As before, our fugitive emissions-based instrument is positive and statistically significant in the first stage equation (Table A1).

Table 3. Increased Risk of Gestational Hypertension (GH) from HAPs.

| | No FE | Tract FE | Maternal FE | IV | IV & Tract FE |
|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) |
| Semi-elasticity ($\nu$) | 0.0026*** | 0.0018*** | 0.0007 | 0.0020*** | 0.0020*** |
| | (0.0005) | (0.0005) | (0.0007) | (0.0006) | (0.0005) |
| Elasticity ($\xi$) | 0.0476*** | 0.0329*** | 0.0134 | 0.0364*** | 0.0366*** |
| | (0.0099) | (0.0089) | (0.0122) | (0.0102) | (0.0094) |
| Month FE | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Child and Maternal Attributes | Yes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes | |
| Tract FE | | Yes | | | Yes |
| Maternal FE | | | Yes | | |
| Instrument | | | | | |
| IHS(Ambient HAPs) | | | | Yes | Yes |
| Observations | 1,495,331 | 1,495,328 | 734,557 | 1,495,331 | 1,495,328 |

[12] The IV models are estimated using the "ivreghdfe" command in Stata 18 MP. The endogeneity test implemented accommodates heteroskedasticity, but under the assumption of homoskedasticity is numerically equivalent to a Hausman test (Baum et al. 2007).

| | | | | | |
|---|---|---|---|---|---|
| Adjusted R-squared | 0.010 | 0.013 | 0.202 | 0.009 | 0.008 |

Note: Estimated elasticities from equations (4) and (5). See Table A1 in Appendix A for the full regression model results. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

The next set of results focuses on GDM. We assess the factors that are associated with higher baseline risks and whether exposure to HAPs can increase those risks. Recall that this analysis is restricted to births from 2011-2016 due to data limitations. The full regression model results are presented in Table A3 in Appendix A. Before we turn to the HAPs results, some patterns in these baseline GDM risk factors are worth highlighting. For example, although only the tract fixed effects model (Model 2) suggests an increased risk of GDM among Black women, we do see robust evidence of an elevated risk of GDM among Hispanic women and those identifying as another race or ethnicity. Hispanic pregnant women experience an elevated risk of almost 0.02 percentage points, compared to White pregnant women (the omitted category). Women identifying as another racial or ethnic group also experience a 0.02 percentage point greater risk, all else constant. Older pregnant women (aged 35-44 years) are susceptible to a greater risk of GDM. The results generally suggest that less educated, perhaps less wealthy, women experience greater baseline risks of GDM, as suggested by the negative coefficients corresponding to having a college degree, and the positive and significant coefficients corresponding to participation in WIC. The coefficients pertaining to Medicaid enrollment are positive, but statistically insignificant. Similar to the GH results, the positive coefficients on the micropolitan, small town, and rural area indicators suggest that pregnant women living in relatively more rural areas are more inclined to experience GDM.

Now turning to the HAP effects of interest, as seen in Table 4 we find robust evidence that increases in HAPs lead to an increased risk of GDM. The results from Model 1 suggest that a one percent increase in HAPs is associated with a 0.0018 percentage point increase in experiencing GDM, or a roughly 0.033% increase in risk. This result is robust to the inclusion of tract fixed effects, and even mother fixed effects – which suggests a slightly greater 0.048% increase in risk. Although only marginally significant ($p < 0.10$) the semi-elasticity and elasticity estimates from the IV model (Model 4) are similar in magnitude to the earlier results. The statistically significant and positive coefficient corresponding to the fugitive emissions variable in our first stage model results lends support to the assumed instrument (see column (2) in Table A2 of Appendix A), as does the corresponding 302.86 F-statistic, which again suggests that fugitive-based ambient HAPs is a strong instrument (Stock and Yogo 2005). All that said, employing the same Hausman-like test for endogeneity (Baum et al. 2007), we fail to reject the null hypothesis that HAPs can be treated as exogenous ($p = 0.848$), suggesting that the more efficient estimates from Model 1 (Table 4) may be more appropriate in the case of GDM. The IV model with tract fixed effects (Model 5) suggests a smaller and statistically insignificant increase in the risk of GDM, but similar to the previous IV model, a Hausman-like test for endogeneity fails to reject the null hypothesis that HAPs can be treated as exogenous ($p = 0.621$), suggesting again that an IV approach is not needed in this case and preference should be given to the more efficient tract fixed effects model (Model 2).

Table 4. Increased Risk of Gestational Diabetes Mellitus (GDM) from HAPs.

| | No FE (1) | Tract FE (2) | Maternal FE (3) | IV (4) | IV & Tract FE (5) |
|---|---|---|---|---|---|
| Semi-elasticity ($\nu$) | 0.0018** | 0.0017*** | 0.0026*** | 0.0017* | 0.0013 |
| | (0.0007) | (0.0007) | (0.0007) | (0.0010) | (0.0012) |
| Elasticity ($\xi$) | 0.0326** | 0.0320*** | 0.0475*** | 0.0308* | 0.0236 |
| | (0.0131) | (0.0121) | (0.0132) | (0.0186) | (0.0222) |
| Month FE | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Child and Maternal Attributes | Yes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes | |
| Tract FE | | Yes | | | Yes |
| Maternal FE | | | Yes | | |
| Instrument | | | | | |
| IHS(Ambient HAPs) | | | | Yes | Yes |
| Observations | 671,201 | 671,200 | 228,449 | 671,201 | 671,200 |
| Adjusted R-squared | 0.015 | 0.021 | 0.304 | 0.015 | 0.014 |

Note: See Table A3 in Appendix A for the full regression model results. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

We assess the robustness of our results in several ways. First, to confirm that multicollinearity between the environmental stressors – i.e., the toxicity-weighted ambient concentration of HAPs, concentrations of criteria air pollutants, and extreme high temperatures – is not a concern, we first examine the simple pairwise correlations among these variables. In addition, we re-estimate our primary regression models, but exclude $PM_{2.5}$, Ozone, $NO_2$, and the percent of days where temperatures exceeded the 95$^{th}$ percentile. The positive correlation coefficients between IHS(HAPs) and $PM_{2.5}$ and $NO_2$ are 0.34 and 0.38, respectively, and the correlations among some of the criteria air pollutants are also fairly high (see Table C3 in Appendix C). However, our HAP results are similar in magnitude and significance if these other environmental stressors are excluded from the regression models of maternal health outcomes (see Tables C4 and C5 in Appendix C).

Second, as noted in section 4.1.1, the birth records data only included information on GDM for 2011-2016. In contrast, information on GH is included in the birth records data for our entire 2002-2016 study period (except for 2010, which is omitted due to NCSCHS's transition in data reporting procedures and missing GH information). For purposes of comparability to the GDM results, and to ensure consistency in outcome ascertainment and exposure-outcome alignment across models, we re-estimate our GH models using only the sample of pregnant women who gave birth from 2011-2016. As in the original models (Table 3), the base model (Model 1) and IV specification

(Model 4) in Table C6 of Appendix C suggest statistically significant increases in the risk of GH from ambient HAP concentrations, and the magnitude of these results are quite similar to the earlier specifications. The maternal fixed effects model again yields no statistically significant results in terms of the relationship between HAPs and GH. The main difference is that when focusing on only births between 2011 and 2016, the tract fixed effects model suggests point estimates that are slightly smaller in magnitude and no longer statistically significant, likely reflecting reduced statistical power and limited with-in tract variation over the shorter study period. Despite the sensitivity of the tract fixed effects GH models to the assumed study period, we believe it is appropriate to take advantage of all available data when possible and thus have more confidence in the findings from the main analysis in Table 3.

As a third robustness check, we employ alternative functional forms, including those where the ambient HAP concentration enters linearly, as a quadratic relationship, and using a series of discrete bins denoting different ambient HAP levels. The findings are summarized here, but the results are presented in Appendix C (Tables C7 through C12). The linear models of GH yield similar results to those from the main specifications, generally suggesting small but statistically significant increases in the risk of GH with elevated ambient HAP concentrations. The estimated effects on GDM, however, are statistically insignificant under the linear model specification. Given the wide range and skewed HAP distribution, assuming a linear relationship may be overly restrictive. Squared HAP terms are added in a subsequent set of models to allow for a quadratic relationship. The estimated association between GDM and HAPs is again statistically insignificant, but the quadratic models of GH do suggest a nonlinear exposure-response relationship, with increasing risks of GH at higher ambient HAP concentrations and diminishing marginal effects at the upper end of the distribution.

For the last set of models, we create a series of four dummy variables to denote observations with an ambient HAP concentration that is: (i) greater than zero but below the lowest quartile among nonzero HAP observations, (ii) between the first and second quartile, (iii) between the second quartile and the $90^{th}$ percentile, and (iv) above the $90^{th}$ percentile. The omitted category entails observations where the ambient HAP level during the pregnancy is zero. The category thresholds were chosen based on visual "breaks" and noticeable modes in the IHS transformed HAP distribution (see Figure 4). The base and IV models using these binned HAP variables suggest statistically significant increases in the risk of GH, relative to zero ambient HAPs. The estimated effects on GH are greater for higher HAP bins, suggesting that the risks of GH are increasing at higher levels of exposure, and F-tests for the base and IV models reject the null hypothesis that the increase in GH risk is equal across the HAP bins ($p=0.0028$ and $p=0.0502$, respectively). As in our main models, the maternal fixed effects specification suggests null results. In contrast to our earlier models, however, the tract FE results now suggest a negative association between HAPs and the risk of GH. An F-test fails to reject the null hypothesis that these effects are equal across HAP bins. Given this and the perverse negative sign, we speculate that there is insufficient within-tract variation in HAPs when using these coarser bin measures.[13]

[13] To investigate this possibility, we create a 0 to 4 variable, where each integer denotes one of the five HAP bins. We then take the absolute value of the difference between each observation and the tract-specific average. More than 90% of the observations have an absolute difference that is less than one, suggesting that when using the coarser HAP bins there is little within-tract variation.

When applying the binned HAP measures to models of GDM, we see that the most parsimonious model and maternal fixed effects model suggest a positive association between ambient HAP levels and GDM, particularly among the highest HAP bins. The tract fixed effect model suggests no relationship between the binned HAP measures and GDM, but again we believe that there is insufficient within tract variation in HAPs when using these coarser HAP bins. The IV model of GDM when using the binned HAPs variables also suggests a statistically insignificant relationship.

Overall, the robustness of our results to alternative HAP specifications is mixed, with some functional forms suggesting a negligible – or perhaps even no – relationship between HAPs and maternal health. Nonetheless, most of the alternative functional forms support the conclusion that increases in HAPs lead to increases in the likelihood of pregnant women experiencing GH and GDM. This is consistent with our main findings, where the IHS transformation is applied to our ambient HAP measure. We believe that the IHS transformation is the most appropriate given the highly skewed distribution of our ambient HAP measure. Furthermore, we find our results are generally robust across the other sensitivity analyses discussed, leading to the broader takeaway that women exposed to higher ambient HAP concentrations during pregnancy experience an increase in the risk of GH and GDM.

### *5.3. Heterogeneity in Maternal Health Effects*

To assess whether, conditional on the same ambient HAP concentrations, pregnant women from racially and ethnically diverse groups experience more severe adverse health effects, we re-estimate our models but include a series of interaction terms between the ambient HAP variable and three other variables – whether a pregnant woman is "Nonwhite" (i.e., Black, Hispanic, or of another racial or ethnic group); has no college degree (Mother no college = 1 – Mother college); and is enrolled in Medicaid.[14] Differing marginal responses in health could occur due to differences in baseline risks, information and awareness, and the ability of pregnant women to take averting and mitigating actions.

For purposes of this exercise to examine health effect heterogeneity, we focus on our two preferred specifications – the tract fixed effects and IV models. We judge these models to be superior to the other models in Tables 3 and 4 because they invoke approaches to mitigate omitted variable bias without excessively absorbing variation in ambient HAPs or introducing selection bias, as may be the case with the maternal fixed effects models.

We present the coefficients for HAPs and the corresponding interaction terms here, but the full results are available in Tables A4 and A5 of Appendix A. The results for GH are displayed in Table 5 and show little evidence of health effect heterogeneity based on race and economic status (as proxied by maternal education and enrollment in Medicaid). The only statistically significant

[14] We group Black, Hispanic, and the other racial or ethnic group classifications into a single "Nonwhite" (and non-Hispanic) category to ease interpretation and simplify the IV models. The IV approach requires instrumentation and estimation of first-stage equations for each endogenous variable, including each interaction term involving the potentially endogenous ambient HAP variable. It is important to note, however, that this generalization may mask more nuanced differences across racial and ethnic groups.

finding pertains to the IV specification in Model 5, which suggests that less educated women experience greater increases in their risk of GH from HAPs. This is consistent with the hypothesis that less educated, perhaps less wealthy, pregnant women may be less aware of HAP levels, and/or have fewer resources to undertake averting and mitigating behaviors to reduce their individual-level exposure and the resulting health effects. This result is not robust to the tract fixed effects specification, so caution is warranted when interpreting this result.

Table 5. Models of heterogeneity in increased risk of Gestational Hypertension (GH) from HAPs.

| | Tract FE (1) | Tract FE (2) | Tract FE (3) | IV (4) | IV (5) | IV (6) |
|---|---|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0020*** | 0.0018*** | 0.0018*** | 0.0017** | 0.0011 | 0.0018*** |
| | (0.0005) | (0.0005) | (0.0005) | (0.0007) | (0.0007) | (0.0006) |
| × Nonwhite | -0.0004 | | | 0.0007 | | |
| | (0.0003) | | | (0.0007) | | |
| × Mother no college | | -0.0000 | | | 0.0012** | |
| | | (0.0003) | | | (0.0006) | |
| × Medicaid | | | 0.0006 | | | 0.0011 |
| | | | (0.0009) | | | (0.0013) |
| Month FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Birth and Maternal Attributes | Yes | Yes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | | | | Yes | Yes | Yes |
| Tract FE | Yes | Yes | Yes | | | |
| Instrument ln(Ambient HAPs) | | | | Yes | Yes | Yes |
| Observations | 1,495,328 | 1,495,328 | 1,495,328 | 1,495,331 | 1,495,331 | 1,495,331 |
| Adjusted R-squared | 0.015 | 0.013 | 0.013 | 0.009 | 0.009 | 0.009 |

Note: Select coefficient estimates. See Table A4 in Appendix A for the full regression model results. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Now, turning to the GDM results in Table 6, the positive and statistically significant coefficient estimates corresponding to the interaction terms between the ambient HAPs measure and Nonwhite indicator suggest that the increased risk of GDM from HAPs is driven primarily by pregnant women who identify as Black, Hispanic, or of another racial or ethnic group. This result is robust in both the tract fixed effects and IV models (columns (1) and (4)). Otherwise, the results in Table 6 suggest no statistically significant heterogeneity in the effects of HAPs on GDM with respect to maternal education and participation in Medicaid.

Table 6. Models of heterogeneity in increased risk of Gestational Diabetes Mellitus (GDM) from HAPs.

| | Tract FE (1) | Tract FE (2) | Tract FE (3) | IV (4) | IV (5) | IV (6) |
|---|---|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0009 | 0.0023* | 0.0025** | -0.0003 | 0.0012 | 0.0023* |
| | (0.0007) | (0.0012) | (0.0010) | (0.0011) | (0.0018) | (0.0013) |
| × Nonwhite | 0.0019** | | | 0.0043*** | | |
| | (0.0007) | | | (0.0014) | | |
| × Mother no college | | -0.0007 | | | 0.0007 | |
| | | (0.0011) | | | (0.0020) | |
| × Medicaid | | | -0.0015 | | | -0.0013 |
| | | | (0.0013) | | | (0.0018) |
| Month FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Birth and Maternal Attributes | Yes | Yes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | | | | Yes | Yes | Yes |
| Tract FE | Yes | Yes | Yes | | | |
| Instrument ln(Ambient HAPs) | | | | Yes | Yes | Yes |
| Observations | 671,200 | 671,200 | 671,200 | 671,201 | 671,201 | 671,201 |
| Adjusted R-squared | 0.021 | 0.021 | 0.021 | 0.015 | 0.015 | 0.015 |

Note: Select coefficient estimates. See Table A5 in Appendix A for the full regression model results. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

## 6. DISCUSSION

Pregnant women from racial and ethnic minority groups face greater risks of adverse maternal health outcomes, including elevated maternal mortality rates (Hoyert 2023, Leonard et al., 2019, Fleszar et al., 2023). Consistent with the literature, we find that irrespective of HAPs and other environmental stressors, Black women, and those with lower income (as proxied by education level and participation in Medicaid and USDA's WIC program) experience greater risks of GH. Similarly, we find that women identifying as Hispanic or another racial or ethnic group, as well as less educated women and those who participate in USDA's WIC program, face higher risks of GDM. At the same time, we find that pregnant women identifying as Black, Hispanic, or as another racial or ethnic group tend to be exposed to higher ambient HAP concentrations, and that greater ambient HAPs concentrations during pregnancy lead to elevated risks of GH and GDM. Together, these results highlight how ambient HAP exposure exacerbates maternal health disparities.

The next important question is: how large are the effects of HAPs on maternal health? The estimated elasticities and semi-elasticities in Tables 3 and 4 may not appear large, but it is difficult to put the magnitude in context. To better illustrate the size of the results, we consider the following illustrative scenario. Considering all births from 2002 through 2016, the annual average of the

toxicity-weighted ambient HAP concentration during pregnancy decreased from 3052 to 2082 points, a 970 point or 31.8% reduction (see Figure 1).

We can calculate a non-marginal change in the risk of adverse health outcome $Y$ due to a non-marginal change in HAPs ($\partial HAP$) as:

$$\partial Y = \frac{\beta_1 HAP}{\sqrt{HAP^2+1}} \frac{\partial HAP}{HAP} = \frac{\beta_1}{\sqrt{HAP^2+1}} \partial HAP \quad (13)$$

Multiplying the change in risk $\partial Y$ in equation (13) by the corresponding population of pregnant women $N$ yields the change in statistical cases that are estimated to result from a $\partial HAP$ change in ambient HAP concentrations. More formally:

$$\Delta cases = \partial Y \times N = \frac{\beta_1}{\sqrt{HAP^2+1}} \partial HAP \times N \quad (14)$$

There was an average of $N$ = 106,809 births each year in North Carolina during our study period. Based on the results from our tract fixed effects and IV models in Tables 3 and 4, and substituting $\partial HAP = -970$, yields estimates of the average number of statistical cases of GH and GDM prevented each year.

As shown in Table 7, a decrease in average HAP concentrations of this magnitude is estimated to lead to an average decrease in the risk of GH of about 0.06 percentage points, and a decrease in the risk of GDM of about 0.05 to 0.06 percentage points. When multiplied over the average number of births each year, this 31.8% reduction in ambient HAP concentrations corresponds to about 61 to 68 statistical cases of GH and about 57 to 59 statistical cases of GDM prevented each year in North Carolina.

Table 7. Illustrative changes in risk due to observed reduction in average maternal HAP exposure from 2002-2016.

| | Gestational Hypertension | | Gestational Diabetes Mellitus | |
|---|---|---|---|---|
| | Tract FE | IV | Tract FE | IV |
| | (1) | (2) | (3) | (4) |
| Change in risk (∂Y) | -0.0573*** | -0.0632*** | -0.0555*** | -0.0533* |
| | (0.0155) | (0.0177) | (0.0210) | (0.0322) |
| Cases prevented (Δcases) | -61.16*** | -67.52*** | -59.24*** | -56.97* |
| | (16.57) | (18.93) | (22.44) | (34.43) |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | | Yes | | Yes |
| Tract FE | Yes | | Yes | |
| Instrument IHS(HAPs) | | Yes | | Yes |

Note: Based on estimates from Tables 3 and 4. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

## 7. CONCLUSIONS

This study contributes to a nascent literature examining the effects of hazardous air pollutants (HAPs) on maternal health. We utilize a comprehensive measure of ambient HAPs to identify patterns in exposure across the population of pregnant women in North Carolina, and quantify plausibly causal relationships between ambient HAP concentrations and maternal health.

Findings of causality should be interpreted with caution due to several limitations, including the temporal coarseness of our annual ambient HAP concentration data and subsequent weakened ability to establish a clear temporal sequence between HAP exposure and maternal health outcomes. Future research using more temporally fine measures of HAP exposures, and ideally examining heterogeneity across trimesters, would be a valuable direction for investigation. Our data is also limited to pregnancies that resulted in a live birth. It is possible that the estimated effects on GH and GDM are understated if increases in those conditions led to a miscarriage or stillbirth. Such cases are not included in the birth records data analyzed in this study. Finally, as with any empirical analysis of observational data, the potential influence of unobserved factors can confound a causal interpretation. We minimize such confounding factors and facilitate a causal interpretation by including an exhaustive set of control variables, Census tract and maternal fixed effects, and an instrumental variables (IV) approach that exploits variation in intermittent, fugitive emissions. Each of these approaches have their advantages and drawbacks. For example, the maternal fixed effects models control for all time-invariant factors that are specific to each individual woman (e.g., baseline health, behavior, and time-invariant environmental, social, and economic conditions). However, the maternal fixed effects introduce severe sample selection bias by excluding a significant portion of the population of pregnant women (i.e., women who gave birth just once during our study period). The retained sample may not be representative of the broader population of pregnant women. Women who have experienced multiple births may have different health profiles, exposures, and healthcare access, leading to less-generalizable findings. The Census tract fixed effects models are open to similar criticisms, but to a far lesser extent, and are still able to control for unobserved, time-invariant location-specific factors that could otherwise confound the results. The IV approach circumvents the concerns with maternal and spatial fixed effects, but hinges on the assumed exogeneity of the fugitive emissions-based instrument.

Using these various approaches, we estimate the effect of ambient HAP concentrations on the risk of gestational hypertension (GH) and gestational diabetes mellitus (GDM). The testing of multiple outcomes increases the possibility of false positives, but the robustness of our results across specifications and empirical strategies helps guard against spurious findings and supports a plausibly causal interpretation. Our preferred tract fixed effects and IV estimates suggest that a one percent increase in HAPs leads to a roughly 0.03 to 0.04 percent increase in the risk of GH, and a 0.03 percent increase in the risk of GDM. Considering the roughly 32% reduction in average ambient HAPs concentration in North Carolina from 2002 to 2016, we estimate an annual reduction in risk of about 61 to 68 statistical cases of GH, and 57 to 59 statistical cases of GDM. Additionally, we find that Nonwhite pregnant women (i.e., Black, Hispanic, or of another racial or ethnic group) experience greater increases in the risk of GDM from a marginal increase in HAPs, compared to White pregnant women. Such heterogenous effects may stem from nonlinearities in the dose-response relationship and differences in baseline risks, information provision and

awareness, and pregnant women's ability to undertake averting and mitigating actions to reduce their individual-level HAPs exposure and minimize the adverse health effects.

Our results demonstrate that, irrespective of HAP exposure, pregnant women who are Black, Hispanic, or of another racial or ethnic group, as well as those who may have lower income, experience elevated baseline risks of GH and GDM. At the same time, women in some of these groups also experience higher ambient HAP concentrations during pregnancy, which we have shown lead to increases in the risk of GH and GDM. Together, the results demonstrate that HAPs exacerbate maternal health disparities. By the same token, our results suggest that pregnant women who are part of these groups would particularly benefit from programs to reduce HAP exposures.

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## APPENDICES

### *Appendix A. Full Results for Primary Regression Models.*

Table A1. Risk of Gestational Hypertension (GH): Full Regression Model Results.

| | No FE (1) | Tract FE (2) | Maternal FE (3) | IV (4) | IV & Tract FE (5) |
|---|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0026*** | 0.0018*** | 0.0007 | 0.0020*** | 0.0020*** |
| | (0.0005) | (0.0005) | (0.0007) | (0.0006) | (0.0005) |
| % days with temp ≥ 95th pct | -0.0007* | 0.0009*** | 0.0004 | -0.0007* | 0.0008*** |
| | (0.0004) | (0.0002) | (0.0004) | (0.0004) | (0.0002) |
| $PM_{2.5}$ ($\mu g/m^3$) | 0.0001 | 0.0020** | 0.0010 | 0.0004 | 0.0020** |
| | (0.0010) | (0.0008) | (0.0010) | (0.0010) | (0.0008) |
| Ozone (ppb) | 0.0007* | -0.0005 | -0.0001 | 0.0008* | -0.0005 |
| | (0.0004) | (0.0003) | (0.0005) | (0.0004) | (0.0003) |
| $NO^2$ (ppb) | -0.0005*** | 0.0001 | -0.0000 | -0.0005*** | 0.0001 |
| | (0.0001) | (0.0001) | (0.0001) | (0.0001) | (0.0001) |
| Black | 0.0100*** | 0.0113*** | 0.0031 | 0.0100*** | 0.0113*** |
| | (0.0011) | (0.0011) | (0.0095) | (0.0011) | (0.0011) |
| Hispanic | -0.0171*** | -0.0161*** | 0.0055 | -0.0171*** | -0.0161*** |
| | (0.0020) | (0.0023) | (0.0080) | (0.0020) | (0.0023) |
| Other race/ethnicity | -0.0243*** | -0.0216*** | -0.0032 | -0.0243*** | -0.0216*** |
| | (0.0014) | (0.0015) | (0.0068) | (0.0014) | (0.0015) |
| Race/ethnicity not reported | -0.0146*** | -0.0124** | 0.0062 | -0.0148*** | -0.0124** |
| | (0.0053) | (0.0052) | (0.0165) | (0.0053) | (0.0052) |
| Second birth | -0.0372*** | -0.0374*** | -0.0423*** | -0.0372*** | -0.0374*** |
| | (0.0019) | (0.0019) | (0.0028) | (0.0019) | (0.0019) |
| Third birth | -0.0414*** | -0.0416*** | -0.0491*** | -0.0414*** | -0.0416*** |
| | (0.0022) | (0.0022) | (0.0035) | (0.0022) | (0.0022) |
| Fourth birth | -0.0433*** | -0.0435*** | -0.0516*** | -0.0433*** | -0.0435*** |
| | (0.0023) | (0.0023) | (0.0037) | (0.0023) | (0.0023) |
| Fifth or greater birth | -0.0396*** | -0.0399*** | -0.0483*** | -0.0396*** | -0.0399*** |
| | (0.0027) | (0.0027) | (0.0048) | (0.0027) | (0.0027) |
| Mother 15-24 years | -0.0143*** | -0.0145*** | 0.0029 | -0.0144*** | -0.0145*** |
| | (0.0011) | (0.0008) | (0.0017) | (0.0011) | (0.0008) |
| Mother 35-44 years | 0.0137*** | 0.0147*** | -0.0012 | 0.0137*** | 0.0147*** |
| | (0.0009) | (0.0008) | (0.0015) | (0.0009) | (0.0008) |
| Not married | -0.0016 | -0.0021 | -0.0019 | -0.0015 | -0.0021 |
| | (0.0016) | (0.0016) | (0.0021) | (0.0017) | (0.0016) |
| Smoked | -0.0075*** | -0.0088*** | 0.0015 | -0.0075*** | -0.0088*** |
| | (0.0014) | (0.0012) | (0.0023) | (0.0014) | (0.0012) |
| Missing: Smoked | -0.0240*** | -0.0217*** | -0.0207 | -0.0242*** | -0.0217*** |

| | | | | | |
|---|---|---|---|---|---|
| | (0.0063) | (0.0064) | (0.0129) | (0.0063) | (0.0064) |
| Maternal education: college | -0.0188*** | -0.0142*** | 0.0030 | -0.0188*** | -0.0142*** |
| | (0.0013) | (0.0011) | (0.0028) | (0.0013) | (0.0011) |
| Missing: Maternal education: college | -0.0088** | -0.0043 | 0.0017 | -0.0090** | -0.0043 |
| | (0.0036) | (0.0036) | (0.0089) | (0.0036) | (0.0036) |
| WIC | 0.0029*** | 0.0022** | -0.0014 | 0.0028*** | 0.0022** |
| | (0.0009) | (0.0009) | (0.0018) | (0.0009) | (0.0009) |
| Missing: WIC | 0.0054 | 0.0053 | -0.0012 | 0.0052 | 0.0053 |
| | (0.0064) | (0.0063) | (0.0104) | (0.0064) | (0.0063) |
| Medicaid | 0.0025* | 0.0016 | 0.0021 | 0.0025* | 0.0017 |
| | (0.0015) | (0.0015) | (0.0016) | (0.0015) | (0.0015) |
| Missing: Medicaid | -0.0453*** | -0.0443*** | -0.0051 | -0.0450*** | -0.0443*** |
| | (0.0025) | (0.0030) | (0.0121) | (0.0026) | (0.0030) |
| Micropolitan area | 0.0061** | | 0.0022 | 0.0056* | |
| | (0.0029) | | (0.0027) | (0.0030) | |
| Small town | 0.0116*** | | 0.0038 | 0.0109*** | |
| | (0.0040) | | (0.0037) | (0.0041) | |
| Rural area | 0.0144*** | | -0.0036 | 0.0134*** | |
| | (0.0039) | | (0.0056) | (0.0040) | |
| Rural/urban not reported | -0.0905** | | -0.0237* | -0.0907** | |
| | (0.0434) | | (0.0133) | (0.0434) | |
| % Poverty in Tract | 0.0002*** | | 0.0000 | 0.0002*** | |
| | (0.0001) | | (0.0001) | (0.0001) | |
| % Black in Tract | -0.0001 | | -0.0000 | -0.0001 | |
| | (0.0001) | | (0.0000) | (0.0001) | |
| % Hispanic in Tract | -0.0000 | | -0.0001 | -0.0000 | |
| | (0.0001) | | (0.0001) | (0.0001) | |
| Missing: % Poverty in Tract | -0.0330*** | | 0.0084 | -0.0321*** | |
| | (0.0036) | | (0.0069) | (0.0038) | |
| Missing: % Black/Hispanic in Tract | 0.0613 | | 0.0041 | 0.0610 | |
| | (0.0447) | | (0.0075) | (0.0447) | |
| Constant | 0.0657*** | 0.0909*** | 0.0667*** | | |
| | (0.0238) | (0.0088) | (0.0156) | | |
| Month FE | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Tract FE | | Yes | | | Yes |
| Maternal FE | | | Yes | | |
| Instrument ln(Ambient HAPs) | | | | Yes | Yes |
| Observations | 1,495,331 | 1,495,328 | 734,557 | 1,495,331 | 1,495,328 |
| Adjusted R-squared | 0.010 | 0.013 | 0.202 | 0.009 | 0.008 |

Note: Dependent variable is a binary indicator equal to one if the mother was diagnosed with GH, and zero otherwise. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table A2. IV First Stage Results of the Model of IHS(HAP).

| | GH IV 1st Stage (1) | GH IV, Tract FE 1st Stage (2) | GDB IV 1st Stage (3) | GDB IV, Tract FE 1st Stage (4) |
|---|---|---|---|---|
| IHS(Fugitive HAPs) | 0.7707*** | 0.6975*** | 0.7267*** | 0.6199*** |
| | (0.0292) | (0.0429) | (0.0418) | (0.0339) |
| % days with temp ≥ 95th pct | -0.0354*** | -0.0044 | -0.0174 | -0.0101 |
| | (0.0129) | (0.0063) | (0.0111) | (0.0062) |
| $PM_{2.5}$ ($\mu g/m^3$) | 0.1098** | 0.0612*** | -0.0017 | -0.0161 |
| | (0.0509) | (0.0217) | (0.0514) | (0.0147) |
| Ozone (ppb) | 0.0268 | -0.0027 | 0.0468* | 0.0114 |
| | (0.0205) | (0.0061) | (0.0273) | (0.0080) |
| $NO^2$ (ppb) | -0.0078* | 0.0059** | -0.0290** | -0.0027 |
| | (0.0042) | (0.0026) | (0.0116) | (0.0027) |
| Black | -0.0189 | 0.0019 | 0.0190 | 0.0023 |
| | (0.0153) | (0.0041) | (0.0281) | (0.0023) |
| Hispanic | -0.0269 | -0.0006 | 0.0007 | 0.0010 |
| | (0.0173) | (0.0053) | (0.0204) | (0.0023) |
| Other race/ethnicity | 0.0259 | 0.0065 | 0.1267* | 0.0024 |
| | (0.0427) | (0.0104) | (0.0705) | (0.0026) |
| Missing: Race/Ethnicity | -0.0924*** | -0.0126 | 0.0485 | 0.0269 |
| | (0.0322) | (0.0209) | (0.0844) | (0.0471) |
| Second birth | 0.0070 | -0.0008 | 0.0018 | -0.0021 |
| | (0.0073) | (0.0015) | (0.0083) | (0.0019) |
| Third birth | 0.0101 | 0.0008 | 0.0151 | 0.0011 |
| | (0.0103) | (0.0020) | (0.0122) | (0.0015) |
| Fourth birth | 0.0108 | -0.0026 | 0.0158 | -0.0010 |
| | (0.0135) | (0.0038) | (0.0167) | (0.0021) |
| Fifth or greater birth | 0.0124 | 0.0030 | 0.0203 | -0.0010 |
| | (0.0153) | (0.0033) | (0.0209) | (0.0030) |
| Mother 15-24 years | -0.0176 | -0.0055* | -0.0041 | -0.0022 |
| | (0.0313) | (0.0029) | (0.0311) | (0.0019) |
| Mother 35-44 years | -0.0246*** | -0.0054** | -0.0272*** | -0.0017 |
| | (0.0093) | (0.0027) | (0.0103) | (0.0014) |
| Not married | 0.0326 | -0.0037 | 0.0154 | 0.0008 |
| | (0.0352) | (0.0032) | (0.0299) | (0.0013) |
| Smoked | 0.0514*** | 0.0076 | 0.0319 | -0.0019 |
| | (0.0161) | (0.0048) | (0.0211) | (0.0029) |
| Missing: Smoked | -0.1936** | -0.1155* | -0.1291 | -0.0009 |

| | | | | |
|---|---|---|---|---|
| | (0.0777) | (0.0661) | (0.0997) | (0.0377) |
| Maternal education: college | -0.0514 | 0.0039 | -0.0122 | 0.0017 |
| | (0.0337) | (0.0036) | (0.0346) | (0.0014) |
| Missing: Maternal education: college | -0.2090*** | -0.1182 | -0.3010*** | -0.0760 |
| | (0.0597) | (0.0746) | (0.0768) | (0.0658) |
| WIC | 0.0322 | 0.0356** | -0.0199 | 0.0035** |
| | (0.0215) | (0.0150) | (0.0176) | (0.0017) |
| Missing: WIC | -0.1653*** | 0.0229 | -0.1919*** | 0.0035 |
| | (0.0579) | (0.0219) | (0.0464) | (0.0157) |
| Medicaid | 0.0330 | -0.0286 | 0.0351 | 0.0031 |
| | (0.0305) | (0.0193) | (0.0346) | (0.0027) |
| Missing: Medicaid | 0.0578 | -0.0246 | 0.1137** | 0.0114 |
| | (0.0783) | (0.0395) | (0.0483) | (0.0115) |
| Micropolitan area | 0.0974 | | 0.1354 | |
| | (0.1670) | | (0.2236) | |
| Small town | -0.1175 | | -0.2082 | |
| | (0.1791) | | (0.2119) | |
| Rural area | -0.0091 | | -0.2203 | |
| | (0.2113) | | (0.2335) | |
| Missing: Rural/urban | 0.2497 | | -0.1080 | |
| | (0.2754) | | (0.1427) | |
| % Poverty in Tract | 0.0101*** | | 0.0078** | |
| | (0.0030) | | (0.0038) | |
| % Black in Tract | -0.0054*** | | -0.0019 | |
| | (0.0020) | | (0.0027) | |
| % Hispanic in Tract | -0.0087*** | | -0.0024 | |
| | (0.0031) | | (0.0033) | |
| Missing: % Poverty in Tract | 1.2615*** | | 1.4441*** | |
| | (0.1637) | | (0.1970) | |
| Missing: % Black/Hispanic in Tract | -1.5100*** | | -1.2402*** | |
| | (0.1563) | | (0.2197) | |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Tract FE | | Yes | | Yes |
| Observations | 1,495,331 | 1,495,328 | 671,201 | 671,200 |
| Adjusted R-squared | 0.846 | 0.942 | 0.757 | 0.955 |

Note: Dependent variable is the inverse hyperbolic sine of the toxicity-weighted ambient HAP concentrations. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table A3. Risk of Gestational Diabetes Mellitus (GDM): Full Regression Model Results.

| | No FE (1) | Tract FE (2) | Maternal FE (3) | IV (4) | IV & Tract FE (5) |
|---|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0018** | 0.0017*** | 0.0026*** | 0.0017 | 0.0013 |
| | (0.0007) | (0.0007) | (0.0007) | (0.0010) | (0.0012) |
| % days with temp ≥ 95th pct | -0.0005 | -0.0001 | -0.0000 | -0.0005 | -0.0001 |
| | (0.0004) | (0.0003) | (0.0004) | (0.0004) | (0.0003) |
| $PM_{2.5}$ ($\mu g/m^3$) | -0.0015 | 0.0000 | -0.0014 | -0.0015 | 0.0000 |
| | (0.0012) | (0.0006) | (0.0010) | (0.0012) | (0.0006) |
| Ozone (ppb) | 0.0004 | -0.0004 | 0.0002 | 0.0004 | -0.0004 |
| | (0.0007) | (0.0005) | (0.0006) | (0.0007) | (0.0005) |
| $NO^2$ (ppb) | 0.0003 | 0.0002* | 0.0004 | 0.0003 | 0.0002* |
| | (0.0002) | (0.0001) | (0.0003) | (0.0002) | (0.0001) |
| Black | 0.0020 | 0.0038** | -0.0023 | 0.0020 | 0.0038** |
| | (0.0018) | (0.0015) | (0.0196) | (0.0018) | (0.0015) |
| Hispanic | 0.0192*** | 0.0198*** | 0.0026 | 0.0192*** | 0.0198*** |
| | (0.0038) | (0.0040) | (0.0152) | (0.0038) | (0.0040) |
| Other race/ethnicity | 0.0203*** | 0.0205*** | 0.0003 | 0.0203*** | 0.0205*** |
| | (0.0054) | (0.0046) | (0.0139) | (0.0054) | (0.0046) |
| Race/ethnicity not reported | -0.0207 | -0.0155 | 0.0435 | -0.0207 | -0.0155 |
| | (0.0176) | (0.0176) | (0.0703) | (0.0176) | (0.0176) |
| Second birth | -0.0049*** | -0.0050*** | -0.0111*** | -0.0049*** | -0.0050*** |
| | (0.0007) | (0.0007) | (0.0025) | (0.0007) | (0.0007) |
| Third birth | -0.0026*** | -0.0030*** | -0.0132*** | -0.0026*** | -0.0030*** |
| | (0.0009) | (0.0010) | (0.0048) | (0.0009) | (0.0010) |
| Fourth birth | -0.0004 | -0.0010 | -0.0108 | -0.0004 | -0.0010 |
| | (0.0017) | (0.0018) | (0.0077) | (0.0017) | (0.0018) |
| Fifth or greater birth | 0.0035 | 0.0027 | -0.0164 | 0.0035 | 0.0027 |
| | (0.0028) | (0.0031) | (0.0102) | (0.0029) | (0.0031) |
| Mother 15-24 years | -0.0392*** | -0.0392*** | 0.0033 | -0.0392*** | -0.0392*** |
| | (0.0016) | (0.0016) | (0.0025) | (0.0016) | (0.0016) |
| Mother 35-44 years | 0.0404*** | 0.0414*** | -0.0012 | 0.0404*** | 0.0414*** |
| | (0.0029) | (0.0029) | (0.0031) | (0.0029) | (0.0029) |
| Not married | -0.0082*** | -0.0089*** | -0.0059* | -0.0081*** | -0.0089*** |
| | (0.0011) | (0.0010) | (0.0030) | (0.0011) | (0.0010) |
| Smoked | -0.0020 | -0.0030* | -0.0045 | -0.0020 | -0.0030* |
| | (0.0018) | (0.0015) | (0.0047) | (0.0018) | (0.0015) |
| Missing: Smoked | -0.0015 | -0.0010 | -0.0240 | -0.0016 | -0.0011 |
| | (0.0181) | (0.0187) | (0.0838) | (0.0181) | (0.0187) |
| Maternal education: college | -0.0226*** | -0.0182*** | -0.0030 | -0.0226*** | -0.0182*** |
| | (0.0027) | (0.0021) | (0.0091) | (0.0027) | 0.0013 |

| | | | | | |
|---|---|---|---|---|---|
| Missing: Maternal education: college | -0.0034 | -0.0002 | -0.0097 | -0.0034 | (0.0012) |
| | (0.0070) | (0.0074) | (0.0203) | (0.0070) | (0.0074) |
| WIC | 0.0162*** | 0.0160*** | 0.0035 | 0.0161*** | 0.0160*** |
| | (0.0019) | (0.0017) | (0.0025) | (0.0019) | (0.0017) |
| Missing: WIC | 0.0213** | 0.0167** | 0.0225 | 0.0212** | 0.0167** |
| | (0.0094) | (0.0082) | (0.0149) | (0.0095) | (0.0082) |
| Medicaid | 0.0041 | 0.0021 | 0.0037 | 0.0041 | 0.0021 |
| | (0.0031) | (0.0027) | (0.0032) | (0.0031) | (0.0027) |
| Missing: Medicaid | -0.0525*** | -0.0532*** | -0.0079 | -0.0524*** | -0.0532*** |
| | (0.0033) | (0.0036) | (0.0130) | (0.0033) | (0.0036) |
| Micropolitan area | 0.0089*** | | 0.0060 | 0.0089*** | |
| | (0.0030) | | (0.0051) | (0.0031) | |
| Small town | 0.0121** | | 0.0034 | 0.0120* | |
| | (0.0061) | | (0.0065) | (0.0060) | |
| Rural area | 0.0150*** | | -0.0005 | 0.0149*** | |
| | (0.0050) | | (0.0104) | (0.0052) | |
| Rural/urban not reported | -0.2467 | | -0.0134 | -0.2468 | |
| | (0.2485) | | (0.0084) | (0.2484) | |
| % Poverty in Tract | 0.0001 | | 0.0000 | 0.0001 | |
| | (0.0001) | | (0.0001) | (0.0001) | |
| % Black in Tract | -0.0001* | | -0.0000 | -0.0001 | |
| | (0.0001) | | (0.0000) | (0.0001) | |
| % Hispanic in Tract | 0.0001 | | -0.0002* | 0.0001 | |
| | (0.0001) | | (0.0001) | (0.0001) | |
| Missing: % Poverty in Tract | -0.0591*** | | 0.0115 | -0.0590*** | |
| | (0.0045) | | (0.0134) | (0.0046) | |
| Missing: % Black/Hispanic in Tract | 0.2570 | | -0.0120 | 0.2570 | |
| | (0.2484) | | (0.0147) | (0.2483) | |
| Constant | 0.0412 | 0.0601*** | 0.0369* | | |
| | (0.0260) | (0.0176) | (0.0212) | | |
| Month FE | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Tract FE | | Yes | | | Yes |
| Maternal FE | | | Yes | | |
| Instrument ln(Ambient HAPs) | | | | Yes | Yes |
| Observations | 671,201 | 671,200 | 228,449 | 671,201 | 671,200 |
| Adjusted R-squared | 0.015 | 0.021 | 0.304 | 0.015 | 0.014 |

Note: Dependent variable is a binary indicator equal to one if the mother was diagnosed with GDM, and zero otherwise. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table A4. Models of heterogeneity in increased risk of Gestational Hypertension (GH) from HAPs: Full regression model results.

| | Tract FE (1) | Tract FE (2) | Tract FE (3) | IV (4) | IV (5) | IV (6) |
|---|---|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0020*** | 0.0018*** | 0.0018*** | 0.0017** | 0.0011 | 0.0018*** |
| | (0.0005) | (0.0005) | (0.0005) | (0.0007) | (0.0007) | (0.0006) |
| × Nonwhite | -0.0004 | | | 0.0007 | | |
| | (0.0003) | | | (0.0007) | | |
| × Mother no college | | -0.0000 | | | 0.0012** | |
| | | (0.0003) | | | (0.0006) | |
| × Medicaid | | | 0.0006 | | | 0.0011 |
| | | | (0.0009) | | | (0.0013) |
| % days with temp ≥ 95th pct | 0.0009*** | 0.0009*** | 0.0009*** | -0.0007* | -0.0007* | -0.0007* |
| | (0.0002) | (0.0002) | (0.0002) | (0.0004) | (0.0004) | (0.0004) |
| $PM_{2.5}$ ($\mu g/m^3$) | 0.0020** | 0.0020** | 0.0021** | 0.0004 | 0.0004 | 0.0004 |
| | (0.0008) | (0.0008) | (0.0008) | (0.0010) | (0.0010) | (0.0010) |
| Ozone (ppb) | -0.0005 | -0.0005 | -0.0005 | 0.0008* | 0.0008* | 0.0008* |
| | (0.0003) | (0.0003) | (0.0003) | (0.0004) | (0.0004) | (0.0004) |
| $NO^2$ (ppb) | 0.0001 | 0.0001 | 0.0001 | -0.0005*** | -0.0004*** | -0.0004*** |
| | (0.0001) | (0.0001) | (0.0001) | (0.0001) | (0.0001) | (0.0001) |
| Black | 0.0141*** | 0.0113*** | 0.0113*** | 0.0050 | 0.0100*** | 0.0100*** |
| | (0.0024) | (0.0011) | (0.0011) | (0.0051) | (0.0011) | (0.0010) |
| Hispanic | -0.0134*** | -0.0161*** | -0.0161*** | -0.0220*** | -0.0172*** | -0.0171*** |
| | (0.0033) | (0.0023) | (0.0023) | (0.0051) | (0.0020) | (0.0020) |
| Other race/ethnicity | -0.0188*** | -0.0216*** | -0.0216*** | -0.0292*** | -0.0242*** | -0.0242*** |
| | (0.0029) | (0.0015) | (0.0015) | (0.0055) | (0.0015) | (0.0014) |
| Race/ethnicity not reported | -0.0124** | -0.0124** | -0.0124** | -0.0147*** | -0.0148*** | -0.0148*** |
| | (0.0052) | (0.0052) | (0.0052) | (0.0053) | (0.0053) | (0.0053) |
| Second birth | -0.0374*** | -0.0374*** | -0.0374*** | -0.0372*** | -0.0372*** | -0.0372*** |
| | (0.0019) | (0.0019) | (0.0019) | (0.0019) | (0.0019) | (0.0019) |
| Third birth | -0.0416*** | -0.0416*** | -0.0416*** | -0.0414*** | -0.0414*** | -0.0414*** |

| | | | | | | |
|---|---|---|---|---|---|---|
| | (0.0022) | (0.0022) | (0.0022) | (0.0022) | (0.0022) | (0.0022) |
| Fourth birth | -0.0435*** | -0.0435*** | -0.0435*** | -0.0433*** | -0.0433*** | -0.0433*** |
| | (0.0023) | (0.0023) | (0.0023) | (0.0023) | (0.0023) | (0.0023) |
| Fifth or greater birth | -0.0399*** | -0.0399*** | -0.0399*** | -0.0396*** | -0.0397*** | -0.0397*** |
| | (0.0027) | (0.0027) | (0.0027) | (0.0027) | (0.0027) | (0.0027) |
| Mother 15-24 years | -0.0145*** | -0.0145*** | -0.0146*** | -0.0144*** | -0.0144*** | -0.0144*** |
| | (0.0008) | (0.0008) | (0.0008) | (0.0011) | (0.0011) | (0.0011) |
| Mother 35-44 years | 0.0147*** | 0.0147*** | 0.0147*** | 0.0138*** | 0.0138*** | 0.0137*** |
| | (0.0008) | (0.0008) | (0.0008) | (0.0009) | (0.0009) | (0.0009) |
| Not married | -0.0021 | -0.0021 | -0.0021 | -0.0015 | -0.0016 | -0.0015 |
| | (0.0016) | (0.0016) | (0.0016) | (0.0017) | (0.0017) | (0.0017) |
| Smoked | -0.0088*** | -0.0088*** | -0.0088*** | -0.0075*** | -0.0074*** | -0.0074*** |
| | (0.0012) | (0.0012) | (0.0012) | (0.0014) | (0.0014) | (0.0014) |
| Missing: Smoked | -0.0217*** | -0.0217*** | -0.0217*** | -0.0242*** | -0.0243*** | -0.0242*** |
| | (0.0064) | (0.0064) | (0.0064) | (0.0063) | (0.0062) | (0.0062) |
| Maternal education: college | -0.0142*** | -0.0144*** | -0.0141*** | -0.0187*** | -0.0094* | -0.0187*** |
| | (0.0011) | (0.0032) | (0.0011) | (0.0013) | (0.0050) | (0.0013) |
| Missing: Maternal education: college | -0.0043 | -0.0043 | -0.0043 | -0.0089** | -0.0089** | -0.0090** |
| | (0.0036) | (0.0036) | (0.0036) | (0.0036) | (0.0036) | (0.0036) |
| WIC | 0.0022** | 0.0022** | 0.0022** | 0.0030*** | 0.0031*** | 0.0029*** |
| | (0.0009) | (0.0009) | (0.0010) | (0.0009) | (0.0009) | (0.0009) |
| Missing: WIC | 0.0052 | 0.0053 | 0.0053 | 0.0053 | 0.0053 | 0.0053 |
| | (0.0063) | (0.0063) | (0.0063) | (0.0064) | (0.0064) | (0.0064) |
| Medicaid | 0.0016 | 0.0016 | -0.0029 | 0.0026* | 0.0027* | -0.0052 |
| | (0.0015) | (0.0015) | (0.0061) | (0.0015) | (0.0015) | (0.0089) |
| Missing: Medicaid | -0.0444*** | -0.0443*** | -0.0442*** | -0.0450*** | -0.0448*** | -0.0449*** |
| | (0.0030) | (0.0030) | (0.0030) | (0.0027) | (0.0026) | (0.0026) |
| Micropolitan area | | | | 0.0056* | 0.0056* | 0.0058* |
| | | | | (0.0030) | (0.0030) | (0.0030) |
| Small town | | | | 0.0110*** | 0.0111*** | 0.0113*** |
| | | | | (0.0041) | (0.0041) | (0.0041) |
| Rural area | | | | 0.0133*** | 0.0136*** | 0.0139*** |

| | | | | | | |
|---|---|---|---|---|---|---|
| | | | | (0.0040) | (0.0040) | (0.0040) |
| Rural/urban not reported | | | | -0.0911** | -0.0911** | -0.0902** |
| | | | | (0.0435) | (0.0434) | (0.0430) |
| % Poverty in Tract | | | | 0.0002*** | 0.0002*** | 0.0002*** |
| | | | | (0.0001) | (0.0001) | (0.0001) |
| % Black in Tract | | | | -0.0001 | -0.0001 | -0.0001 |
| | | | | (0.0001) | (0.0001) | (0.0001) |
| % Hispanic in Tract | | | | -0.0000 | -0.0000 | -0.0000 |
| | | | | (0.0001) | (0.0001) | (0.0001) |
| Missing: % Poverty in Tract | | | | -0.0322*** | -0.0324*** | -0.0326*** |
| | | | | (0.0037) | (0.0037) | (0.0037) |
| Missing: % Black/Hispanic in Tract | | | | 0.0608 | 0.0611 | 0.0606 |
| | | | | (0.0447) | (0.0447) | (0.0446) |
| Constant | 0.0898*** | 0.0910*** | 0.0907*** | | | |
| | (0.0088) | (0.0091) | (0.0088) | | | |
| Month FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Tract FE | Yes | Yes | Yes | | | |
| Instrument ln(Ambient HAPs) | | | | Yes | Yes | Yes |
| Observations | 1,495,328 | 1,495,328 | 1,495,328 | 1,495,331 | 1,495,331 | 1,495,331 |
| Adjusted R-squared | 0.015 | 0.013 | 0.013 | 0.009 | 0.009 | 0.009 |

Note: Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table A5. Models of heterogeneity in increased risk of Gestational Diabetes Mellitus (GDM) from HAPs: Full regression model results.

| | Tract FE (1) | Tract FE (2) | Tract FE (3) | IV (4) | IV (5) | IV (6) |
|---|---|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0009 | 0.0023* | 0.0025** | -0.0003 | 0.0012 | 0.0023* |
| | (0.0007) | (0.0012) | (0.0010) | (0.0011) | (0.0018) | (0.0013) |
| × Nonwhite | 0.0019** | | | 0.0043*** | | |
| | (0.0007) | | | (0.0014) | | |
| × Mother no college | | -0.0007 | | | 0.0007 | |
| | | (0.0011) | | | (0.0020) | |
| × Medicaid | | | -0.0015 | | | -0.0013 |
| | | | (0.0013) | | | (0.0018) |
| % days with temp ≥ 95th pct | -0.0001 | -0.0001 | -0.0001 | -0.0004 | -0.0005 | -0.0005 |
| | (0.0003) | (0.0003) | (0.0003) | (0.0004) | (0.0004) | (0.0004) |
| $PM_{2.5}$ ($\mu g/m^3$) | 0.0000 | 0.0000 | 0.0000 | -0.0011 | -0.0014 | -0.0015 |
| | (0.0006) | (0.0006) | (0.0006) | (0.0012) | (0.0012) | (0.0012) |
| Ozone (ppb) | -0.0004 | -0.0004 | -0.0004 | 0.0002 | 0.0004 | 0.0004 |
| | (0.0005) | (0.0005) | (0.0005) | (0.0007) | (0.0007) | (0.0007) |
| $NO^2$ (ppb) | 0.0002* | 0.0002* | 0.0002* | 0.0003 | 0.0003* | 0.0003 |
| | (0.0001) | (0.0001) | (0.0001) | (0.0002) | (0.0002) | (0.0002) |
| Black | -0.0094* | 0.0038** | 0.0038** | -0.0280*** | 0.0020 | 0.0020 |
| | (0.0052) | (0.0015) | (0.0016) | (0.0098) | (0.0018) | (0.0018) |
| Hispanic | 0.0068 | 0.0199*** | 0.0198*** | -0.0104 | 0.0192*** | 0.0191*** |
| | (0.0047) | (0.0040) | (0.0040) | (0.0087) | (0.0038) | (0.0039) |
| Other race/ethnicity | 0.0072 | 0.0205*** | 0.0205*** | -0.0099 | 0.0204*** | 0.0203*** |
| | (0.0057) | (0.0045) | (0.0045) | (0.0101) | (0.0053) | (0.0053) |
| Race/ethnicity not reported | -0.0156 | -0.0155 | -0.0156 | -0.0207 | -0.0207 | -0.0207 |
| | (0.0175) | (0.0176) | (0.0176) | (0.0176) | (0.0177) | (0.0176) |
| Second birth | -0.0050*** | -0.0050*** | -0.0049*** | -0.0050*** | -0.0050*** | -0.0049*** |
| | (0.0007) | (0.0007) | (0.0007) | (0.0007) | (0.0007) | (0.0007) |

| | | | | | | |
|---|---|---|---|---|---|---|
| Third birth | -0.0031*** | -0.0030*** | -0.0030*** | -0.0026*** | -0.0026*** | -0.0026*** |
| | (0.0010) | (0.0010) | (0.0010) | (0.0009) | (0.0009) | (0.0009) |
| Fourth birth | -0.0010 | -0.0010 | -0.0010 | -0.0004 | -0.0004 | -0.0004 |
| | (0.0018) | (0.0018) | (0.0018) | (0.0017) | (0.0017) | (0.0017) |
| Fifth or greater birth | 0.0027 | 0.0027 | 0.0027 | 0.0036 | 0.0035 | 0.0036 |
| | (0.0031) | (0.0030) | (0.0030) | (0.0029) | (0.0028) | (0.0028) |
| Mother 15-24 years | -0.0392*** | -0.0392*** | -0.0392*** | -0.0393*** | -0.0392*** | -0.0391*** |
| | (0.0016) | (0.0016) | (0.0016) | (0.0016) | (0.0016) | (0.0016) |
| Mother 35-44 years | 0.0414*** | 0.0414*** | 0.0414*** | 0.0405*** | 0.0404*** | 0.0404*** |
| | (0.0029) | (0.0029) | (0.0029) | (0.0029) | (0.0029) | (0.0029) |
| Not married | -0.0088*** | -0.0088*** | -0.0089*** | -0.0080*** | -0.0082*** | -0.0082*** |
| | (0.0010) | (0.0010) | (0.0009) | (0.0011) | (0.0011) | (0.0010) |
| Smoked | -0.0031** | -0.0031** | -0.0031** | -0.0021 | -0.0019 | -0.0021 |
| | (0.0015) | (0.0015) | (0.0014) | (0.0018) | (0.0018) | (0.0017) |
| Missing: Smoked | -0.0015 | -0.0011 | -0.0012 | -0.0028 | -0.0015 | -0.0018 |
| | (0.0188) | (0.0187) | (0.0187) | (0.0180) | (0.0181) | (0.0181) |
| Maternal education: college | -0.0181*** | -0.0229*** | -0.0183*** | -0.0222*** | -0.0178 | -0.0228*** |
| | (0.0021) | (0.0070) | (0.0021) | (0.0027) | (0.0129) | (0.0025) |
| Missing: Maternal education: college | 0.0001 | -0.0002 | -0.0002 | -0.0027 | -0.0034 | -0.0034 |
| | (0.0074) | (0.0074) | (0.0074) | (0.0071) | (0.0070) | (0.0070) |
| WIC | 0.0160*** | 0.0160*** | 0.0160*** | 0.0161*** | 0.0162*** | 0.0162*** |
| | (0.0017) | (0.0017) | (0.0017) | (0.0019) | (0.0019) | (0.0019) |
| Missing: WIC | 0.0167** | 0.0167** | 0.0168** | 0.0210** | 0.0212** | 0.0214** |
| | (0.0082) | (0.0082) | (0.0081) | (0.0095) | (0.0094) | (0.0094) |
| Medicaid | 0.0021 | 0.0021 | 0.0123 | 0.0042 | 0.0041 | 0.0132 |
| | (0.0027) | (0.0027) | (0.0081) | (0.0031) | (0.0030) | (0.0107) |
| Missing: Medicaid | -0.0527*** | -0.0532*** | -0.0534*** | -0.0514*** | -0.0523*** | -0.0528*** |
| | (0.0035) | (0.0036) | (0.0037) | (0.0033) | (0.0034) | (0.0035) |
| Micropolitan area | | | | 0.0089*** | 0.0089*** | 0.0087*** |
| | | | | (0.0030) | (0.0031) | (0.0030) |
| Small town | | | | 0.0127** | 0.0121** | 0.0115* |
| | | | | (0.0058) | (0.0060) | (0.0059) |

| | | | | | | |
|---|---|---|---|---|---|---|
| Rural area | | | | 0.0145*** | 0.0150*** | 0.0143*** |
| | | | | (0.0050) | (0.0051) | (0.0050) |
| Rural/urban not reported | | | | -0.2490 | -0.2467 | -0.2484 |
| | | | | (0.2498) | (0.2482) | (0.2461) |
| % Poverty in Tract | | | | 0.0001 | 0.0001 | 0.0001 |
| | | | | (0.0001) | (0.0001) | (0.0001) |
| % Black in Tract | | | | -0.0002* | -0.0001 | -0.0001 |
| | | | | (0.0001) | (0.0001) | (0.0001) |
| % Hispanic in Tract | | | | 0.0001 | 0.0001 | 0.0001 |
| | | | | (0.0001) | (0.0001) | (0.0001) |
| Missing: % Poverty in Tract | | | | -0.0590*** | -0.0592*** | -0.0584*** |
| | | | | (0.0046) | (0.0046) | (0.0046) |
| Missing: % Black/Hispanic in Tract | | | | 0.2548 | 0.2567 | 0.2583 |
| | | | | (0.2488) | (0.2475) | (0.2463) |
| Constant | 0.0658*** | 0.0610*** | 0.0549*** | | | |
| | (0.0186) | (0.0185) | (0.0162) | | | |
| Month FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes | Yes |
| Tract FE | Yes | Yes | Yes | | | |
| Instrument ln(Ambient HAPs) | | | | Yes | Yes | Yes |
| Observations | 671,200 | 671,200 | 671,200 | 671,201 | 671,201 | 671,201 |
| Adjusted R-squared | 0.021 | 0.021 | 0.021 | 0.015 | 0.015 | 0.015 |

Note: Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

***Appendix B. Supplemental Analyses of Mother Fixed Effects Models and Sample.***

Estimation of the mother fixed effects (FE) models rely solely on mothers who experienced two or more births during our study period. As such, 62% and 76% of the mothers in our GH and GDM estimating samples, respectively, are excluded when including maternal FE.

Additionally, the birth order coefficient estimates shown in Tables A1 and A3 suggest that GH and GDM risks, respectively, are highest among first births, suggesting that mothers who experienced these adverse health outcomes are less likely to give birth to additional children in the future, and are thus less likely to be included in the maternal FE model samples. This is confirmed by two-sample t-tests of the means. As demonstrated in Table B1, the average rate of GH is 1.3 percentage points lower among mothers who have multiple births and are thus included in the maternal FE sample. The rate of GDM is about a 1.0 percentage point lower. Both differences are statistically significant.

Table B1. Two-sample t-test comparing averages of binary indicators of Gestational hypertension (GH) and Gestational diabetes mellitus (GDM).

| | In Maternal FE Sample: | | |
|---|---|---|---|
| | No | Yes | Difference |
| GH | 0.0611 | 0.0482 | 0.0129** |
| GDM | 0.0577 | 0.0482 | 0.0096*** |

Note: * p<0.10, ** p<0.05, *** p<0.01.

We next estimate a series of cross-sectional Probit models where the dependent variable is an indicator equal to one if a mother is included in the maternal FE sample and zero otherwise. The unit of observation in these cross-sectional models is each mother, rather than each mother-by-birth event. The results are displayed in Table B2. The dependent variable for the models in the first two columns denotes inclusion in the GH maternal FE model, and the last two columns pertain to inclusion in the GDM maternal FE model. All models include characteristics of the mother as explanatory variables. The models in the even numbered columns also include neighborhood characteristics. We find statistically significant differences among mothers in the maternal fixed effects models in terms of race, ethnicity, maternal age, marriage and college status, rurality of where they live, and the sociodemographic characteristics of their neighborhood. Overall, these findings suggest that the maternal fixed effect samples are not representative of the broader population of mothers and births in North Carolina, and so in that sense, models of GH and GDM risks using maternal FE may suffer from a selection bias.

Table B2. Probit models of whether a mother is included in the maternal FE models of GH and GDM.

| | GH Maternal FE Sample | | GDM Maternal FE Sample | |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Black | -0.0221*** | -0.0163*** | -0.1062*** | -0.1011*** |
| | (0.0046) | (0.0051) | (0.0073) | (0.0081) |
| Hispanic | 0.0341*** | 0.0466*** | -0.0760*** | -0.0667*** |
| | (0.0055) | (0.0058) | (0.0090) | (0.0093) |
| Other race/ethnicity | -0.1297*** | -0.1274*** | -0.2165*** | -0.2135*** |
| | (0.0073) | (0.0074) | (0.0103) | (0.0103) |
| Race/ethnicity not reported | -0.4936*** | -0.4866*** | -0.3722* | -0.3686* |
| | (0.0697) | (0.0697) | (0.1965) | (0.1967) |
| Mom 15-24 years | 0.2549*** | 0.2519*** | 0.1579*** | 0.1569*** |
| | (0.0044) | (0.0044) | (0.0069) | (0.0069) |
| Mom 35-44 years | -0.4910*** | -0.4909*** | -0.3777*** | -0.3777*** |
| | (0.0075) | (0.0075) | (0.0114) | (0.0114) |
| Not married | -0.0304*** | -0.0334*** | -0.2489*** | -0.2495*** |
| | (0.0043) | (0.0043) | (0.0071) | (0.0071) |
| Smoked | -0.0003 | -0.0041 | -0.0180* | -0.0199* |
| | (0.0062) | (0.0062) | (0.0104) | (0.0104) |
| Missing: Smoked | -0.2712*** | -0.2713*** | -0.0878 | -0.0900 |
| | (0.0694) | (0.0693) | (0.2278) | (0.2275) |
| Maternal education: college | 0.1509*** | 0.1548*** | 0.2025*** | 0.2034*** |
| | (0.0046) | (0.0047) | (0.0073) | (0.0073) |
| Missing: Maternal education: college | -0.0799* | -0.0761* | -0.1256 | -0.1231 |
| | (0.0441) | (0.0441) | (0.0802) | (0.0802) |
| WIC | -0.1460*** | -0.1480*** | 0.0099 | 0.0081 |
| | (0.0069) | (0.0069) | (0.0076) | (0.0076) |
| Missing: WIC | 0.0020 | 0.0024 | 0.0197 | 0.0206 |
| | (0.0580) | (0.0582) | (0.0652) | (0.0653) |
| Medicaid | 0.0069 | 0.0037 | 0.1365*** | 0.1338*** |
| | (0.0070) | (0.0070) | (0.0076) | (0.0076) |
| Missing: Medicaid | 0.5586*** | 0.5562*** | 0.2052 | 0.2093 |
| | (0.0580) | (0.0581) | (0.1285) | (0.1284) |
| Micropolitan area | | 0.0092* | | 0.0104 |
| | | (0.0051) | | (0.0081) |
| Small town | | 0.0291*** | | 0.0317** |
| | | (0.0088) | | (0.0137) |
| Rural area | | -0.0455*** | | -0.0402** |
| | | (0.0131) | | (0.0193) |
| Rural/urban not reported | | 0.6178 | | |
| | | (0.8942) | | |

| | | | | |
|---|---|---|---|---|
| % Poverty in Tract | | 0.0023*** | | 0.0010*** |
| | | (0.0002) | | (0.0003) |
| % Black in Tract | | -0.0006*** | | -0.0003* |
| | | (0.0001) | | (0.0002) |
| % Hispanic in Tract | | -0.0019*** | | -0.0014*** |
| | | (0.0002) | | (0.0003) |
| Missing: % Poverty in Tract | | -0.6933* | | -0.1345 |
| | | (0.3954) | | (0.6736) |
| Missing: % Black/Hispanic in Tract | | 0.4091 | | |
| | | (0.6243) | | |
| Constant | -0.7376*** | -0.7430*** | -0.7121*** | -0.7107*** |
| | (0.0052) | (0.0057) | (0.0067) | (0.0078) |
| | 617613 | 617613 | 275507 | 275505 |
| Log-likelihood | -385567.8813 | -385450.3514 | -151103.0321 | -151083.8250 |

Note: Robust standard errors in parentheses. * p<0.10, ** p<0.05, *** p<0.01. The rural/urban not reported and missing % black/Hispanic variables are dropped from model 4 because they perfectly predict being in the maternal FE sample.

***Appendix C. Supplemental Robustness Checks and Descriptive Statistics.***

Table C1. Linear Regression Model of Stack-based Ambient HAP Concentrations.

| | No Covariates (1) | No FE (2) | Tract FE (3) | Maternal FE (4) |
|---|---|---|---|---|
| Fugitive HAPs | -2.4962e-06 | -2.2432e-05* | -1.3118e-05*** | -7.3031e-06 |
| | (6.7285e-06) | (1.2870e-05) | (4.3199e-06) | (5.7596e-06) |
| Other covariates | | Yes | Yes | Yes |
| Month FE | | Yes | Yes | Yes |
| Year FE | | Yes | Yes | Yes |
| Tract FE | | | Yes | |
| Maternal FE | | | | Yes |
| Observations | 1,495,331 | 1,495,331 | 1,495,328 | 734,557 |
| Adjusted R-squared | 0.000 | 0.024 | 0.299 | 0.188 |

Note: Dependent variable is the toxicity-weighted ambient HAP concentration based only on smokestack emissions. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C2. Regression Model of IHS of Stack-based Ambient HAP Concentrations.

| | No Covariates (1) | No FE (2) | Tract FE (3) | Maternal FE (4) |
|---|---|---|---|---|
| IHS(Fugitive HAPs) | 0.3751*** | 0.2998*** | 0.0166 | 0.0848* |
| | (0.0888) | (0.0846) | (0.0271) | (0.0445) |
| Other covariates | | Yes | Yes | Yes |
| Month FE | | Yes | Yes | Yes |
| Year FE | | Yes | Yes | Yes |
| Tract FE | | | Yes | |
| Maternal FE | | | | Yes |
| Observations | 1,495,331 | 1,495,331 | 1,495,328 | 734,557 |
| Adjusted R-squared | 0.281 | 0.368 | 0.854 | 0.794 |

Note: Dependent variable is the inverse hyperbolic sine (IHS) of the toxicity-weighted ambient HAP concentration based only on smokestack emissions. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C3. Pairwise correlations among environmental stressors.

| | IHS(HAPs) | % days with temp ≥ 95th pct | $PM_{2.5}$ ($\mu g/m^3$) | Ozone (ppb) | $NO^2$ (ppb) |
|---|---|---|---|---|---|
| IHS(HAPs) | 1 | | | | |
| % days with temp ≥ 95th pct | -0.0181 | 1 | | | |
| $PM_{2.5}$ ($\mu g/m^3$) | 0.3403 | -0.1245 | 1 | | |
| Ozone (ppb) | 0.1647 | 0.1387 | 0.4632 | 1 | |
| $NO^2$ (ppb) | 0.3803 | -0.0576 | 0.4917 | 0.1341 | 1 |

Table C4. Risk of Gestational Hypertension (GH): Full regression model results, excluding other environmental stressors.

| | No FE (1) | Tract FE (2) | Maternal FE (3) | IV (4) |
|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0024*** | 0.0020*** | 0.0008 | 0.0017*** |
| | (0.0005) | (0.0005) | (0.0007) | (0.0006) |
| Black | 0.0093*** | 0.0114*** | 0.0031 | 0.0094*** |
| | (0.0010) | (0.0011) | (0.0095) | (0.0010) |
| Hispanic | -0.0178*** | -0.0160*** | 0.0055 | -0.0177*** |
| | (0.0018) | (0.0022) | (0.0080) | (0.0019) |
| Other race/ethnicity | -0.0249*** | -0.0215*** | -0.0031 | -0.0247*** |
| | (0.0014) | (0.0015) | (0.0068) | (0.0014) |
| Race/ethnicity not reported | -0.0156*** | -0.0124** | 0.0061 | -0.0157*** |
| | (0.0055) | (0.0052) | (0.0165) | (0.0055) |
| Second birth | -0.0371*** | -0.0374*** | -0.0423*** | -0.0371*** |
| | (0.0020) | (0.0019) | (0.0028) | (0.0020) |
| Third birth | -0.0412*** | -0.0416*** | -0.0490*** | -0.0412*** |
| | (0.0023) | (0.0022) | (0.0035) | (0.0023) |
| Fourth birth | -0.0431*** | -0.0435*** | -0.0516*** | -0.0431*** |
| | (0.0024) | (0.0023) | (0.0037) | (0.0024) |
| Fifth or greater birth | -0.0394*** | -0.0399*** | -0.0482*** | -0.0395*** |
| | (0.0027) | (0.0027) | (0.0048) | (0.0027) |
| Mother 15-24 years | -0.0141*** | -0.0145*** | 0.0029 | -0.0142*** |
| | (0.0012) | (0.0008) | (0.0018) | (0.0012) |
| Mother 35-44 years | 0.0135*** | 0.0147*** | -0.0012 | 0.0136*** |
| | (0.0009) | (0.0008) | (0.0015) | (0.0009) |
| Not married | -0.0019 | -0.0020 | -0.0019 | -0.0017 |
| | (0.0016) | (0.0016) | (0.0021) | (0.0017) |
| Smoked | -0.0072*** | -0.0088*** | 0.0015 | -0.0073*** |
| | (0.0015) | (0.0012) | (0.0023) | (0.0015) |

| | | | | |
|---|---|---|---|---|
| Missing: Smoked | -0.0239*** | -0.0219*** | -0.0206 | -0.0242*** |
| | (0.0062) | (0.0064) | (0.0129) | (0.0061) |
| Maternal education: college | -0.0199*** | -0.0142*** | 0.0030 | -0.0197*** |
| | (0.0015) | (0.0011) | (0.0028) | (0.0015) |
| Missing: Maternal education: college | -0.0097** | -0.0044 | 0.0016 | -0.0100*** |
| | (0.0037) | (0.0035) | (0.0089) | (0.0037) |
| WIC | 0.0031*** | 0.0022** | -0.0014 | 0.0029*** |
| | (0.0010) | (0.0009) | (0.0018) | (0.0010) |
| Missing: WIC | 0.0049 | 0.0057 | -0.0011 | 0.0047 |
| | (0.0064) | (0.0063) | (0.0104) | (0.0064) |
| Medicaid | 0.0025* | 0.0015 | 0.0021 | 0.0025* |
| | (0.0014) | (0.0015) | (0.0016) | (0.0014) |
| Missing: Medicaid | -0.0457*** | -0.0447*** | -0.0053 | -0.0453*** |
| | (0.0025) | (0.0031) | (0.0121) | (0.0026) |
| Micropolitan area | 0.0070** | | 0.0021 | 0.0061* |
| | (0.0030) | | (0.0027) | (0.0031) |
| Small town | 0.0137*** | | 0.0035 | 0.0124*** |
| | (0.0041) | | (0.0036) | (0.0042) |
| Rural area | 0.0170*** | | -0.0042 | 0.0153*** |
| | (0.0038) | | (0.0054) | (0.0039) |
| Rural/urban not reported | -0.0928** | | -0.0236* | -0.0931** |
| | (0.0436) | | (0.0134) | (0.0437) |
| % Poverty in Tract | 0.0002*** | | 0.0000 | 0.0002*** |
| | (0.0001) | | (0.0001) | (0.0001) |
| % Black in Tract | -0.0001 | | -0.0000 | -0.0001 |
| | (0.0001) | | (0.0000) | (0.0001) |
| % Hispanic in Tract | -0.0001 | | -0.0001 | -0.0001 |
| | (0.0001) | | (0.0001) | (0.0001) |
| Missing: % Poverty in Tract | -0.0353*** | | 0.0084 | -0.0343*** |
| | (0.0035) | | (0.0066) | (0.0037) |
| Missing: % Black/Hispanic in Tract | 0.0612 | | 0.0049 | 0.0613 |
| | (0.0450) | | (0.0076) | (0.0450) |
| Constant | 0.0910*** | 0.0937*** | 0.0745*** | |
| | (0.0076) | (0.0064) | (0.0122) | |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument ln(Ambient HAPs) | | | | Yes |
| Observations | 1,495,331 | 1,495,328 | 734,557 | 1,495,331 |
| Adjusted R-squared | 0.010 | 0.013 | 0.202 | 0.009 |

Note: Dependent variable is a binary indicator equal to one if the mother was diagnosed with GH, and zero otherwise. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C5. Risk of Gestational Diabetes Mellitus (GDM): Full regression model results, excluding other environmental stressors.

| | No FE (1) | Tract FE (2) | Maternal FE (3) | IV (4) |
|---|---|---|---|---|
| IHS(Ambient HAPs) | 0.0020** | 0.0017** | 0.0027*** | 0.0020* |
| | (0.0008) | (0.0007) | (0.0007) | (0.0010) |
| Black | 0.0020 | 0.0039** | -0.0022 | 0.0020 |
| | (0.0018) | (0.0016) | (0.0196) | (0.0018) |
| Hispanic | 0.0193*** | 0.0199*** | 0.0026 | 0.0193*** |
| | (0.0038) | (0.0040) | (0.0153) | (0.0038) |
| Other race/ethnicity | 0.0202*** | 0.0206*** | 0.0003 | 0.0202*** |
| | (0.0054) | (0.0046) | (0.0139) | (0.0054) |
| Race/ethnicity not reported | -0.0205 | -0.0155 | 0.0441 | -0.0205 |
| | (0.0176) | (0.0176) | (0.0703) | (0.0176) |
| Second birth | -0.0050*** | -0.0050*** | -0.0112*** | -0.0050*** |
| | (0.0007) | (0.0007) | (0.0025) | (0.0007) |
| Third birth | -0.0027*** | -0.0030*** | -0.0133*** | -0.0027*** |
| | (0.0009) | (0.0010) | (0.0048) | (0.0009) |
| Fourth birth | -0.0005 | -0.0010 | -0.0108 | -0.0005 |
| | (0.0017) | (0.0018) | (0.0077) | (0.0017) |
| Fifth or greater birth | 0.0034 | 0.0027 | -0.0164 | 0.0034 |
| | (0.0029) | (0.0031) | (0.0102) | (0.0029) |
| Mother 15-24 years | -0.0393*** | -0.0392*** | 0.0033 | -0.0393*** |
| | (0.0016) | (0.0016) | (0.0025) | (0.0016) |
| Mother 35-44 years | 0.0405*** | 0.0414*** | -0.0013 | 0.0405*** |
| | (0.0030) | (0.0029) | (0.0031) | (0.0030) |
| Not married | -0.0081*** | -0.0088*** | -0.0060* | -0.0081*** |
| | (0.0011) | (0.0010) | (0.0030) | (0.0011) |
| Smoked | -0.0021 | -0.0030* | -0.0045 | -0.0021 |
| | (0.0018) | (0.0015) | (0.0047) | (0.0018) |
| Missing: Smoked | -0.0020 | -0.0012 | -0.0237 | -0.0020 |
| | (0.0181) | (0.0187) | (0.0840) | (0.0181) |
| Maternal education: college | -0.0225*** | -0.0182*** | -0.0030 | -0.0225*** |
| | (0.0028) | (0.0021) | (0.0091) | (0.0028) |
| Missing: Maternal education: college | -0.0034 | -0.0002 | -0.0095 | -0.0034 |
| | (0.0070) | (0.0074) | (0.0202) | (0.0070) |
| WIC | 0.0161*** | 0.0160*** | 0.0035 | 0.0161*** |
| | (0.0019) | (0.0017) | (0.0024) | (0.0019) |
| Missing: WIC | 0.0211** | 0.0167** | 0.0226 | 0.0211** |
| | (0.0093) | (0.0082) | (0.0149) | (0.0093) |
| Medicaid | 0.0044 | 0.0021 | 0.0037 | 0.0044 |

| | | | | |
|---|---|---|---|---|
| | (0.0031) | (0.0027) | (0.0032) | (0.0031) |
| Missing: Medicaid | -0.0518*** | -0.0532*** | -0.0080 | -0.0518*** |
| | (0.0032) | (0.0036) | (0.0129) | (0.0031) |
| Micropolitan area | 0.0083*** | | 0.0061 | 0.0083*** |
| | (0.0031) | | (0.0052) | (0.0032) |
| Small town | 0.0117* | | 0.0033 | 0.0118* |
| | (0.0061) | | (0.0065) | (0.0061) |
| Rural area | 0.0164*** | | -0.0003 | 0.0165*** |
| | (0.0048) | | (0.0103) | (0.0050) |
| Rural/urban not reported | -0.2452 | | -0.0115 | -0.2452 |
| | (0.2477) | | (0.0085) | (0.2476) |
| % Poverty in Tract | 0.0001 | | 0.0000 | 0.0001 |
| | (0.0001) | | (0.0001) | (0.0001) |
| % Black in Tract | -0.0002* | | 0.0000 | -0.0002* |
| | (0.0001) | | (0.0000) | (0.0001) |
| % Hispanic in Tract | 0.0001 | | -0.0002* | 0.0001 |
| | (0.0001) | | (0.0001) | (0.0001) |
| Missing: % Poverty in Tract | -0.0596*** | | 0.0138 | -0.0597*** |
| | (0.0046) | | (0.0133) | (0.0047) |
| Missing: % Black/Hispanic in Tract | 0.2576 | | -0.0139 | 0.2576 |
| | (0.2474) | | (0.0146) | (0.2473) |
| Constant | 0.0435*** | 0.0465*** | 0.0373*** | |
| | (0.0052) | (0.0045) | (0.0091) | |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument ln(Ambient HAPs) | | | | Yes |
| Observations | 671,201 | 671,200 | 228,449 | 671,201 |
| Adjusted R-squared | 0.015 | 0.021 | 0.304 | 0.015 |

Note: Dependent variable is a binary indicator equal to one if the mother was diagnosed with GDM, and zero otherwise. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C6. Increased Risk of Gestational Hypertension (GH) from HAPs: Limiting sample to only birth from 2011-2016.

| | No FE | Tract FE | Maternal FE | IV |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Semi-elasticity | 0.0025*** | 0.0011 | 0.0003 | 0.0021** |
| | (0.0009) | (0.0008) | (0.0011) | (0.0009) |
| Elasticity | 0.0461*** | 0.0203 | 0.0046 | 0.0383** |
| | (0.0172) | (0.0146) | (0.0200) | (0.0165) |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument IHS(Ambient HAPs) | | | | Yes |
| Observations | 671,201 | 671,200 | 228,449 | 671,201 |
| Adjusted R-squared | 0.009 | 0.013 | 0.223 | 0.008 |

Note: Estimated elasticities from equations (4) and (5). Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C7. Risk of Gestational Hypertension (GH): Linear HAPs Specification Regression Model Results.

| | No FE | Tract FE | Maternal FE | IV |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ambient HAPs | 8.0106e-10*** | 4.5303e-10*** | 2.6697e-10 | 7.9807e-10*** |
| | (1.8800e-10) | (6.5108e-11) | (2.1823e-10) | (1.8571e-10) |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument Ambient HAPs | | | | Yes |
| Observations | 1,495,331 | 1,495,328 | 734,557 | 1,495,331 |
| Adjusted R-squared | 0.010 | 0.013 | 0.202 | 0.009 |

Note: Regression coefficient estimates. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C8. Risk of Gestational Diabetes Mellitus (GDM): Linear HAPs Specification Regression Model Results.

| | No FE | Tract FE | Maternal FE | IV |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ambient HAPs | 7.9853e-08 | 3.5683e-08 | -1.1331e-07 | 6.4119e-08 |
| | (4.9758e-08) | (2.7173e-08) | (7.7497e-08) | (4.2920e-08) |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument Ambient HAPs | | | | Yes |
| Observations | 671,201 | 671,200 | 228,449 | 671,201 |
| Adjusted R-squared | 0.015 | 0.021 | 0.304 | 0.015 |

Note: Regression coefficient estimates. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C9. Risk of Gestational Hypertension (GH): Quadratic HAPs Specification Regression Model Results.

| | No FE | Tract FE | Maternal FE |
|---|---|---|---|
| | (1) | (2) | (3) |
| Ambient HAPs | 2.9323e-09*** | 1.9015e-09*** | 1.8990e-09*** |
| | (3.2631e-10) | (4.6151e-10) | (4.4078e-10) |
| Ambient HAPs^2 | -1.8493e-17*** | -1.1955e-17*** | -1.3599e-17*** |
| | (2.2942e-18) | (2.4837e-18) | (1.6918e-18) |
| Month FE | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes |
| Tract FE | | Yes | |
| Maternal FE | | | Yes |
| Instrument Ambient HAPs | | | |
| Observations | 1,495,331 | 1,495,328 | 734,557 |
| Adjusted R-squared | 0.010 | 0.013 | 0.202 |

Note: Regression coefficient estimates. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01. Corresponding IV model of GH risk could not be estimated when including quadratic HAPs term due to multicollinearity issues between the endogenous variable and the instrument.

Table C10. Risk of Gestational Diabetes Mellitus (GDM): Quadratic HAPs Specification Regression Model Results.

| | No FE | Tract FE | Maternal FE | IV |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Ambient HAPs | 1.8506e-07 | 1.5571e-08 | -1.0870e-07 | 1.0275e-07 |
| | (1.2181e-07) | (8.5075e-08) | (1.4473e-07) | (1.0220e-07) |
| Ambient HAPs^2 | -3.6035e-13 | 4.7121e-14 | -1.2656e-14 | -1.2834e-13 |
| | (2.8159e-13) | (1.4786e-13) | (3.2742e-13) | (1.9523e-13) |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument Ambient HAPs | | | | Yes |
| Observations | 671,201 | 671,200 | 228,449 | 671,201 |
| Adjusted R-squared | 0.015 | 0.021 | 0.304 | 0.015 |

Note: Regression coefficient estimates. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01.

Table C11. Risk of Gestational Hypertension (GH): Binned HAP Specification Regression Model Results.

| | No FE (1) | Tract FE (2) | Maternal FE (3) | IV (4) |
|---|---|---|---|---|
| HAPs Bin 1 | 0.0130*** | -0.0112*** | -0.0031 | 0.0202* |
| | (0.0037) | (0.0034) | (0.0103) | (0.0114) |
| HAPs Bin 2 | 0.0133*** | -0.0111*** | -0.0044 | 0.0254** |
| | (0.0042) | (0.0036) | (0.0104) | (0.0114) |
| HAPs Bin 3 | 0.0187*** | -0.0113*** | -0.0057 | 0.0213* |
| | (0.0052) | (0.0039) | (0.0106) | (0.0119) |
| HAPs Bin 4 | 0.0260*** | -0.0070 | -0.0042 | 0.0330*** |
| | (0.0049) | (0.0043) | (0.0105) | (0.0114) |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument IHS(Ambient HAPs) | | | | Yes |
| Observations | 1,495,331 | 1,495,328 | 734,557 | 1,495,331 |
| Adjusted R-squared | 0.010 | 0.013 | 0.202 | 0.009 |

Note: Regression coefficient estimates. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01. The omitted category is a toxicity-weighted ambient HAP concentration of zero. The HAP bin categories are indicator variables equal to one if the ambient HAP concentration is: (1) greater than zero but below the lowest quartile among nonzero HAP observations, (2) between the first and second quartile, (3) between the second quartile and the 90$^{th}$ percentile, or (4) above the 90$^{th}$ percentile, respectively, and zero otherwise.

Table C12. Risk of Gestational Diabetes Mellitus (GDM): Binned HAP Specification Regression Model Results.

| | No FE | Tract FE | Maternal FE | IV |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| HAPs Bin 1 | 0.0068 | 0.0084 | 0.0120 | -0.0872 |
| | (0.0063) | (0.0071) | (0.0074) | (0.0980) |
| HAPs Bin 2 | 0.0029 | 0.0096 | 0.0149* | -0.1042 |
| | (0.0065) | (0.0072) | (0.0078) | (0.0985) |
| HAPs Bin 3 | 0.0163** | 0.0110 | 0.0177** | -0.0712 |
| | (0.0069) | (0.0072) | (0.0077) | (0.0980) |
| HAPs Bin 4 | 0.0155* | 0.0075 | 0.0213** | -0.0868 |
| | (0.0078) | (0.0074) | (0.0088) | (0.0984) |
| Month FE | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes |
| Child and Parent Attributes | Yes | Yes | Yes | Yes |
| Neighborhood Attributes | Yes | | Yes | Yes |
| Tract FE | | Yes | | |
| Maternal FE | | | Yes | |
| Instrument IHS(Ambient HAPs) | | | | Yes |
| Observations | 671,201 | 671,200 | 228,449 | 671,201 |
| Adjusted R-squared | 0.015 | 0.021 | 0.304 | 0.014 |

Note: Regression coefficient estimates. Standard errors in parentheses, clustered at the county level. * p<0.10, ** p<0.05, *** p<0.01. The omitted category is a toxicity-weighted ambient HAP concentration of zero. The HAP bin categories are indicator variables equal to one if the ambient HAP concentration is: (1) greater than zero but below the lowest quartile among nonzero HAP observations, (2) between the first and second quartile, (3) between the second quartile and the 90$^{th}$ percentile, or (4) above the 90$^{th}$ percentile, respectively, and zero otherwise.