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## **The Effects of Short-Term, In Utero Lead Exposure on Birth Outcomes by Trimester: Quasi-Experimental Evidence from NASCAR's Deleading Policy**

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**ABSTRACT:** The objective of this paper is to estimate the effects, both aggregated and disaggregated by trimester, of in utero lead exposure on birth outcomes. These outcomes are not well understood. Our identification strategy exploits National Association for Stock Car Racing's (NASCAR) voluntary switch from leaded to unleaded racing fuel in 2007, more than a decade after the U.S. had otherwise banned leaded gasoline for on-road use. We examine 147,673 births from the Charlotte-Concord-Gastonia Metropolitan Statistical Area in North Carolina from 2004-2009 and use a quasi-experiment exploiting the NASCAR switch from leaded to unleaded racing fuel in 2007. We use a multivariate regression model, controlling for potentially confounding variables, combined with coarsened exact matching, and estimate a difference-in-differences model to obtain the average treatment effect on the treated from the deleading policy on birth outcomes around the Charlotte Motor Speedway.

Our results indicate that after deleading, children born to mothers residing within 4,000 meters of the racetrack experienced an average increase in birth weight of 103.9 grams and gestational age by 0.36 weeks. The probability of low birth weight declined by 4.1 percentage points, preterm births by 2.7 percentage points, and small for gestational age by 4.1 percentage points. Exploiting variation in the timing of racing events across trimesters revealed heterogeneous effects for birth weight, gestational age, and preterm birth, with first trimester exposures having the largest impact. The implication of our results is that reducing exposure to even short-term lead emissions can significantly improve infant health outcomes.

**KEYWORDS:** Birth outcomes, infant health, lead exposure, quasi-experiment

**JEL CODES:** I18, Q53

## **DISCLAIMER**

The views expressed in this paper are those of the author(s) and do not necessarily represent those of the U.S. Environmental Protection Agency (EPA). In addition, although the research described in this paper may have been funded entirely or in part by the U.S EPA, it has not been subjected to the Agency's required peer and policy review. No official Agency endorsement should be inferred.

# The Effects of Short-Term, In Utero Lead Exposure on Birth Outcomes by Trimester: Quasi-Experimental Evidence from NASCAR's Deleading Policy\*

Linda TM Bui<sup>†</sup>, Ron Shadbegian<sup>‡</sup>, Alicia Marquez<sup>§</sup>, Heather Klemick<sup>\*\*</sup>, Dennis Guignet<sup>††</sup>

## Introduction

U.S. regulatory policies developed during the 1970s restricting lead in products including residential paint, pipes, solder, and especially, gasoline, played a central role in reducing blood lead levels (BLL) in the American population. Children, however, remain at risk for lead poisoning. Cognitive and behavioral deficits associated with childhood lead exposure, even at low to moderate levels, are well documented (Aizer et al (2018), Bellinger et al (1992), Evens et al (2015), Hu et al (2006), Landrigan et al (2018), Lanphear et al (2005), Magzamen et al (2015)). There is also a growing body of evidence suggesting that those effects persist, and may have implications for the cognitive development, well-being, and general long-term health of the child that may extend into adulthood (Bellinger et al (1992), Mazumdar et al (2015), Needleman et al (1990), Reuben et al (2017), Shadbegian et al (2019)). Less well understood, however, are the health effects associated with in utero exposure. The latter is an important pathway to consider, as there is evidence of a weak placental-fetal barrier to lead transport, suggesting that maternal lead exposure could affect the fetus. Studies of maternal and umbilical cord BLL have estimated fetal-maternal BLL ratios ranging between 0.6-0.9 (Bellinger & Needleman (1985), Goyer (1990)).

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High maternal BLL ( $\geq 30 \mu\text{g/dL}$ ), usually due to occupational exposure, is associated with an increased risk of spontaneous abortion and stillbirth (Hertz-Picciotto (2000), Centers for Disease Control and Prevention (2010), Troesken (2008)). At lower levels of prenatal exposure, the impact on adverse health outcomes is less certain (Bellinger & Needleman (1985), Center for Disease Control and Prevention (14), Andrews et al (1994)). A few studies examine the relationship between maternal BLL and birth outcomes, in particular birth weight, preterm (or gestational age), and small for gestational age; but notably, no consensus has emerged (Bellinger & Needleman (1985), Centers for Disease Control and Prevention (2010), Andrews et al (1994), Zhu et al (2010), Rabito et al (2014), Jelliffe-Pawlowski et al (2006)). The United States Environmental Protection Agency's (EPA) 2013 *Integrated Science Assessment for Lead* concludes that the evidence linking lead exposure to adverse birth outcomes is "suggestive of a causal relationship," but insufficient from which to infer a causal or likely causal relationship.

Our objective is to estimate the effects, both aggregated and disaggregated by trimester, of in utero lead exposure on various measures of birth weight and gestational age. Our identification strategy exploits NASCAR's voluntary switch from leaded to unleaded racing fuel in 2007, more than a decade after the U.S. had otherwise banned leaded gasoline for on-road use. We believe this is the first study to estimate the short-term effects of in utero exposure to ambient lead on birth outcomes, decomposed by trimester of exposure.

## **Methods**

*Data:* This research was conducted under an agreement with the Children's Environmental Health Initiative (CEHI), according to a research protocol approved by Rice University's institutional review board. We use data from individual birth certificates which includes birth date, weight, gestational age, birth order, and sex. Parental characteristics include age, education, race, and a self-reported measure of whether the mother smoked during pregnancy. We use the mother's place of residence at time of birth to link birth records to Census tract level socio-demographic information taken from the 2000 and 2010

Decennial Census and the 2006-2010 American Community Survey. Data from the Toxics Release Inventory (TRI) for the years 2005-2009 (<https://www.epa.gov/toxics-release-inventory-tri-program/tri-basic-data-files-calendar-years-1987-2019>) are used to identify all TRI reporting facilities in North Carolina that emit lead or lead compounds. Public and private airports where single piston engine planes, potentially using leaded aviation gasoline, were identified using data taken from the year 2005 from NC OneMap (<http://nconemap.gov>).

*Background:* A typical NASCAR event occurs over a single week and consists of multiple races. For a *single* NASCAR race, upwards of 10,000 gallons of racing fuel are consumed: 6,500 – 7,500 gallons of fuel are consumed during the race, with an additional 1,500 – 2,500 gallons used during qualifying laps (Mallory (2017)). At a mid-level lead content of 4 grams/gallon (“High Octane Fuels, High Octane Gas Comparison Chart” (2020), “Racing Fuel Comparisons” (2020)), as much as 40 kg of lead could be released as a result of a single racing event. As a point of reference, in 2005, the second largest U.S. point source for lead emissions was a Missouri lead recycling facility that had an average daily release of 30.82 kg (Schmidt (2010)).

Three studies provide evidence that lead emissions from racing events could increase the lead exposure to nearby residents. The first, a 2017 Industrial Hygiene Assessment Report, measures levels of airborne lead at air monitoring stations located around the Portland International Raceway (PIR), located in Portland, Oregon, during and immediately after the Sportscar Vintage Racing Association (SVRA) Portland Vintage Racing Festival on July 28, 2017, which used leaded racing fuel (Industrial Hygiene Assessment Report (2017)). The Report finds ambient lead levels to be up to 232% higher than a baseline taken at a Sports Car Club of America Track Night event on August 11, 2017, which was also held at PIR, but that did not use leaded gasoline. We estimate that the Vintage Racing event used less than 1/10<sup>th</sup> of the leaded racing fuel consumed at a single NASCAR event. Even so, the air monitoring stations located within the facility after the SVRA event recorded ambient lead concentrations as high as 0.64 µg/m<sup>3</sup>. While that is significantly lower than the Oregon OSHA permissible exposure level for lead (50

$\mu\text{g}/\text{m}^3$ ), if races were held daily, it could exceed the EPA's National Ambient Air Quality Standard's (NAAQS) allowable 3-month concentration average of  $0.15 \mu\text{g}/\text{m}^3$ .

Second, Hollingsworth and Rudik (2020) estimate that each 100,000 leaded race miles increased ambient air lead concentrations by 10% at air monitors within 50 miles of a NASCAR racetrack. Furthermore, using a similar quasi-experimental approach to ours, with county level demographic data, they find that NASCAR's deleading policy caused a statistically significant drop in elderly mortality of 91 deaths per 100,000 in counties containing a racetrack.

Finally, a pilot study by O'Neil et al. (2007) found that in 2004, the BLLs of a sample of NASCAR racing crews ranged from 1-22  $\mu\text{g}/\text{dL}$ , with a median of 9.4  $\mu\text{g}/\text{dL}$ . Over 40% of the crew members sampled had a BLL  $> 10 \mu\text{g}/\text{dL}$ , with those in closer proximity to the racing fumes having higher BLLs. By contrast, during the mid-2000s, geometric mean and 95<sup>th</sup> percentile BLLs in the U.S. male population were less than 2  $\mu\text{g}/\text{dL}$  and less than 5  $\mu\text{g}/\text{dL}$ , respectively (Centers for Disease Control 2018). Taken together, the evidence from these three studies suggests that the adoption of unleaded racing fuel at the Charlotte Motor Speedway likely led to a discernible reduction in lead exposure to expectant mothers living near the track.

*Identification Strategy:* Our study begins with all live births in the Charlotte-Concord-Gastonia (CCG) MSA between 2004-2009. We use exogenous variation in maternal exposure to lead resulting from NASCAR's switch in 2007 from leaded to unleaded racing fuel as a quasi-experiment. The Charlotte Motor Speedway, located in the CCG, is the only NASCAR racetrack in the state at which races were held every year during our sample period. Races occur bi-annually, around October and May, ensuring that all full and near full-term births in the sample are exposed in utero to *at least* one NASCAR event.

The distance between the mother's residence and Charlotte Motor Speedway is used to determine whether the birth is classified as being in the "treatment" group. Geodesic distances are calculated based

on the latitude-longitude coordinates of the mother's residential address (provided by birth certificate) and that of the Speedway, using geographic information system (GIS) software. We consider a birth to be "treated" if the mother's residence at time of birth is within a specified distance from the racetrack centroid. Because of uncertainty regarding the distance airborne lead can travel (Miranda et al. (2011), Hollingsworth and Rudik (2020)), we considered various treatment groups with distances beginning at  $D=3,000$  meters from the track, increasing in 1,000-meter increments. After examining the number of births in each treatment area (Supplemental Material Table S2), we settled on a treatment group of 0 - 4,000 meters for our primary specification. That definition provides a reasonable balance between capturing highly localized effects near the racetrack while including enough births to provide sufficient statistical power to detect potential effects on birth outcomes. A more detailed discussion around the choice of the size of the preferred treatment area is available in the Supplemental Materials.

Our control group consists of births where the mother's residential address is in the CCG but is at least 10,000 meters from the racetrack centroid: this provides a physical "buffer" of 6,000 meters between the primary treatment and control groups. That buffer minimizes the possibility of "spillover" effects from lead exposure that occur beyond our defined treatment area, which could otherwise bias our results towards zero. Our study area, and the spatially defined treatment and control groups, are depicted in Figure 1.

[FIGURE 1, HERE]

We focus on the following birth outcomes: birth weight (*BW*), low birth weight (*LBW*), gestational age (*Weeks*), preterm birth (*PRE*), and small for gestational age (*SGA*). Birth weight is a raw measure of the weight of the child at birth in grams, and gestational age is the clinical age at birth, measured in weeks. Following the existing literature, low birth weight is an indicator variable that takes on the value of 1 ( $LBW = 1$ ) if birth weight  $< 2,500$  grams (and  $LBW = 0$ , otherwise). Both small for gestational age and preterm birth are also indicator variables, where a newborn is classified as small for gestational age ( $SGA = 1$ ) if the weight is below the tenth percentile for clinical gestational age (and  $SGA = 0$ , otherwise),

and a birth is deemed to be preterm ( $PRE = 1$ ) if the clinical gestational age is  $< 37$  weeks (and  $PRE = 0$ , otherwise).

*Empirical Model:* We examine the effects of short-term in utero lead exposure on birth outcomes using a difference-in-differences (DD) model to compare birth outcomes in a non-randomized treatment group before and after deleading to those in the control group. The general estimating equation takes the form:

$$(1) Y_{imtc} = \beta_0 + \beta_1 \mathbb{1}(d_i \leq D) + \gamma \{ \mathbb{1}(d_i \leq D) \times delead_t \} + X_{it} \beta_2 + Z_{tc} \beta_3 + \beta_4 TRI_i + \beta_5 AP_i + tract_c \alpha + month_m \omega + year_t \eta + \epsilon_{imtc}$$

where  $Y_{imtc}$  is the birth outcome of infant  $i$ , born in month  $m$ , of year  $t$ , and to a mother residing in census tract  $c$ . The indicator variable  $\mathbb{1}(d_i \leq D)$  equals one if the distance between the mother's place of residence when the child was born and the CMS ( $d_i$ ) is within the spatial extent of the "treatment area" (distance  $D$ ), and zero otherwise. The indicator variable,  $delead_t$  equals one after NASCAR switched to unleaded racing fuel (in 2007), and is zero otherwise. We interact the treatment variable with the post-deleading variable creating the interacted variable  $\mathbb{1}(d_i \leq D) \times delead_t$ . The interacted variable equals one if infant  $i$  is born after NASCAR's voluntary deleading policy in 2007, to a mother living in the treatment area; and zero otherwise. This interaction term is of primary interest because it denotes the treated group, post treatment.

We adjust for several potentially confounding effects in the model. In particular,  $X_i$ , is a vector of individual controls that can affect the birth outcome, including mother's age, mother's education, mother's race, a binary variable for whether the mother smoked during pregnancy, the sex of the child, and birth order. We also include father's education as a control variable; however, it is important to take into account that because a mother may choose to exclude the birth father's information on the birth certificate, missing data in this field may not be random. (Approximately 15% of births in the MSA during our sample period do not include father's education.) To allow for this possibility in the variable



for father's education, we allow for an additional category, "missing" to be included when that data field is empty.

To avoid confounding effects from other sources of lead exposure, we also adjust for whether the mother lives within 2,000 meters of a TRI reporting facility ( $TRI_i$ ) that emits lead or lead compounds, whether she lives within 2,000 meters of an airport ( $AP_i$ ), and the percentage of the housing stock built pre-1950 in the Census tract. Distances are calculated using GIS software and based on geodesic distance.  $Z_{tc}$  is a vector of time-varying census tract controls that can affect birth outcomes, including both pre-1950 housing stock and median household income. We also include a set of Census tract ( $tract_c$ ), birth month ( $month_m$ ), and birth year ( $year_t$ ) indicator variables to adjust for all unobserved determinants of outcomes that are associated with infants whose mothers reside in a particular census tract, and who were born in a particular month, and year. Note that with the inclusion of individual year indicator variables, we cannot include a separate, uninteracted  $delead_t$  variable to capture the post-deleading policy effect as this will be absorbed by the individual year effects.  $\epsilon$  is assumed to be an unobserved, well-behaved, stochastic error term.

The coefficients to be estimated are  $\beta_0 \dots \beta_5$ ,  $\gamma$ ,  $\alpha$ ,  $\omega$ , and  $\eta$ . The coefficient of primary interest is  $\gamma$ , which measures the average effect of the switch to unleaded racing fuel on birth outcomes among infants with mothers residing close to the racetrack at the time of birth. More formally,  $\gamma$  is the average treatment effect of the treated ( $ATT = \gamma$ ) (Angrist and Pischke, (2009)).

Note that when the birth outcome is a binary variable (LBW, PRE, SGA), Equation (1) may be interpreted as a linear probability model, and the ATT is the change in the probability of the outcome variable resulting from the adoption of unleaded racing fuel. An alternative to the linear probability model, however, would be to use a logistic model and estimate the average change in the odds ratio resulting from the deleading policy. This model is described fully in the Supplemental Material.

Finally, to examine whether the effects of lead exposure are dependent upon when exposure occurs during the pregnancy, we decompose the average treatment effect by trimester(s) of exposure. To do this, we categorize all births in our matched sample by the trimester they either *were exposed to* a NASCAR event or *would have been exposed to* a NASCAR event, if they were located in the treatment area, creating the counterfactual for comparison. The estimating model, and more detailed description, may be found in the Supplemental Material. The difference in the average effects across trimesters are tested using an F-test.

*Internal Validity:* There are several potential sources of bias that may jeopardize our ability to estimate associations that are causal in nature. To minimize the possibility of bias arising from *unobserved* factors correlated with our outcome variables, we use several strategies. We include Census tract indicator variables to absorb any time-invariant neighborhood socioeconomic, environmental, or other characteristics that could be correlated with birth outcomes. The inclusion of year and month indicator variables adjust for broader temporal trends. In addition, our quasi-experimental, DD approach mitigates potential concerns from omitted variables that may be correlated with birth outcomes; such variables would have to be correlated with proximity to Charlotte Motor Speedway *and* the timing of the deleading event to bias our estimates. Finally, we pre-process our data using a many-to-one coarsened exact matching (CEM) algorithm. CEM is designed to better balance the distributions between the control and treatment groups over a set of *observable* variables that may be correlated with birth outcomes (Iacus, King & Porro, (2012)). If the samples are more balanced over observable characteristics, they may also be more comparable in terms of unobserved characteristics, further reducing bias.

Using CEM, we “prune” observations in the control and treatment groups that cannot be matched and use weights to simultaneously balance the distribution of the observed covariates across the remaining observations in the treatment and control groups. The variables that we use for exact matching are infant sex, birth month, birth year, and mother’s smoking behavior. Coarsened matching, based on bins, is used for birth order (5 bins); mother and father’s education level (4 and 5 bins, respectively); mother’s age (4

bins); mother's race (4 bins); Census block median household income (5 bins); and percentage of pre-1950 housing stock (4 bins). Bins for mother's age and race, and mother and father's education were chosen to match commonly used categories; percentage of pre-1950s housing bins were based on quartiles, and median household income bins were equally sized as the distribution was approximately normal.

## Results

Between 2004-2009, there were 147,673 live births in the CCG MSA. We have complete data on all birth outcomes and control variables for 147,160 of those births. After excluding births in the 4,000-10,000 meter buffer and running coarsened exact matching algorithm, we have 15,699 live births, of which 92.4% (14,508) are in the control group, and 7.6% (1,191) are in the 0-4,000 meter treatment group. While the treatment and control groups are similar prior to matching, (Table 1), after matching, we confirm there are no statistically significant differences in means between the treatment and control groups for all control variables, except for median household income and percentage of housing stock built pre-1950. Median household income is still higher, and the percentage of older housing stock is still lower, in the treatment group, but they are much closer after matching.

Estimation results for the aggregate DD models are summarized in Table 2, panels A and B. (Supplemental Material Table S3 provides complete estimation results and Table S6 summarizes the results for all treatment groups.) For each birth outcome, we find that the ATT is of the expected sign indicating birth outcomes improve after NASCAR delead its racing fuel. At 0 - 4,000 meters, the estimated ATT on birth weight is 103.94 grams [95% CI: 48.75, 159.13 grams]; and for gestational age it is 0.36 weeks [95% CI: 0.13, 0.59 weeks], or 2.5 days. For both outcomes that is an increase of almost 0.2 standard deviations relative to their mean values over the sample period.

For our binary variables we find that after deleading, the probability of low birth weight (LBW) declines by 4.07 percentage points [95% CI: 2.10, 6.17], preterm (PRE) births decline by 2.67 [95% CI:

0.20, 5.4], and small for gestational age (SGA) declines by 4.08 [95% CI: 1.50, 6.67]. From the logistic model (Table 2B), we find the change in the odds ratio due to the deleading policy is 0.488 [95% CI: 0.348, 0.684] for LBW, 0.714 [95% CI: 0.517, 0.985] for PRE, and 0.575 [95% CI: 0.419, 0.788] for SGA. The corresponding reductions in the odds of LBW, PRE, and SGA are 51%, 29%, and 43%, respectively. (Supplemental Material Table S4 provides complete estimation results and Table S7 summarizes the results for all treatment groups.)

Results from the CEM-weighted trimester model are summarized in Table 3. (Supplemental Material Table S5 provides additional trimester results and Tables S8-S9 present estimation results for all treatment groups.) For ease of interpretation, we exclude all births that had no lead exposures from this model (approximately 0.025% of our matched sample). We reject the null hypothesis that the ATTs are the same by trimester of exposure for birth weight, preterm birth, and gestational age, but cannot do so for low birth weight or small for gestational age births.

For birth weight, preterm birth, and gestational age, we find that the largest effects from lead exposure occur during the first trimester, followed by the third. In all three cases the ATT during the second trimester is not statistically significant. For the 0-4,000 meter treatment group, the ATT on birth weight for a first trimester exposure is 469.2 grams [95% CI: 221.8, 716.6]; for a second trimester exposure it is 64.6 grams [95% CI: -15.0, 144.2]; and for a third trimester exposure it is 193.1 grams [95% CI: 38.1, 348.1]. For gestational age, by trimester, the estimated effects of deleading are 1.86 weeks [95% CI: 0.84, 2.83]; -0.02 weeks [95% CI: -0.27, 0.22]; and 0.83 weeks [95% CI: 0.055, 1.71]. And for preterm births, by trimester, the ATTs are -0.22 percentage points [95% CI: -0.39, - 0.06]; 0.01 percentage points [95% CI: -0.04, 0.64]; and -0.13 percentage points [95% CI: -0.23, - 0.03]. While not shown here (see Supplemental Material Table S5), the effects of lead exposure during multiple trimesters, not surprisingly, are associated with larger adverse effects on birth outcomes and are all statistically significant.

*Robustness Checks.* We conduct a falsification test with 35 airports located in the CCG MSA, to ensure that our identification strategy is statistically valid. Single piston engine airplanes continue to use leaded aviation gasoline (U.S. EPA (2020), Zahran et al (2017)), and as such, we should not see a change in birth outcomes after 2007 around these airports from a reduction in lead emissions related to NASCAR’s deleading policy unless there are unobserved corresponding factors correlated with the timing of NASCAR’s deleading policy and proximity to lead emitting facilities. In the falsification test, for each birth outcome, we find that the average (false) treatment effect is smaller than the corresponding treatment effect and not statistically significant, supporting our identification strategy. (Table 4, Supplemental Material Table S10.)

To verify that the treatment year 2007 is not capturing some other effect that is unrelated to NASCAR’s adoption of unleaded racing fuel, we perform falsification tests using different pseudo-policy adoption years (Table 5) using the treatment group at 4,000 meters. To do this, we extend our data set back in time to include births from 2000 and re-estimate average treatment effects under the assumption that the pseudo-treatment year occurs in 2001, 2003, 2005, or 2007 (the true year). Because we include all years in our analysis, including the post-deleading years, we do not expect our estimated ATT necessarily to be statistically insignificant for all years, excluding the “true” policy adoption year of 2007. Instead, if 2007 is, indeed, the true year of the treatment, we would expect to find the magnitude and statistical significance of the effect to be largest in 2007. This is, in fact, what we observe for all birth outcomes. That result also is robust to extending the sample period beyond 2009, through at least 2013, the last year for which we have available data.

Our quasi-experimental design relies on the “parallel trends” assumption to provide a statistically appropriate counterfactual. The assumption is that in the absence of the adoption of unleaded racing fuel, we would expect the treatment and control groups to have similar trends in birth outcomes. If the treatment and control group trends are approximately parallel before the deleading policy is implemented, then that is evidence that the constructed control group serves as a good counterfactual. In Figure 2, we

plot the average individual-level birth outcomes over time for our treatment and control groups. In the period prior to deleading, the trends between the groups shows some variability, but for all birth outcomes (except, possibly for SGA), the pre-deleading trends between the groups are roughly similar, suggesting that the parallel trends assumption is not violated. This is further supported through F-tests where we compare the difference in the sample means of each birth outcome each year between the treatment and control groups, before deleading. In each case, we cannot reject the equality of the difference in sample means, suggesting that the pre-deleading trends are statistically parallel. The only exception is again for SGA, which could be rejected ( $p = 0.015$ ). (Supplemental Material Table S11.)

[FIGURE 2 HERE]

## Discussion

Our quasi-experiment provides compelling evidence that even a single, short-term, in utero lead exposure from a NASCAR event lowers birth weight, reduces gestational age, and increases the likelihood of low birth weight, preterm, and small for gestational age births for mothers who reside within 4,000 meters of the event. We also find that the fetus is most vulnerable to lead exposure during the first trimester of the pregnancy for birth weight, weeks of gestation, and preterm births. The average treatment effect of a first trimester exposure to lead is 469 grams – or 14% of the mean birth weight; 1.89 weeks, or 5% of mean gestational age; and a 22.2 percentage point reduction in the probability of preterm birth. We do not find statistical support for differences in trimester effects for the other two birth outcomes (probability of low birth weight, and small for gestational age). We believe this is the first study to focus on the spatial effects of *transient* lead emissions on birth outcomes, as well as the first to investigate whether the effects on birth outcomes of in utero ambient lead exposure are heterogeneous across trimesters.

Our study has some limitations. First, we assume that the mother's residence at the time of birth reflects the in utero lead exposure of the fetus. If not, it could introduce measurement error into our

analysis, biasing our results towards zero. Our study also is based on a single racetrack in North Carolina, which makes the quasi-experiment more susceptible to spatially and temporally correlated confounders. Our use of numerous control variables, a DD methodology, and coarsened exact matching helps minimize such concerns, and the falsification test and examination of parallel trends bolsters our causal interpretation. Third, we cannot link lead emissions from NASCAR races directly to ambient air concentrations. Even if air monitoring stations were readily available, they would not necessarily provide us with accurate information. It has recently been shown based on experiments using a 1957 Ford Thunderbird, (Griffiths (2020)) that the average tailpipe particle size from leaded gasoline was 0.035 micrometers. This is far below the particle size that can be measured in the field (2.5 micrometers), and calls into question the accuracy of lead readings from air monitoring stations. Fourth, we do not know how transient exposure to ambient lead affects the mother's blood lead or other measures of lead load to the fetus that would allow us to translate our findings to other lead exposure contexts.

Despite these limitations, our results are robust, and highlight how even a short window of exposure (NASCAR events typically occur over three days) is enough to adversely affect birth outcomes in a widely dispersed area around the emitting source. Moreover, the magnitude of the estimated effects on birth weight, preterm, and gestational age, resulting from a first trimester exposure, alone, are large and significant. Failing to account for the heterogeneity of the effects of lead exposure across trimesters could understate the negative effects of lead on birth outcomes. Currently, the EPA's National Ambient Air Quality Standards for lead, set to protect public health and the environment, are based on a 3-month moving average. Our findings suggest that this approach may not be well-suited for identifying risks from short-term exposure that may affect fetal health.

## **Conclusion**

Using NASCAR's voluntarily decision to switch from leaded to unleaded racing fuel in 2007 as a quasi-experiment, we estimate the average treatment effect resulting from the reduction in lead emissions on birth outcomes around the Charlotte Motor Speedway in North Carolina. We find robust evidence that

the reduction in emissions resulted in an increase in average birth weight and weeks of gestation, while also reducing the likelihood of preterm, low birth weight, and small for gestational age births for mothers residing between 0 to 4,000 meters from the racetrack. For birth weight, weeks of gestation, and preterm births, the effects of in utero lead exposure vary by trimester, with the largest effects arising in the first and then third trimester. We do not find heterogeneous effects in exposure for low birth weight or small for gestational age births. Our findings highlight the importance of considering the spatial and temporal nature of ambient lead emissions when assessing their public health effects and suggests the possibility of prenatal lead interventions to improve birth outcomes.



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Figure 1. Map of General Treatment and Control Group

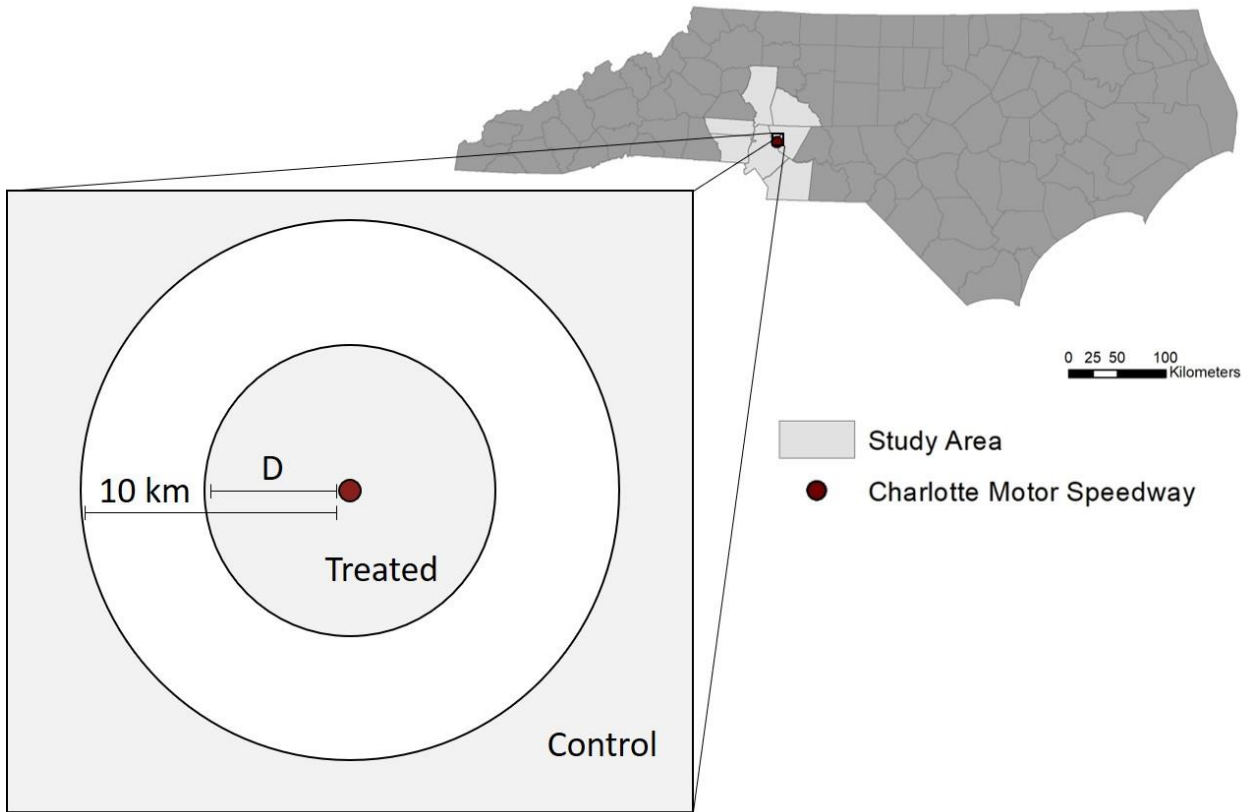
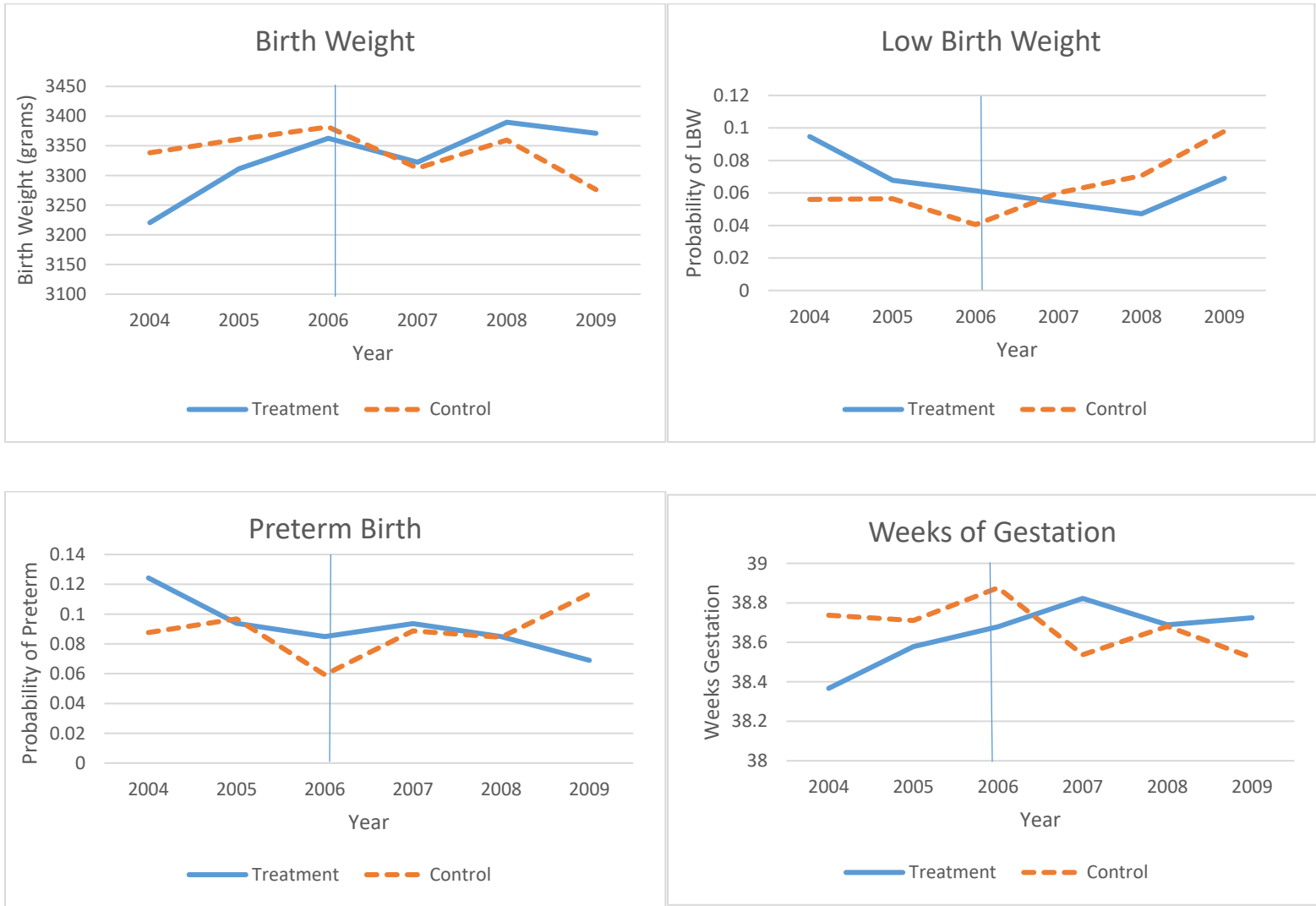


Figure 2. CEM weighted trends for birth outcomes in control and treatment area of 4,000 meters



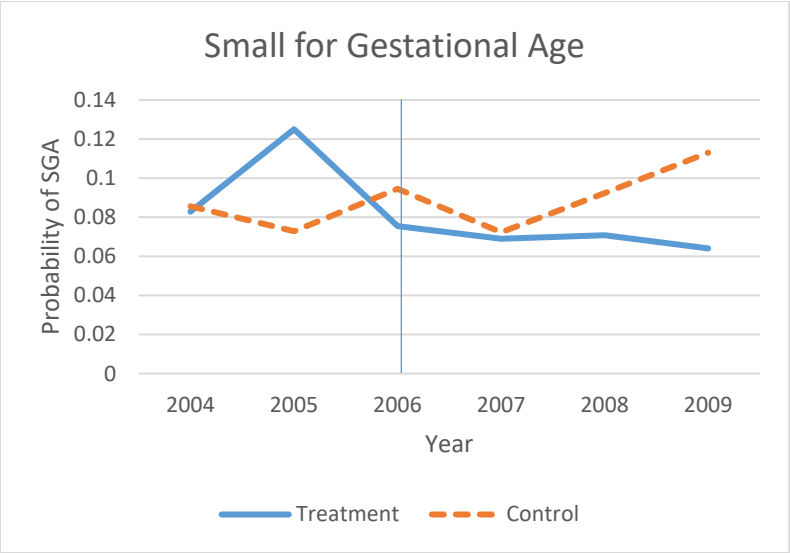


Table 1: Characteristics of the Study Population, MSA, Control and Treatment Groups, 0 – 4,000 meters: 2004-2009

	Unmatched Control	Unmatched Treatment	CEM-Weighted Control	CEM-Weighted Treatment	P
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
BW (grams)	3,258.55 ± 609.35	3,315.34 ± 589.41	3342.89 ± 583.08	3333.53 ± 564.58	0.620
LBW	0.09 ± 0.28	0.07 ± 0.26	0.06 ± 0.24	0.06 ± 0.25	0.716
Weeks	38.55 ± 2.24	38.59 ± 2.11	38.69 ± 2.07	38.65 ± 1.97	0.604
PRE	0.11 ± 0.31	0.10 ± 0.29	0.09 ± 0.28	0.09 ± 0.29	0.707
SGA	0.11 ± 0.31	0.09 ± 0.28	0.09 ± 0.28	0.08 ± 0.27	0.515
Mother <20 yo	0.11 ± 0.31	0.06 ± 0.24	0.05 ± 0.23	0.05 ± 0.23	0.981
Mother 20-29 yo	0.50 ± 0.50	0.47 ± 0.50	0.48 ± 0.50	0.48 ± 0.50	0.917
Mother 30-39 yo	0.37 ± 0.48	0.45 ± 0.50	0.46 ± 0.50	0.46 ± 0.50	0.897
Mother >39 yo	0.02 ± 0.15	0.02 ± 0.15	0.01 ± 0.09	0.01 ± 0.09	0.946
Mother < High School	0.22 ± 0.42	0.14 ± 0.35	0.11 ± 0.32	0.12 ± 0.32	0.786
Mother High School	0.26 ± 0.44	0.20 ± 0.40	0.17 ± 0.37	0.17 ± 0.38	0.819
Mother Some College	0.21 ± 0.41	0.25 ± 0.43	0.24 ± 0.43	0.24 ± 0.43	0.856
Mother College+	0.31 ± 0.46	0.42 ± 0.49	0.48 ± 0.50	0.47 ± 0.50	0.832
Mother White	0.55 ± 0.50	0.58 ± 0.49	0.62 ± 0.49	0.62 ± 0.49	0.875
Mother Black	0.23 ± 0.42	0.21 ± 0.41	0.21 ± 0.41	0.21 ± 0.40	0.958
Mother Hispanic	0.19 ± 0.39	0.15 ± 0.36	0.13 ± 0.33	0.13 ± 0.34	0.789
Mother Other Race	0.04 ± 0.19	0.07 ± 0.25	0.05 ± 0.21	0.05 ± 0.21	0.985
Father < High School	0.17 ± 0.37	0.11 ± 0.31	0.09 ± 0.28	0.09 ± 0.28	0.934
Father High School	0.25 ± 0.43	0.21 ± 0.41	0.20 ± 0.40	0.20 ± 0.40	0.743
Father Some College	0.15 ± 0.36	0.22 ± 0.42	0.22 ± 0.42	0.22 ± 0.41	0.879
Father College+	0.28 ± 0.45	0.38 ± 0.49	0.43 ± 0.50	0.42 ± 0.49	0.701
Father Educ. Missing	0.15 ± 0.36	0.08 ± 0.27	0.06 ± 0.24	0.07 ± 0.25	0.733
1=Male Infant	0.51 ± 0.50	0.52 ± 0.50	0.53 ± 0.50	0.52 ± 0.50	0.863
Smoked	0.09 ± 0.29	0.04 ± 0.21	0.02 ± 0.14	0.02 ± 0.15	0.753
Birth Order	2.03 ± 1.18	1.97 ± 1.07	1.82 ± 0.92	1.83 ± 0.93	0.810
1=TRI Facility < 2000m	0.11 ± 0.31	0.00 ± 0.00	0.08 ± 0.27	0.00 ± 0.00	0.000
1=Airport < 2000 m	0.11 ± 0.31	0.01 ± 0.11	0.04 ± 0.21	0.02 ± 0.12	0.000
Median Household Income	52,317 ± 22,281	62,875 ± 8,073	66149.97 ± 13837.22	63022.86 ± 7964.25	0.000
% Pre-1950s Housing	0.10 ± 0.12	0.01 ± 0.01	0.02 ± 0.01	0.01 ± 0.01	0.000
No. of Observations	135,845	1,451	14,508	1,191	15,699

Note: BW = birth weight, LBW = low birth weight (< 2,500 grams), Weeks = weeks gestation (clinical), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age). P = probability value associated with the difference in means between the treatment and control group.

Table 2: CEM weighted, estimated ATT of deleading on birth outcomes for treatment area 0 – 4,000 meters, 2004-2009

Panel A: Multivariable Regression Model	
Birth Outcome	ATT
Birth Weight (grams)	103.937 (48.745, 159.128)
Low Birth Weight (< 2500 g)	-0.041 (-0.062, -0.020)
Gestational Age (Clinical weeks)	0.360 (0.127, 0.594)
Preterm (< 37 weeks)	-0.027 (-0.052, -0.002)
Small for Gestational Age	-0.041 (-0.066, -0.015)
Observations: 15,699	
Panel B: Logistic Regression Model	
Birth Outcome	Odds Ratio (ATT)
Low Birth Weight (< 2500 g)	0.488 (0.348, 0.684)
Observations: 15,419	
Preterm (< 37 weeks)	0.714 (0.517, 0.985)
Observations: 15,632	
Small for Gestational Age	0.575 (0.419, 0.788)
Observations: 15,559	

ATT = average treatment effect of the treated

95% confidence intervals provided in parentheses, below estimated coefficient.

Note: All regressions adjust for mother's age, mother's race, mother's education, father's education, male infant, whether the mother smoked during pregnancy, birth order, proximity to TRI lead emitting facility, proximity to airport, tract-level median household income, and tract-level share of pre-1950's homes. All regressions include a set of indicator variables for census tract, month, and year. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.



Table 3: CEM weighted, estimated ATT on birth outcome of deleading by trimester of in utero exposure for treatment area 0 – 4,000 meters, 2004-2009

Trimester of Exposure	Birth Weight (Grams)	Low Birth Weight (<2500g)	Gestational Age (Clinical Weeks)	Preterm Birth (<37 Weeks)	Small for Gestational Age
Trimester 1	469.173 (219.346, 719.000)	-0.069 (-0.182, 0.044)	1.859 (0.830, 2.887)	-0.222 (-0.390, -0.054)	-0.065 (-0.246, 0.117)
Trimester 2	64.69 (-15.790, 144.928)	-0.035 (-0.072, 0.003)	-0.022 (-0.269, 0.224)	0.014 (-0.037, 0.065)	-0.064 (-0.130, 0.002)
Trimester 3	193.126 (36.683, 349.580)	-0.127 (-0.282, 0.018)	0.827 (-0.064, 1.712)	-0.129 (-0.229, -0.028)	-0.040 (-0.124, 0.044)

Observations: 15,695

Small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age)

ATT = average treatment effect of the treated

95% confidence intervals provided in parentheses, below estimated coefficient.

Note: All regressions adjust for mother's age (indicator), mother's race (indicator), mother's education (indicator), father's education (indicator), male infant (indicator), whether the mother smoked during pregnancy (indicator), birth order, proximity to TRI lead emitting facility (indicator), proximity to airport (indicator), tract-level median household income (continuous), and tract-level share of pre-1950's homes (percentage). All regressions include a set of indicator variables for census tract, month, and year. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

Table 4: CEM weighted ATT for airports from NASCAR deleading policy for “treatment” area 0 – 4,000 meters, 2004-2009

Birth Outcome	ATT
Birth Weight (grams)	1.324 (-31.290, 33.938)
Low Birth Weight	-0.00541 (-0.020, 0.009)
Weeks of Gestation (weeks)	-0.0530 (-0.169, 0.063)
Preterm	-0.00249 (-0.019, 0.014)
Small for Gestational Age	-0.00825 (-0.023, 0.006)
Observations:	34,700

95% confidence intervals provided in parentheses, below estimated coefficient.

ATT = average treatment effect of the treated

Note: All regressions adjust for mother's age, mother's race, mother's education, father's education, male infant, whether the mother smoked during pregnancy, birth order, proximity to TRI lead emitting facility, proximity to airport, tract-level median household income, and tract-level percentage of pre-1950's homes. All regressions include a set of indicator variables for census tract, month, and year. All regressions are weighted using CEM-matching weights. Standard errors are clustered on the census tract.

Table 5. CEM weighted ATT for various false adoption dates for treatment group 0 – 4,000 meters, 2000-2009

“Adoption” Date	Birth Weight (grams)	Low Birth Weight (< 2500 g)	Gestational Age (Clinical weeks)	Preterm (<37 weeks)	Small for Gestational Age
False 2001	-56.090 (-134.709, 22.530)	0.024 (-0.007, 0.055)	-0.139 (0.476, 0.198)	0.026 (-0.001, 0.054)	0.048 (0.015, 0.081)
False 2002	-32.569 (-103.292, 38.153)	0.025 (-0.0005, 0.050)	-0.026 (-0.334, 0.282)	0.011 (-0.015, 0.037)	0.034 (-0.007, 0.075)
False 2003	-2.274 (-62.120, 57.572)	0.005 (-0.029, 0.038)	0.060 (-0.312, 0.432)	0.009 (-0.027, 0.046)	0.016 (-0.017, 0.049)
False 2004	1.116 (-40.907, 43.139)	-0.003 (-0.025, 0.020)	0.081 (-0.175, 0.338)	-0.004 (-0.034, 0.026)	0.007 (-0.007, 0.021)
False 2005	41.857 (-1.180, 84.894)	-0.016 (-0.046, 0.014)	0.190 (-0.110, 0.490)	-0.015 (-0.054, 0.024)	0.002 (-0.016, 0.020)
False 2006	57.712 (17.951, 97.491)	-0.019 (-0.043, 0.006)	0.215 (0.025, 0.405)	-0.011 (-0.043, 0.022)	-0.023 (-0.036, -0.009)
True 2007	73.170 (24.590, 121.750)	-0.031 (-0.048, -0.0130)	0.308 (0.156, 0.460)	-0.022 (-0.047, 0.003)	-0.025 (-0.046, -0.005)
Observations: 24,784					

BW = birth weight, LBW = low birth weight (< 2,500 grams), Weeks = Weeks gestation (clinical), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age). P = probability value associated with the difference in means between the treatment and control group. ATT = average treatment effect of the treated.

Note: 95% confidence intervals provided in parentheses, below estimated coefficient.

All regressions adjust for mother's age (indicator), mother's race (indicator), mother's education (indicator), father's education (indicator), male infant (indicator), whether the mother smoked during pregnancy (indicator), birth order, proximity to TRI lead emitting facility (indicator), proximity to airport (indicator), tract-level median household income, and tract-level share of pre-1950's homes (percentage). All regressions include a set of indicator variables for census tract, month, and year. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

## Supplemental Material

In this section, we present additional materials for readers who would like to see our model and results in greater detail.

**Alternative Treatment Areas:** Several considerations were taken in choosing the size of our preferred treatment area. We started by considering areas beginning at 3,000 meters from the racetrack and extending out to 5,000 meters. Descriptive statistics for treatment areas defined by 0 – 3,000 meters, and 0 – 5,000 meters are given in Table S1.

[Table S1, Here]

Although we wanted to examine birth outcomes as close to the racetrack as possible, we were limited by the number of birth outcomes in those areas (see Table S2).

[Table S2, Here]

The appropriateness of the assumed 0 - 4,000 meters zone for the preferred treatment group is confirmed by earlier data diagnostics. If anything, this assumed treatment area could even be considered conservative. For each birth outcome we separately trace out the pre- and post-deleading policy distance gradients. These gradients are estimated using local polynomial regression techniques, and thus provide a detailed, data-driven depiction of how the birth outcomes vary with respect to proximity to the Charlotte Motor Speedway. Consider birthweight, for example, the intuition is that if births are adversely affected by lead emissions from the racetrack, then the pre-deleading gradient would show slightly lower birth weights closer to the racetrack, and the gradient would then gradually increase at farther distances. In contrast, the post-deleading gradient would presumably not be lower nearest the track because leaded fuel is no longer used. The distance at which the pre-treatment distance gradient converges to the post-treatment gradient provides some insight as to how far lead emissions from the racetrack spread and adversely affected birth outcomes, and thus informs our decision of the appropriate spatial extent for the treatment group.

We estimated the distance gradients in two ways. First, we used local polynomial regressions to graph the raw outcome variables. These are shown in the left panels of Figure S1. This non-parametric depiction is attractive as it is based on the raw data, but the estimated gradients are still susceptible to potentially confounding factors. For the second approach, we use a semi-parametric strategy where we first estimate regression models similar to Eq. 1 (from main text and replicated, below), but do not include any covariates related to proximity to the racetrack or the deleading policy. All other control variables, however, are included.

$$(1) Y_{imtc} = \beta_0 + \beta_1 \mathbb{1}(d_i \leq D) + \gamma \{ \mathbb{1}(d_i \leq D) \times delead_t \} + X_{it} \beta_2 + Z_{tc} \beta_3 + \beta_4 TRI_i \\ + \beta_5 AP_i + tract_c \alpha + month_m \omega + year_t \eta + \epsilon_{imtc}$$

The estimated residuals ( $\hat{\epsilon}_{imtc}$ ) from Eq. 1 represent all unexplained variation in the birth outcome, after conditioning out otherwise confounding factors. The same local polynomial regression techniques are used to graph how these residuals vary with distance from the racetrack, both before and after the deleading policy, as shown in the right panels of Figure S1. Although there is some fluctuation nearest the racetrack for some outcome variables, the distance gradients across both approaches and all five birth outcomes generally suggest that the pre- and post-deleading distance gradients converge between 4,000 and 6,000 meters. To be conservative, for the main analysis we chose a treatment group consisting of all mothers who lived within 0 - 4,000 meters of Charlotte Motor Speedway (CMS) at the time of their child's birth.

### Logistic Model:

If we define  $p$  as the probability that an adverse birth outcome occurs, then the logistic model to be estimated is:

$$(2) \quad p_{imtc} = F\{\beta_0 + \beta_1 \mathbb{1}(d_{it} \leq D) + \gamma \{\mathbb{1}(d_{it} \leq D) \times delead_t\} + X_{it} \beta_2 + Z_{tc} \beta_3 + \beta_4 TRI_i \\ + \beta_5 AP_i + tract_c \alpha + month_m \omega + year_t \eta\}$$

where  $F\{\cdot\}$ , in this case is a logistic cumulative density function.

The coefficient of interest continues to be  $\gamma$ . Under this logistic model specification,  $exp(\gamma)$  equals the average change in the odds ratio attributable to the deleading policy.

### Trimester Model.

We decompose the average treatment effect by trimester(s) of exposure to examine whether the effects of lead are dependent upon when, during the pregnancy, exposure occurs. To do this, we categorize all births in our matched sample by the trimester they either *were exposed to* a NASCAR event or *would have been exposed to* a NASCAR event, if they were located in the treatment area. We then estimate the following model:

$$(3) \quad Y_{imtc} = \beta_0 + \sum_{j=1}^3 \beta_{0j} T_{ij} + \sum_{j=1}^3 \beta_{1j} \{\mathbb{1}(d_i \leq D) \times T_{ij}\} + \sum_{j=1}^3 \beta_{2j} \{delead_{it} \times T_{ij}\} + \\ + \sum_{j=1}^3 \gamma_{1j} \{\mathbb{1}(d_i \leq D) \times after_{it} \times T_{ij}\} + \beta_3 T_{i1} \times T_{i2} + \beta_4 T_{i1} \times T_{i3} + \beta_5 T_{i2} \times T_{i3} + \beta_6 T_{i1} \times T_{i2} \times \\ T_{i3} + \beta_7 \{\mathbb{1}(d_i \leq D) \times T_{i1} \times T_{i2}\} + \beta_8 \{\mathbb{1}(d_i \leq D) \times T_{i1} \times T_{i3}\} + \beta_9 \{\mathbb{1}(d_i \leq D) \times T_{i2} \times T_{i3}\} + \\ \beta_{10} \{\mathbb{1}(d_i \leq D) \times T_{i1} \times T_{i2} \times T_{i3}\} + \gamma_4 \{\mathbb{1}(d_i \leq D) \times T_{i1} \times T_{i2} \times after_{it}\} + \gamma_5 \{\mathbb{1}(d_i \leq D) \times T_{i1} \times \\ T_{i3} \times delead_{it}\} + \gamma_6 \{\mathbb{1}(d_i \leq D) \times T_{i2} \times T_{i3} \times delead_{it}\} + \gamma_7 \{\mathbb{1}(d_i \leq D) \times T_{i1} \times T_{i2} \times T_{i3} \times \\ delead_{it}\} + X_{it} \beta_6 + Z_{tc} \beta_7 + tract_c \alpha + month_m \omega + year_t \eta + \epsilon_{imtcj},$$

where the variable  $T_{ij}$ , is an indicator equal to one if infant  $i$  is exposed in utero to a race during trimester  $j = 1, 2, 3$ , and zero, otherwise. Exposures that occur in multiple trimesters are captured by interacting the trimester indicator variables, for example,  $T_{ij} \times T_{ik}, j \neq k$ , which equals one if infant  $i$  is exposed in utero during trimester  $j$  and  $k$ , and zero otherwise. The average treatment effect from a single exposure during trimester  $j$  is given by  $\gamma_j$  for  $j = 1, 2, 3$ . To estimate the average treatment effect from multiple exposures, one must add the individual average treatment effects for the affected trimesters to the average treatment effect for the multiple exposures, given by one of  $\gamma_k$  for  $k = 4, 5, 6, 7$ . We test for equality of the average treatment effect of the treated (ATT) across the three trimesters using an F-test.

### Results for 0 – 4,000 meters:

[Tables S3, S4, S5, Here]

### Results for Alternative Treatment Areas:

[Tables S6, S7, S8, S9, Here]

### Airport Falsification Test:

[Table S10, Here]

### Testing for Parallel Trends Assumption:

In addition to using visual techniques to determine whether the parallel trends assumption holds, we also examine whether the trends in birth outcomes are statistically the same prior to NASCAR adopting unleaded racing fuel in 2007. To do so we estimate the following model:

$$(3) Y_{imtc} = \beta_0 + \beta_t \sum_{t=2004}^{2009} \mathbb{1}(d_i \leq 4,000 m) * year_t + tract_c \alpha + year_t \eta + \epsilon_{imtc}$$

where  $Y_{imtc}$  is the birth outcome of infant  $i$ , born in month  $m$ , and year  $t$ , with the mother residing in census tract,  $c$ . The indicator variable  $\mathbb{1}(d_i \leq 4000 m)$  equals one if the distance from the mother's place of residence to the racetrack when the child was born ( $d_i$ ) is within the spatial extent of the treatment zone (0 - 4,000 m), and zero otherwise. We interact the treatment variable with each year indicator variable creating the interacted variable  $\mathbb{1}(d_i \leq 4000 m) \times year_t$ . The interacted variable equals one if infant  $i$  is born to a mother living in the treatment area in year  $t$ ; and zero otherwise. The interaction term is of primary interest as it measures the difference in the birth outcome between the treated and control groups for each year.

If trends in the birth outcome are statistically the same (i.e., parallel) prior to 2007, then the difference in birth outcomes between the treatment and control groups should be the same for each year from 2004-2006 (i.e.,  $\beta_{2004} = \beta_{2005} = \beta_{2006}$ ). We summarize the results in Table S11. The F-statistic on the joint test  $\beta_{2004} = \beta_{2005} = \beta_{2006}$  indicates that the difference between the treatment and control groups, for each birth outcome, except SGA, is statistically the same each year from 2004-2006. We therefore conclude the pre-deleading trends do not violate the parallel trends assumption, except for SGA.

[Table S11, Here]

[Figure S1, Here]

**Table S1. Characteristics of the study population for alternate treatment groups: 2004-2009**

	Unmatched 0 – 3,000 m	Unmatched 0 – 5,000m	CEM Weighted 0 – 3,000m	CEM Weighted 0 – 5,000m
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
BW (grams)	3,278.34 ± 602.65	3,306.28 ± 588.97	3,309.48 ± 550.51	3,317.9 ± 572.5
LBW	0.08 ± 0.27	0.08 ± 0.27	0.07 ± 0.25	0.1 ± 0.3
Weeks	38.64 ± 2.27	38.60 ± 2.09	38.76 ± 1.92	38.6 ± 2.0
PRE	0.09 ± 0.29	0.10 ± 0.29	0.09 ± 0.29	0.1 ± 0.3
SGA	0.10 ± 0.30	0.09 ± 0.29	0.10 ± 0.29	0.1 ± 0.3
Mother <20 yo	0.08 ± 0.28	0.06 ± 0.24	0.08 ± 0.26	0.1 ± 0.2
Mother 20-29 yo	0.49 ± 0.50	0.48 ± 0.50	0.50 ± 0.50	0.5 ± 0.5
Mother 30-39 yo	0.41 ± 0.49	0.43 ± 0.49	0.41 ± 0.49	0.4 ± 0.5
Mother >39 yo	0.01 ± 0.12	0.03 ± 0.16	0.01 ± 0.09	0.0 ± 0.1
Mother < High School	0.24 ± 0.42	0.13 ± 0.34	0.20 ± 0.40	0.1 ± 0.3
Mother High School	0.20 ± 0.40	0.20 ± 0.40	0.17 ± 0.37	0.2 ± 0.4
Mother Some College	0.23 ± 0.42	0.25 ± 0.43	0.24 ± 0.43	0.2 ± 0.4
Mother College+	0.34 ± 0.47	0.42 ± 0.49	0.39 ± 0.49	0.5 ± 0.5
Mother White	0.50 ± 0.50	0.56 ± 0.50	0.53 ± 0.50	0.6 ± 0.5
Mother Black	0.18 ± 0.39	0.22 ± 0.41	0.19 ± 0.39	0.2 ± 0.4
Mother Hispanic	0.27 ± 0.44	0.15 ± 0.36	0.24 ± 0.43	0.1 ± 0.3
Mother Other Race	0.04 ± 0.21	0.06 ± 0.24	0.04 ± 0.19	0.0 ± 0.2
Father < High School	0.19 ± 0.39	0.11 ± 0.31	0.16 ± 0.37	0.1 ± 0.3
Father High School	0.21 ± 0.41	0.22 ± 0.41	0.20 ± 0.40	0.2 ± 0.4
Father Some College	0.20 ± 0.40	0.22 ± 0.41	0.21 ± 0.41	0.2 ± 0.4
Father College+	0.31 ± 0.46	0.38 ± 0.48	0.35 ± 0.48	0.4 ± 0.5
Father Educ. Missing	0.09 ± 0.29	0.08 ± 0.28	0.08 ± 0.27	0.1 ± 0.3
1=Male Infant	0.55 ± 0.50	0.51 ± 0.50	0.57 ± 0.50	0.5 ± 0.5
Smoked	0.05 ± 0.22	0.05 ± 0.21	0.02 ± 0.15	0.0 ± 0.2
Birth Order	1.95 ± 1.05	1.94 ± 1.07	1.84 ± 0.93	1.8 ± 0.9
1=TRI Facility <2000m	0.00 ± 0.00	0.01 ± 0.08	0.00 ± 0.00	0.0 ± 0.1
1=Airport< 2000 m	0.03 ± 0.16	0.05 ± 0.21	0.03 ± 0.18	0.1 ± 0.2
Median Household Income \$	60,937.13 ± 5,041.48	62,712.87 ± 11,564.53	60,982.26 ± 5,079.23	62,851.2 ± 11,391.4
% Pre-1950s Housing	0.01 ± 0.01	0.02 ± 0.02	0.01 ± 0.01	0.0 ± 0.0
No. of Observations	627	2,355	504	1,951

BW = birth weight, LBW = low birth weight (< 2,500 grams), Weeks = Weeks gestation (clinical), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age)

**Table S2: Number of births and adverse birth outcomes by distance from racetrack: 2004- 2009 (unweighted)**

Treatment Area	Births	LBW	PRE	SGA
0 – 3,000 m	627	48	48	63
0 – 4,000 m	1,451	108	139	127
0 – 5,000 m	2,355	180	225	210

LBW = low birth weight (< 2,500 grams), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age)



**Table S3. The CEM-weighted ATT of deleading on birth outcomes, 0 – 4,000 meters: 2004-2009**

VARIABLES	BW (grams)	LBW	Weeks	PRE	SGA
ATE: 4,000m	103.9*** (27.89)	-0.0407*** (0.0107)	0.360*** (0.118)	-0.0267** (0.0126)	-0.0408*** (0.0132)
Mother 20-29 yo	-27.30 (56.60)	0.0232 (0.0226)	-0.196 (0.177)	0.00640 (0.0291)	-0.00825 (0.0415)
Mother 30-39 yo	-7.385 (57.83)	0.0265 (0.0241)	-0.370* (0.196)	0.0176 (0.0302)	-0.0211 (0.0435)
Mother >39 yo	-151.0 (143.0)	0.0647 (0.0698)	-0.415 (0.493)	0.0355 (0.0724)	0.0600 (0.0674)
Mother Black	-260.7*** (25.29)	0.0575*** (0.0114)	-0.502*** (0.120)	0.0329*** (0.0125)	0.0929*** (0.0146)
Mother Hispanic	-15.81 (41.43)	-0.000114 (0.0150)	0.104 (0.114)	-0.00495 (0.0221)	0.0124 (0.0269)
Mother Other Race	-200.6*** (55.43)	0.0345* (0.0190)	-0.108 (0.130)	0.000115 (0.0198)	0.106*** (0.0328)
Mother High School	-17.07 (56.33)	-0.000628 (0.0185)	-0.0950 (0.164)	0.0117 (0.0289)	0.0142 (0.0377)
Mother Some College	11.76 (65.70)	-0.00727 (0.0206)	-0.128 (0.192)	0.0201 (0.0336)	0.00644 (0.0458)
Mother College+	12.39 (65.66)	0.00702 (0.0218)	-0.119 (0.226)	0.0206 (0.0317)	0.0126 (0.0426)
Father High School	71.35 (47.44)	0.0169 (0.0144)	-0.0334 (0.195)	-0.00927 (0.0336)	-0.0373 (0.0422)
Father Some College	45.09 (60.35)	0.0196 (0.0140)	-0.0400 (0.191)	-0.00608 (0.0381)	-0.0235 (0.0450)
Father College+	74.95 (56.05)	0.00827 (0.0152)	0.0829 (0.215)	-0.0296 (0.0389)	-0.0436 (0.0444)
Father Educ. Missing	-78.17 (55.30)	0.0421 (0.0270)	-0.129 (0.265)	-0.000704 (0.0453)	0.0147 (0.0391)
Male Infant	126.8*** (16.09)	-0.00463 (0.00658)	-0.0383 (0.0609)	-0.00475 (0.00855)	-0.00836 (0.00977)
Smoked	-224.3** (95.85)	0.0562 (0.0525)	-0.235 (0.297)	0.0539 (0.0552)	0.0416 (0.0725)
Birth Order	47.24*** (12.09)	-0.00822* (0.00484)	-0.0764* (0.0408)	-0.00737 (0.00641)	-0.0139* (0.00731)
Median HH Income \$	-0.00227 (0.00158)	9.66e-08 (4.35e-07)	1.20e-06 (3.80e-06)	4.00e-07 (6.72e-07)	5.38e-07 (8.82e-07)
% Pre-1950s Housing	-1,883* (1,132)	0.503 (0.408)	-7.524* (4.261)	0.953 (0.637)	0.309 (0.735)
Airport< 2000 m	-58.35** (27.49)	0.0226* (0.0125)	-0.0368 (0.145)	0.0109 (0.0132)	0.0257** (0.0115)
TRI Facility <2000m	-40.51 (33.78)	-0.0177 (0.0122)	0.0689 (0.209)	-0.0214 (0.0185)	-0.0173 (0.0231)
Observations	15,699	15,699	15,699	15,699	15,699
R-squared	0.088	0.052	0.046	0.044	0.053

BW = birth weight, LBW = low birth weight (<2,500 grams), Weeks = Weeks gestation (clinical), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age), ATT = average treatment effect of the treated.

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Robust standard in parentheses

Notes: All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Table S4. The CEM-weighted ATT of deleading on birth outcomes, Logit odds-ratios 0 – 4,000 meters: 2004-2009**

VARIABLES	LBW odds ratio	PRE odds ratio	SGA odds ratio
ATE: 4,000m	0.488*** (0.0840)	0.714** (0.117)	0.575*** (0.0927)
Mother 20-29 yo	1.362 (0.610)	1.031 (0.395)	0.950 (0.414)
Mother 30-39 yo	1.458 (0.707)	1.203 (0.483)	0.781 (0.362)
Mother >39 yo	2.489 (2.225)	1.477 (1.156)	2.310 (1.575)
Mother Black	2.432*** (0.336)	1.471*** (0.202)	3.087*** (0.426)
Mother Hispanic	1.018 (0.300)	0.933 (0.279)	1.330 (0.442)
Mother Other Race	1.789*** (0.426)	1.014 (0.261)	3.492*** (0.932)
Mother High School	1.046 (0.401)	1.172 (0.457)	1.174 (0.459)
Mother Some College	0.985 (0.414)	1.356 (0.607)	1.056 (0.542)
Mother College+	1.240 (0.536)	1.359 (0.579)	1.134 (0.531)
Father High School	1.837** (0.556)	0.887 (0.416)	0.654 (0.281)
Father Some College	1.928** (0.597)	0.914 (0.469)	0.789 (0.363)
Father College+	1.602 (0.516)	0.675 (0.360)	0.598 (0.268)
Father Educ. Missing	2.706** (1.091)	0.992 (0.572)	1.062 (0.431)
Male Infant	0.924 (0.106)	0.935 (0.103)	0.891 (0.107)
Smoked	2.288 (1.248)	1.895 (0.964)	1.739 (1.210)
Birth Order	0.854* (0.0789)	0.904 (0.0765)	0.819* (0.0873)
Median HH Income \$	1.000 (8.15e-06)	1.000 (9.62e-06)	1.000 (9.94e-06)
% Pre-1950s Housing	24.933 (178,384)	602,741 (5.084e+06)	22.81 (221.2)
Airport < 2000 m	1.839** (0.476)	1.251 (0.242)	1.402** (0.228)
TRI Facility <2000m	0.772 (0.168)	0.779 (0.178)	0.836 (0.251)
Observations	15,419	15,632	15,559

BW = birth weight, LBW = low birth weight (<2,500 grams), Weeks = Weeks gestation (clinical), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age), ATT = average treatment effect of the treated

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Robust standard in parentheses

Notes: All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Table S5. The CEM-weighted ATT of deleading by trimesters of exposure, 0 – 4,000 meters: 2004 - 2009**

Trimester of Exposure	Birth Weight (grams)	Low Birth Weight	Gestational Age (Weeks)	Preterm	Small for Gestational Age
Trimester 1	469.173*** (219.346, 719.000)	-0.0687 (-0.182, 0.044)	1.859*** (0.830, 2.887)	-0.222*** (-0.390, -0.054)	-0.0649 (-0.246, 0.117)
Trimester 2	64.569 (-15.790, 144.928)	-0.0345* (-0.072, 0.003)	-0.0224 (-0.269, 0.224)	0.0136 (-0.037, 0.065)	-0.0640* (-0.130, 0.002)
Trimester 3	193.126** (36.673, 349.580)	-0.127* (-0.272, 0.018)	0.827* (-0.064, 1.719)	-0.129** (-0.229, -0.028)	-0.0402 (-0.124, 0.044)
Trimester 1 & 2	606.773*** (389.912, 823.634)	-0.1485*** (-0.296, -0.002)	2.277*** (1.008, 3.547)	-0.219*** (-0.421, -0.016)	-0.113 (-0.334, 0.108)
Trimester 1 & 3	691.432*** (376.929, 1005.935)	-0.2168*** (-0.328, -0.106)	2.819*** (1.977, 3.661)	-0.328*** (-0.451, -0.203)	-0.111 (-0.292, 0.069)
Trimester 2 & 3	311.080** (100.720, 521.439)	-0.1691*** (-0.293, -0.046)	0.968* (-0.054, 1.991)	-0.131*** (-0.217, -0.044)	-0.189*** (-0.323, -0.054)
Trimester 1 & 2 & 3	770.838*** (413.641, 1128.036)	-0.1733*** (-0.046, -0.050)	2.353*** (1.019, 3.687)	-0.313*** (-0.507, -0.118)	-0.241*** (-0.470, -0.011)
Observations: 15,695					

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

ATT = average treatment effect of the treated.

Notes: All regressions adjust for mother's age, mother's race, mother's education, father's education, male infant, whether the mother smoked during pregnancy, birth order, proximity to TRI lead emitting facility, proximity to airport, tract-level median household income, and tract-level share of pre-1950's homes. All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Table S6: The CEM weighted ATT of deleading on birth outcomes for alternate treatment areas 0 – 3,000 meters and 0 – 5,000 meters: 2004-2009**

Birth Outcome	ATT
Treatment Area: 0 – 3,000 m	
Birth Weight (grams)	183.400** (74.197, 292.603)
Low Birth Weight (< 2500 g)	-0.0267 (-0.100, 0.047)
Gestational Age (Clinical weeks)	0.321 (-0.222, 0.864)
Preterm (< 37 weeks)	0.0364 (-0.018, 0.090)
Small for Gestational Age	-0.0884** (-0.132, -0.045)
Observations: 8,505	
Treatment Area: 0 – 5,000 m	
Birth Weight (grams)	65.206** (14.820, 115.592)
Low Birth Weight (< 2500 g)	-0.0169 (-0.035, 0.001)
Gestational Age (Clinical weeks)	0.140 (-0.049, 0.328)
Preterm (< 37 weeks)	-0.0191 (-0.042, 0.004)
Small for Gestational Age	-0.0273 (-0.064, 0.009)
Observations: 20,961	

ATT = average treatment effect of the treated

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Notes: All regressions adjust for mother's age, mother's race, mother's education, father's education, male infant, whether the mother smoked during pregnancy, birth order, tract-level median household income, and tract-level share of pre-1950's homes. All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Table S7: The CEM-weighted ATT of deleading on birth outcomes, Logit odds-ratio for alternate treatment areas 0 – 3,000 meters and 0 – 5,000 meters: 2004-2009**

Birth Outcome	Odds Ratio (ATT)
Treatment Area: 0 – 3,000 m	
Low Birth Weight (< 2500 g)	0.644 (0.189, 2.188)
Observations: 8,167	
Preterm (< 37 weeks)	1.658 (0.676, 4.066)
Observations: 8,352	
Small for Gestational Age	0.336*** (0.211, 0.535)
Observations: 8,331	
Treatment Area: 0 – 5,000 m	
Low Birth Weight (< 2500 g)	0.760* (0.567, 1.020)
Observations: 20,495	
Preterm (< 37 weeks)	0.790 (0.596, 1.047)
Observations: 20,669	
Small for Gestational Age	0.686 (0.420, 1.122)
Observations: 20,711	

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

ATT = average treatment effect of the treated

Notes: All regressions adjust for mother's age, mother's race, mother's education, father's education, male infant, whether the mother smoked during pregnancy, birth order, tract-level median household income, and tract-level share of pre-1950's homes. All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Table S8. CEM-weighted ATT, by trimester of exposure: 0 – 3,000 meters**

Trimester of Exposure	Birth Weight (grams)	Low Birth Weight	Gestational Age (weeks)	Preterm	Small for Gestational Age
Trimester 1	494.262*** (264.060, 724.463)	-0.011 (-0.130, 0.108)	0.907** (0.157, 1.658)	-0.076 (-0.223, 0.072)	-0.203*** (-0.356, -0.051)
Trimester 2	101.734 (-80.140, 283.608)	0.028 (-0.051, 0.108)	0.100 (-0.427, 0.626)	0.075*** (0.032, 0.118)	-0.085** (-0.164, -0.006)
Trimester 3	423.386* (-55.433, 902.204)	-0.256 (-0.602, 0.090)	1.580* (-0.288, 3.448)	-0.182 (-0.405, 0.041)	0.045 (-0.144, 0.235)
Trimester 1 & 2	745.629*** (304.385, 1,186.873)	0.003 (-0.209, 0.215)	1.525 (-0.494, 3.545)	0.010 (-0.227, 0.247)	-0.252*** (-0.427, -0.078)
Trimester 1 & 3	938.239*** (415.366, 1,461.112)	-0.284 (-0.644, 0.076)	2.449** (0.321, 4.576)	-0.177 (-0.465, 0.111)	-0.233 (-0.541, 0.075)
Trimester 2 & 3	716.589** (126.253, 1,306.924)	-0.256 (-0.655, 0.144)	1.449 (-1.151, 4.049)	0.040 (-0.185, 0.265)	-0.264** (-0.471, -0.057)
Trimester 1 & 2 & 3	1,326.889*** (402.793, 2,250.986)	-0.159 (-0.483, 0.164)	1.587 (-1.715, 4.889)	-0.043 (-0.355, 0.270)	-0.708*** (-1.041, -0.376)
Observations: 8,503					

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

ATT = average treatment effect of the treated

Notes: All regressions adjust for mother's age, mother's race, mother's education, father's education, male infant, whether the mother smoked during pregnancy, birth order, proximity to TRI lead emitting facility, proximity to airport, tract-level median household income, and tract-level share of pre-1950's homes. All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Table S9. CEM-weighted ATT, by trimester of exposure: 0 – 5,000 meters**

Trimester of Exposure	Birth Weight (grams)	Low Birth Weight	Gestational Age (weeks)	Preterm	Small for Gestational Age
Trimester 1	342.014*** (116.573, 567.456)	-0.083* (-0.181, 0.014)	1.347*** (0.546, 2.148)	-0.217*** (-0.335, -0.098)	-0.058 (-0.166, 0.051)
Trimester 2	33.386 (-48.664, 115.437)	0.002 (-0.043, 0.046)	-0.210 (-0.521, 0.102)	0.020 (-0.024, 0.063)	-0.070** (-0.136, -0.003)
Trimester 3	50.625 (-53.674, 154.923)	-0.063* (-0.127, 0.000)	0.317 (-0.144, 0.779)	-0.079*** (-0.129, -0.028)	0.005 (-0.086, 0.096)
Trimester 1 & 2	373.945*** (154.250, 593.640)	-0.095* (-0.205, 0.014)	1.270** (0.069, 2.470)	-0.202*** (-0.352, -0.053)	-0.048 (-0.179, 0.083)
Trimester 1 & 3	418.902** (94.640, 743.163)	-0.143*** (-0.241, -0.045)	1.657*** (0.819, 2.500)	-0.273*** (-0.379, -0.168)	-0.052 (-0.187, 0.82)
Trimester 2 & 3	140.490* (-18.682, 299.662)	-0.073 (-0.155, 0.010)	0.217 (-0.530, 0.964)	-0.079** (-0.157, -0.000)	-0.143 (-0.327, 0.042)
Trimester 1 & 2 & 3	516.027*** (185.008, 847.045)	-0.120* (-0.243, 0.004)	1.120* (-0.133, 2.371)	-0.226** (-0.407, -0.045)	-0.187* (-0.397, 0.023)
Observations: 20,956					

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

ATT = average treatment effect of the treated

Notes: All regressions adjust for mother's age, mother's race, mother's education, father's education, male infant, whether the mother smoked during pregnancy, birth order, proximity to TRI lead emitting facility, proximity to airport, tract-level median household income, and tract-level share of pre-1950's homes. All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Table S10. The CEM-weighted ATT of FALSE deleading around airports on birth outcomes, 0 – 4,000 meters: 2004-2009**

VARIABLES	BW (grams)	LBW	Weeks	PRE	SGA
ATE: 4,000m	1.309 (16.64)	-0.00537 (0.00732)	-0.0532 (0.0592)	-0.00250 (0.00839)	-0.00824 (0.00735)
	8.309 (14.94)	0.00722 (0.00761)	-0.102* (0.0585)	0.00401 (0.00847)	-0.0111 (0.00869)
Mother 20-29 yo	-3.812 (18.54)	0.0260*** (0.00870)	-0.306*** (0.0731)	0.0264*** (0.00967)	-0.00556 (0.00955)
Mother 30-39 yo	-112.0** (46.37)	0.0488** (0.0244)	-0.423*** (0.132)	0.0411* (0.0218)	0.0574* (0.0295)
Mother >39 yo	-271.2*** (16.91)	0.0705*** (0.00788)	-0.412*** (0.0617)	0.0362*** (0.00761)	0.0950*** (0.00937)
Mother Black	-40.57** (19.41)	-0.0124* (0.00667)	0.178** (0.0697)	-0.0118 (0.00898)	0.00604 (0.00895)
Mother Hispanic	-210.8*** (27.27)	0.0114 (0.0121)	-0.0636 (0.101)	-0.00157 (0.0146)	0.112*** (0.0200)
Mother Other Race	30.66** (15.45)	-0.0130 (0.00825)	-0.0660 (0.0629)	-0.00236 (0.00711)	-0.0257*** (0.00941)
Mother High School	50.86** (20.11)	-0.0202** (0.00999)	-0.0849 (0.0792)	-0.00233 (0.0101)	-0.0344*** (0.0105)
Mother Some College	92.56*** (19.92)	-0.0301*** (0.0102)	-0.00317 (0.0856)	-0.00636 (0.0111)	-0.0417*** (0.0105)
Mother College+	-19.28 (18.07)	0.00850 (0.00824)	-0.103 (0.0786)	0.00962 (0.00838)	0.0114 (0.00872)
Father High School	3.722 (20.33)	0.00153 (0.00920)	-0.0312 (0.0752)	0.00772 (0.0112)	0.0126 (0.0102)
Father Some College	-2.441 (20.54)	0.00459 (0.00988)	-0.0210 (0.0885)	0.000318 (0.0118)	0.000781 (0.0113)
Father College+	-35.47* (19.82)	0.00557 (0.00923)	-0.0724 (0.0669)	0.00693 (0.00937)	0.0181 (0.0128)
Father Edu. Missing	119.3*** (7.376)	-0.0153*** (0.00327)	-0.0350 (0.0230)	0.00624** (0.00307)	-0.00277 (0.00383)
Male Infant	268.0*** (19.75)	0.0616*** (0.0113)	-0.354*** (0.0801)	0.0387*** (0.0109)	0.105*** (0.0107)
Smoked	39.14*** (4.874)	-0.00629*** (0.00225)	-0.0978*** (0.0171)	0.00196 (0.00259)	-0.0160*** (0.00217)
Birth Order	-0.000604 (0.000682)	-2.72e-07 (3.27e-07)	-1.72e-07 (2.38e-06)	-8.17e-08 (3.29e-07)	7.36e-08 (3.69e-07)
Median HH Income \$					
% Pre-1950s Housing	-251.0* (145.9)	-0.0267 (0.0817)	0.141 (0.503)	-0.0296 (0.0769)	0.112* (0.0646)
	28.85 (21.19)	-0.0137 (0.00934)	0.118 (0.0912)	-0.0153 (0.0131)	-0.00431 (0.0135)
TRI Facility <2000m	34,700	34,700	34,700	34,700	34,700
Observations					
R-squared	0.067	0.026	0.027	0.017	0.036

BW = birth weight, LBW = low birth weight (<2,500 grams), Weeks = Weeks gestation (clinical), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age), ATT = average treatment effect of the treated

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Robust standard in parentheses

Notes: All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.



**Table S11. The CEM weighted, estimated average difference in birth outcomes between the treatment control groups for treatment area 0 – 4,000 meters: 2004-2009**

	BW (grams)	LBW	Weeks	PRE	SGA
Treat*2004	-49.296 (62.727)	-0.016 (0.034)	1.011*** (0.274)	0.013 (0.035)	-0.103*** (0.016)
Treat*2005	23.047 (32.868)	-0.047*** (0.015)	1.256*** (0.110)	-0.027* (0.015)	-0.055*** (0.019)
Treat*2006	59.856*** (21.796)	-0.036*** (0.007)	1.186*** (0.060)	-0.001 (0.008)	-0.118*** (0.016)
Treat*2007	85.012** (35.870)	-0.061*** (0.021)	1.654*** (0.169)	-0.019 (0.015)	-0.108*** (0.023)
Treat*2008	109.340** (54.690)	-0.079*** (0.017)	1.359*** (0.126)	-0.025 (0.020)	-0.128*** (0.014)
Treat*2009	159.532*** (53.329)	-0.087*** (0.020)	1.579*** (0.231)	-0.057*** (0.021)	-0.160*** (0.024)
Constant	3334.773*** (23.975)	0.059*** (0.008)	38.624*** (0.092)	0.090*** (0.011)	0.088*** (0.013)
$\beta_{2004} = \beta_{2005} = \beta_{2006}$					
F-Statistic	1.46	1.21	0.42	2.09	4.34**
Prob > F	0.236	0.301	0.655	0.128	0.015

Observations: 15,699

BW = birth weight, LBW = low birth weight (<2,500 grams), Weeks = Weeks gestation (clinical), SGA = small for gestational age (birth weight is below the 10<sup>th</sup> percentile for clinical gestational age)

\*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Robust standard in parentheses

Notes: All regressions include a set of census tract, month, and year indicator variables. All regressions are weighted using coarsened exact matching weights. Standard errors are clustered on the census tract.

**Figure S1. Pre- and Post-Deleading Policy Distance Gradients.**

