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ABSTRACT

The recent lead-in-water crisis in Newark has renewed concerns about the crisis being a widespread problem in the nation. Using data on the exact home addresses of pregnant women residing in the city combined with information on the spatial boundary separating areas within the city serviced by two water treatment plants, we exploit an exogenous change in water chemistry that resulted in lead leaching into the tap water of one plant's service area, but not the other's, to identify a causal effect of prenatal lead exposure on fetal health. We find robust evidence of adverse health impacts, which has important policy implications in light of the substantial number of lead water pipes that remain in use as part of our aging infrastructure and the cost-benefit calculus of lead abatement interventions.

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1 Introduction

The recent crisis of drinking water contamination in Newark, New Jersey's largest city, has renewed concerns regarding elevated lead levels in drinking water becoming a persistent and pervasive problem owing to the nation's aging infrastructure. Indeed, the newly passed infrastructure bill (an approximately $1 trillion package) by the U.S. Congress (on November 5, 2021) allocates $15 billion over the next five years to states, tribes, and territories for replacing lead pipes in the U.S. drinking water systems.[1] Corrosion of lead plumbing materials is the most common source of lead in drinking water.[2] Although lead has been banned from use in new plumbing systems in the United States since 1986, much of the country's drinking water infrastructure largely predates this ban (Brown and Margolis, 2012; Centers for Disease Control and Prevention (CDC), 2019). Consequently, drinking water constitutes a significant source of lead exposure for Americans.[3]

Lead is known to have bio-accumulative properties, collecting over time in the human body through repeated exposure and stored in the bones alongside calcium. Of particular concern is in utero exposure, since accumulated lead in a mother's bones can be mobilized during pregnancy and released as a calcium substitute to aid in the formation of the bones of the fetus (Gulson et al., 1997; Hu and Hernandez-Avila, 2002). Lead in a mother's blood can also easily cross the placenta, directly exposing the fetus to lead poisoning (Al-Saleh et al., 2011). There is no safe threshold of lead exposure that has been identified for children (American Academy of Pediatrics (AAP), 2016; CDC, 2019; EPA, 2020). Lead is a potent neurotoxin, and prenatal lead exposure is associated with impaired neurodevelopment, plac-

[1] See https://www.wsj.com/articles/how-the-1-trillion-infrastructure-bill-aims-to-affect-americans-lives-11636173786 for details (accessed on November 10, 2021).

[2] For more details, see https://www.cdc.gov/nceh/lead/prevention/sources/water.htm (accessed on June 22, 2020).

[3] The Environmental Protection Agency (EPA) estimates that drinking water can account for 20 or more percent of total lead exposure for adults and 40 to 60 percent for infants (EPA, 2020). Other modes of exposure occur through other forms of ingestion (e.g., food and chipped lead paint) and inhalation (e.g., tobacco smoking, emissions from leaded gasoline, and industrial pollution). Dermal absorption, mainly through occupational exposure for workers directly handling or working in proximity to lead materials, is also possible.

ing exposed children at higher risks for cognitive impairment, reduced IQ, learning disability, behavioral problems and other functional difficulties (CDC, 2010; WHO, 2011).

Drinking water contamination is becoming an increasingly important and widespread source of prenatal exposure to environmental pollution. Between 2018 and 2020, nearly 30 million people received their drinking water from community water systems that were in violation of the EPA's Lead and Copper Rule, which sets maximum enforceable levels of these metals in drinking water (Fedinick, 2021). Almost a third of community water systems report that at least some of their public service pipelines contain lead, with the exact number of lead service lines estimated to be between 6.1 and 10.2 million (Cornwell, Brown and Via, 2016; EPA, 2016a).[4] Moreover, these lead service lines contribute as much as 75 percent of the lead that seeps into tap water (Sandvig et al., 2008).

In this study, we leverage a unique natural experiment provided by the 2016 water crisis in Newark, in order to identify the causal effect of prenatal exposure to lead-contaminated drinking water on fetal health. Specifically, we compare two groups of mothers whose homes are served by Newark's two water treatment plants, respectively, over the period prior to and subsequent to the first discovery of lead contamination of drinking water in the city, which happened in March 2016.[5] Of the two groups, one was exposed to elevated lead levels in drinking water because of an unintended consequence of one water treatment plant's decision made in 2015 to increase the acidity level of its treated water. The unintended consequence of this increase in the acidity level was reduced effectiveness of the corrosion inhibitor (sodium silicate) used by the plant to control lead release. This caused lead from the pipes and plumbing fixtures to seep into the water, thereby exposing homes serviced by

[4] Service lines are pipes that connect residences with the water mains (i.e., pipes delivering the water supplied to a city or town). There does not exist a complete national inventory of lead service lines to date, and estimates are uncertain (Government Accountability Office, 2018). Following the water crisis in Flint, Michigan, the EPA encouraged all states in February 2016 to work with water systems to conduct inventories of lead service lines. Noted challenges include lead service lines on private property, which makes them difficult to locate, as well as a lack of records about the locations of older lead service lines.

[5] Our identification strategy is enlightened by Snow (1855), who compares locations that are near one another but with different sources of water supply (resulting in different exposure to water contamination) to study the cause of the cholera outbreak in London.

this water treatment plant to significantly elevated levels of lead in their tap water.

Using data on all live births in New Jersey between 2011 and 2019, we estimate the effects of prenatal exposure to lead contamination in drinking water on birth outcomes. A unique feature of our study is that we have data on the exact home addresses of pregnant women and the information on the spatial boundary of service areas between Newark's two water treatment plants. We find robust evidence that prenatal lead exposure significantly raises the probability of low birth weight (LBW, birth weight $<$ 2,500 grams) by about 1.5 percentage points (or 18 percent) as well as the probability of a preterm birth (gestational length $<$ 37 weeks) by about 1.9 percentage points (or 19 percent). There is little indication that these effects are driven by selection into births. We also find some notable dynamics in the response that coincide with how the Newark water crisis unfolded, and some evidence indicative of mitigation behavior including pregnant women in affected areas seeking out greater prenatal care in response to the water crisis. The effects on fetal health that we capture are the ones of a shift in water quality, including biological effects and effects of avoidance or compensatory behaviors conditional on any increased use of prenatal care which we control for.

Our study is directly relevant in informing how lapses in water quality due to a failure in the water system's infrastructure—which have become increasingly more prevalent and a target of recent policy proposal—translates into population ill-health. The extent of these population-level health effects remains unclear, as they depend on avoidance behaviors of the mother, the level of exposure, and other factors that may regulate the body's absorption of lead (such as micronutrient inadequacies or maternal health endowment). Moreover, lead exposure among women of childbearing age has fallen substantially over the past four decades with regulations limiting lead emissions (Ettinger et al., 2020). With average blood lead levels being quite low in the modern era, it is uncertain how a shock to water quality, which exposes these mothers to higher levels of lead, would affect their pregnancy outcomes.[6]

[6] This would depend on the non-linearity and shape of the "damage function" linking the level of lead exposure and health production, based on baseline exposure, for which there is very limited evidence in the literature.

Our study makes several contributions to the literature. We provide the first evidence on the impact of the Newark water crisis on birth outcomes. In the process, we add to the very limited evidence base on the adverse effects of water pollution,[7] and specifically on the effects of prenatal exposure to lead in drinking water—a dearth underscored by Keiser and Shapiro (2019) and by the CDC in its report on lead exposure among pregnant women (CDC, 2010).[8] Even recent evidence from the Flint water crisis on infant health outcomes has been somewhat mixed (Abouk and Adams, 2018; Grossman and Slusky, 2019; Wang, Chen and Li, 2021). In contrast to these studies of the Flint water crisis, which rely on intra-state comparisons between Flint and other cities in Michigan, the nature of the cause of the water crisis in Newark allows us to exploit plausibly exogenous within-city variation across affected and non-affected households within Newark.

While Newark provides the natural experiment in this study, failure to upgrade the nation's aging water infrastructure has made lead in the water system a national problem, and prompted predictive warnings that Newark's lead-water crisis will not be the nation's last (Khazan, 2019).[9] Our study also broadly contributes to the fetal origins literature, regarding effects of in utero shocks on health (Almond and Currie, 2011; Barker, 1995). With fetal health being an important predictor of later-life outcomes, these estimates are critical towards evaluating the cost-benefit calculus of infrastructure investments, including

[7] Regarding the literature on the causal effects of early-life exposure to pollution, the majority of that literature has been about air pollution, for which Currie et al. (2014) provide detailed reviews.

[8] The CDC notes that research on prenatal lead exposure and LBW is inconclusive and "[f]urther research is needed for a better understanding of several biomedical issues, including pregnancy outcomes and infant development associated with maternal lead exposure during pregnancy" (CDC, 2010, p. iii). While there is a large literature on the health effects of lead exposure (e.g., Bellinger, 2005; CDC, 2010; Gardella, 2001; WHO, 2011), much of this literature is correlational and based on relatively small or selected samples. The better of these studies are longitudinal and prospective (see WHO, 2011). Furthermore, much of the work on children is based on relatively high blood levels of lead (Aizer et al., 2018). Given that lead exposure and blood lead levels among children and mothers (and in the general population) have decreased in the United States (Brown and Margolis, 2012; CDC, 2010) over the past 40 years, it is important to understand how lead contamination affects health in a population that has on average low baseline blood levels of lead that are common today.

[9] Several large cities are served by water systems that have recently exceeded the EPA's action levels for lead, including Baltimore (MD), Chicago (IL), Detroit (MI), Green Bay (MI), Jackson (MS), Milwaukee (WI), New York (NY), Pittsburgh (PA), Portland (OR), Providence (RI), and Washington D.C. (Bendix, 2020; Fedinick, 2021; Mulvihill, 2021).

replacing all of the nation's lead service lines, an initiative supported by the EPA (EPA, 2019) and a need finally recognized in the newly passed infrastructure bill by the U.S. Congress in November 2021.

2 Background

2.1 Lead and EPA Compliance

Lead was widely used in water pipes and plumbing because of its strength, durability and malleability.[10] When public water systems were designed in the United States, lead became the material of choice, and lead service lines, which are used to connect homes and buildings to the water main, were widespread; virtually all large public water systems in the United States had installed lead service pipes (Rabin, 2008; Troesken, 2008).[11] As public health concern regarding adverse effects of lead exposure intensified in the 1960s and 1970s, the Safe Drinking Water Act (SDWA) was passed in 1974, giving the EPA authority to set and enforce limits on levels of lead and other contaminants in drinking water (Dignam et al., 2019). Interim standards were set in 1975 for lead concentration in drinking water to be below 50 $\mu g/L$ (i.e., 50 parts per billion or ppb). The 1986 amendment to the SDWA banned the use of lead from all new plumbing materials.[12] In 1991, the EPA's Lead and Copper Rule (LCR) established a lower threshold of 15 ppb for the maximum contaminant level (MCL) for lead in drinking water at customer taps, which is an actionable and enforceable level.[13]

[10] The symbol for the chemical element, Pb, is derived from the Latin word "plumbum", referencing back to ancient times when the metal was widely used in the construction of water pipes.

[11] See Rabin (2008) for a history of lead water pipes and the influence of the lead industry in the U.S..

[12] Lead in residential paint was banned in 1978. A gradual phase-out of lead content in gasoline began in 1973, and lead was virtually eliminated from gasoline by 1988 (Brown and Margolis, 2012).

[13] The EPA's maximum contaminant level goal (MCLG) for lead is zero, consistent with the best available evidence that there is no safe level of exposure to lead; however, this goal is neither actionable nor enforceable as reducing lead levels to zero would be prohibitively costly and may not be possible. MCLs are set as close as possible to MCLG, at levels that are economically and technically feasible. States can set more stringent standards if they choose, but most, including New Jersey, follow the EPA's standards. The maximum allowable lead level for bottled water, set by the Food and Drug Administration, is 5 ppb. For more details, see https://www.epa.gov/ground-water-and-drinking-water/basic-information-about-lead-drinking-water and https://www.atsdr.cdc.gov/csem/csem.asp?csem=34&po=8 (accessed on June 22, 2020).

As lead contamination of drinking water can result from corrosion of plumbing materials, community water systems are required to follow accepted treatment techniques to contain the corrosiveness of the water. Public water systems are generally required to monitor compliance with the EPA's LCR once every three years, by testing first-draw samples at taps in homes and buildings in the service area, including those deemed to be at high risk of contamination.[14] Required actions for non-compliance are triggered when more than 10 percent of the sampled customer taps exceed the 15 ppb MCL for lead. Utilities are required in this case to accelerate their monitoring to consecutive 6-month cycles, undertake further steps to optimize corrosion control until water quality improves, and educate customers about lead in drinking water and actions they can take to reduce their exposure to lead in the meantime. Water systems that continue to exceed the MCL for lead even after installing corrosion control must then start replacing the lead service lines (at a rate of at least 7 percent annually) until compliance is achieved (EPA, 2008).

2.2 Newark Water Crisis

Newark is the most populous city in the state of New Jersey (with a population of approximately 283,000.[15] It is also one of the oldest cities in the nation, with water supply to residents sourced and serviced through two distribution systems: the Pequannock Water Treatment Plant (WTP) and the Wanaque WTP (shown in Figure 1).

The first indication of elevated lead levels in Newark appeared in March 2016, when an annual program of spot testing and a subsequent new round of testing[16] revealed that 30 Newark public schools recorded lead levels in drinking water above the MCL (15 ppb).[17]

[14] Sampling sites consist of single and/or multiple family structures that are served by a lead service line and/or contain copper pipes with lead solder. For details, see EPA (2008) and https://www.govinfo.gov/app/details/CFR-2011-title40-vol23/CFR-2011-title40-vol23-sec141-86 (accessed on June 22, 2020).

[15] See Appendix Table A1.

[16] For details, see https://www.nytimes.com/2016/04/01/nyregion/lead-in-newark-schools-water.html (accessed on March 31, 2021).

[17] The Newark School District consists of 66 schools (see https://www.nps.k12.nj.us/info/ for details). Almost a quarter of the samples (76 out of 324) tested above the MCL. Follow-up results from additional samples found that 19 percent (735 out of 3,922) tested at elevated lead levels. For a summary and timeline

Newark public schools receive water from the same sources as the rest of the city. In 2017, under a mandate from the New Jersey Department of Environmental Protection, Newark switched its monitoring to testing drinking water for contamination twice a year; until then, it was on a triennial monitoring cycle with the previous tests being carried out between 2013 and 2015. The first test results, under the new frequent monitoring cycle and based on tap water samples from residences throughout the city, indicated sharply elevated lead levels for the first half of 2017. At least 22 percent of drinking water samples citywide exceeded the EPA's MCL of 15 ppb. However, most of the lead-contaminated samples were concentrated in the western part of the city that receives water treated by the Pequannock WTP. In this service area, 32 percent of samples contained lead levels exceeding 15 ppb (the EPA standard) and 44 percent exceeded 10 ppb (the European Union and the WHO standards); in contrast, samples from residences in the eastern part of the city where sourced water is treated by the Wanaque WTP continued to show compliance (only 6.5 percent of samples—below the EPA's 10-percent trigger—tested positive for lead levels exceeding 15 ppb or even 10 ppb). As we discuss below, elevated lead levels in drinking water for some residences, but not others, were the result of the two water treatment plants relying on different chemical agents for corrosion control (i.e., corrosion inhibitors). An increase in the acidity level of water treated by the Pequannock WTP reduced the effectiveness of the corrosion inhibitor (sodium silicate) that it was using, exposing homes serviced by this WTP to significantly higher levels of lead in their tap water. This was the conclusion reached in a study conducted by an independent engineering firm, commissioned by the city to investigate the cause of the elevated lead levels in Newark upon receiving the notice of non-compliance with the EPA's LCR; the report was released in October 2018 (City of Newark, 2018).[18] By then, Newark had continuously violated the EPA's actionable MCL for lead since the start of the frequent

of the Newark drinking water crisis, see City of Newark (2018); Corasaniti, Kilgannon and Schwartz (2019); McGeehan (2016); and https://www.nrdc.org/newark-drinking-water-crisis (accessed on June 22, 2020). This section draws information from these sources.

[18] Independent testing by the firm (CDM Smith) suggests that some residents may have been exposed to lead levels even higher than those reported in the city's testing samples.

biannual monitoring cycle.

In the city's 2017 annual water quality brochure, mailed to all residents as required by law, Mayor Ras Baraka reassured residents on the first page that "[m]any of you have heard or read the outrageously false statements about our water but please know that the quality of our water meets all federal and state standards" (City of Newark, 2017).[19] Under the pressure of litigation from several groups, and with the release of the city-commissioned study results on the extent and cause of the lead contamination, Newark started distributing water filtration devices in October 2018 to residents in the Pequannock service area.[20]

Until this time, the public remained largely unaware of the full extent of the water contamination. As pointed to by trends in Google search queries related to the water crisis in Newark (Figure 2), the first significant spike in interest coincided with reports of elevated lead levels in Newark public schools (in March 2016),[21] which faded within a month or two. The next major spike in interest occurred in October 2018 with the city's plans to distribute water filters to impacted residents.

In March 2019, Newark commenced a program to remove and replace all of the city's lead service lines in the water system at no cost to the homeowner.[22] The Pequannock WTP switched its corrosion inhibitor on May 7, 2019 from sodium silicate, which had become

[19] The 2017 Report noted on the first page that the only high lead readings were confined to older homes. Results of the lead testing showing non-compliance with the EPA's MCL for lead were included at the end of the report (p. 5 and p. 7).

[20] Water filters could be picked up at various distribution centers. Community organizations and city employees also canvassed homes in the Pequannock service area and delivered water filters to those with suspected lead service lines. As of August 2019 and by the city's estimate, some 38,000 water filters had been distributed since October 2018. For detail, see https://www.newarknj.gov/news/faqs-regarding-the-city-of-newarks-water-filters-efforts-to-address-lead-in-the-water (accessed on June 22, 2020).

[21] For example, on March 9, 2016 the Associated Press published an article in *The New York Times*, titled "Elevated Lead Levels Found in Newark Schools' Drinking Water" (https://www.nytimes.com/2016/03/10/nyregion/elevated-lead-levels-found-in-newark-schools-drinking-water.html, accessed in December 2019).

[22] The city's lead service line inventory, undertaken in response to its EPA's LCR violations, shows 18,406 (out of 29,938) of its service pipes were lead (source: https://www.nj.com/essex/2019/08/newarks-handing-out-bottled-water-what-you-need-to-know-about-the-citys-lead-crisis.html, accessed on September 9, 2019). Replacement was originally intended to take place over eight years, with costs shared between the city and the homeowner, but was accelerated in September 2019 to be completed within 24 to 30 months at no cost to the homeowner. By May 2020, the city had replaced about 10,000 of its lead service lines.

ineffective, to orthophosphate, the same chemical agent used by the Wanaque WTP, which services the eastern part of the city. As it takes at least six months or longer for the orthophosphate to start working, elevated lead levels in water serviced by the Pequannock WTP continued through 2019. Among samples tested from this area in the second half of 2019, 26.9 percent (38.4 percent) contained lead levels in excess of 15 ppb (10 ppb); in contrast, samples from the Wanaque WTP service area continued to show compliance as in all prior periods.[23] Following tests showing elevated lead levels even among homes using the distributed filters, the EPA warned that the filtration devices might not be adequately eliminating lead, and it instructed the city in August 2019 to provide bottled water to its impacted residents.[24] Figure 2 shows the largest spike in Google search queries related to water contamination in Newark at this time, coinciding with this EPA order and the distribution of bottled water to residents.

2.3 Prior Studies

General Lead Exposure

Lead is a poison, and high levels of lead in blood affect nearly all of the body's organs, with the brain particularly susceptible to its damaging effects. Exposure to lead is associated with adverse neurological, renal, hematological, endocrine, gastrointestinal, cardiovascular, reproductive, and developmental effects (ATSDR, 2007; ATSDR, 2017; WHO 2011). Lead is readily transferred from the mother to the fetus throughout gestation via the placenta. As lead hinders absorption of iron, zinc and calcium, which are essential to proper neurological development, lead exposure in utero (and through breastfeeding) can have lasting adverse health effects, independent of additional exposure at other stages of the life cycle (CDC,

[23] Source: City of Newark (2018) and New Jersey Drinking Water Watch from the New Jersey Department of Environmental Protection (https://www9.state.nj.us/DEP_WaterWatch_public/index.jsp, accessed in February 2020).

[24] Bottled water was available to residents in the Pequannock service area through distribution centers, with assistance offered to residents unable to pick up in person. A later report by the city in November 2019 confirmed that the distributed filters were often improperly installed or maintained, diminishing their effectiveness in removing the lead.

2010).

Most prior epidemiological studies have found measures of blood lead levels to be correlated with health outcomes, cross-sectionally or longitudinally, based on small selected samples. However, those studies are unable to rule out other confounding factors associated with lead exposure (CDC, 2010; Grossman and Slusky, 2019; WHO, 2011). That is, those studies consider the direct association between high blood lead levels and health outcomes, rather than the effects of exposure to lead, and estimate a "treatment-on-the-treated" effect where variation in the "treatment" is not necessarily exogenous. Although some studies suggest that higher maternal blood lead levels may reduce birth weight, results are mixed and inconclusive (Bellinger, 2005; Gardella, 2001). There is also some evidence that maternal lead exposure may increase the risk of a miscarriage, although the most reliable evidence comes from a population of women with baseline blood lead levels substantially higher than the current mean for the U.S. women.[25] Surveys of the epidemiological literature on lead and pregnant women generally qualify that these studies may not have adequately controlled for confounding factors, and further research is warranted (Bellinger, 2005; CDC, 2010; Gardella, 2001).

More recent work has exploited natural experiments and more plausible exogenous variation in lead exposure to identify its health and developmental effects.[26] Using data linking preschool blood lead levels and school records in Rhode Island, with a multitude of identification strategies including sibling variation, residential proximity to roads and de-leading of gasoline, and policies requiring landlords to ensure that rental homes are lead-free, Aizer and Currie (2019) and Aizer et al. (2018) find that higher lead exposure results in greater anti-social behaviors, and lower reading and math achievement among children, respectively. Billings and Schnepel (2018) link data on children's blood lead levels with school and ar-

[25] See Bellinger (2005), Borja-Aburto et al. (1999), Edwards (2014), and Hertz-Picciotto (2000).

[26] Klemick, Mason and Sullivan (2020) use data on blood test results for children in six states and exploit residential proximity to Superfund cleanup sites to estimate effects of reduced exposure on blood lead levels. They find that Superfund cleanups lowered the risk of elevated blood lead levels by 13–26 percent for children living within 2 km of lead-contaminated sites.

rest records in North Carolina. Comparing children whose blood lead levels are just above and below the cutoff at which children become eligible for lead remediation interventions, the study finds that reducing lead exposure through such early-life interventions improves children's anti-social and educational outcomes and reduces criminal activity. Drawing on variation in airborne lead across counties, driven by the Interstate Highway System and compliance with the 1977 Clean Air Act (CAA) Amendments, Clay, Portnykh and Severnini (2018) find that reduced exposure to lead in the air increased completed fertility (as measured by children ever born) and improved birth weight. Hollingsworth et al. (2020) leverage NASCAR's 2007 switch from leaded to unleaded gasoline, in conjunction with distance to racetracks, to find large adverse effects of exposure to atmospheric emissions on children's academic performance.

Exposure to Lead Contamination in Water

As atmospheric lead emissions have declined since the CAA and regulations restricting leaded gasoline, lead exposure through the water system has taken on added relevance. Keiser and Shapiro (2019) provide excellent discussions of the history of regulating water pollution in the United States, notably through the Clean Water Act of 1972 and SDWA of 1974, and the effectiveness of these regulations in decreasing surface water pollution. They also draw attention to the dearth of economic research on water pollution, noting as important challenges the limited availability of data on water quality, hurdles with causal inference, and difficulty in focusing on and disentangling the effects of specific pollutants.

As such, research on the effects of lead exposure through drinking water on fetal health has been very limited. Clay, Troesken and Haines (2014) find higher rates of infant mortality historically, over 1900–1920, in American cities with more lead pipes and more acidic water, which would have resulted in greater corrosion and exposure to lead. Similarly, Troesken (2008) finds higher infant mortality and stillbirth rates in cities in Massachusetts at the turn of the 20th century that used lead pipes and had acidic water. Currie et al. (2013) use data on birth records in New Jersey during the period of 1997–2007, matched with water district-level EPA's MCL violations for any chemical and/or bacterial contaminant, to identify the effects of contaminated drinking water on fetal health. Exploiting variation across births for the same mother (i.e., using mother fixed effects), they find that residing in a water district with contaminated water during pregnancy is associated with an increase in LBW (by 14.5 percent) and prematurity (by 10.3 percent) among low-educated mothers. Their study, however, does not specifically identify the effects of lead or any particular contaminant.

Evidence from Recent Drinking Water Crises

More recently, researchers have studied the effects of water contamination crises in Washington, D.C. and Flint, Michigan. Edwards (2014) compares outcomes in Washington, D.C., which experienced high levels of lead in drinking water during 2000–2004, using neighboring City of Baltimore as a control, and finds an increase in fetal death rates and a decrease in birth rates during the crisis period.[27]

The Flint water crisis began in 2014, after the city changed its water source to the Flint River and failed to apply corrosion inhibitors to the water. Studies that have assessed the effects of the Flint water crisis on fetal health generally conclude that greater exposure to lead in drinking water adversely impacted birth outcomes, although findings are not uniform. Grossman and Slusky (2019) find a decrease in birth rates, though no significant effects on birth weight or gestation. They interpret the reduction in birth rates as driven by an increase in miscarriages, which would imply that births carried to term may be a selected healthy sample biasing against finding negative effects on birth weight or gestation. They also find suggestive evidence that the reduction in birth rates is not driven by behavioral changes related to conception, such as reduced sexual activity. Nonetheless, it is also possible that the reduction in birth rates could be driven by a reduction in conception due to adverse reproductive effects of lead exposure for both the mother and the father (ATSDR, 2017;

[27] Lead was inadvertently released from plumbing materials into drinking water starting in 2000 due to a switch in drinking water disinfectant from chlorine to chloramine.

WHO, 2011). In contrast, Abouk and Adams (2018) find a significant reduction in birth weight and a higher incidence of LBW, though only among white mothers. Wang, Chen and Li (2021) also find an increase in LBW, though in contrast to Abouk and Adams (2018), they find a larger effect on LBW among babies born to black mothers, and in contrast to Grossman and Slusky (2019), they find little evidence of an increase in fetal deaths. All three studies use 20 months of post-treatment data, while differing somewhat in the control cities and counties compared against the city of Flint.

Contributions

To the best of our knowledge, we provide the first study of how the drinking water crisis in Newark has affected birth outcomes. The source of Newark's water crisis is fundamentally different from the sources of other recent high-profile water crises that happened in Washington D.C. and Flint of Michigan. The D.C. water crisis was caused by a change in the drinking water disinfectant being used, and the Flint water crisis was caused by a change in the water source and failure to use any anti-corrosion agent at all with the new water source. In contrast, our study examines a case where there are no changes in the water source and no changes in the chemical agents used for water treatment, but rather the ineffectiveness of a currently used anti-corrosion agent due to an increase in the acidity level of the treated water—a decision made by the water treatment plant to reduce disinfection byproduct (e.g., carcinogens) formation. Furthermore, the Flint water crisis was much more condensed in its timeline, and fast-moving in the diffusion of the information shock into the public sphere, in contrast to the Newark water crisis.[28]

Our study broadly contributes to the limited literature on the causal effects of water

[28] The city of Flint switched its water source to the Flint River on April 25, 2014, which is considered to be the start of the water crisis there, and reconnected back to the original source—the Detroit water system—on October 16, 2015. Soon after the start of the crisis, Flint residents began complaining of discolored and foul-smelling water, attributed to high levels of fecal coliform bacteria in the water. In contrast to the Newark water crisis, with the Flint water crisis it was thus relatively apparent that something was wrong with the water, which may have led residents to also engage in greater avoidance behaviors. The city of Flint issued a water boiling advisory on August 14, 2014. The Flint water crisis also coincided with an outbreak of Legionnaire's disease, and the water supply change was considered a possible cause of this outbreak.

contamination, and specifically to the scarce and mixed evidence base on the effects of prenatal lead exposure on fetal health. The unique cause of the lead crisis in Newark allows us to exploit plausibly exogenous within-city variation, across impacted and non-impacted mothers, to identify causal effects. In contrast, the nature of the Flint water crisis necessitated comparing Flint to control cities and counties, with findings apparently sensitive to the choice of these controls. Since we have data on the mother's exact residential address, unlike the Flint studies, we can control for residential address fixed effects and separate out effects of elevated levels of lead in drinking water (natural experiment) from the effect of having lead pipes at home (past exposure) or from time-invariant heterogeneities in environmental exposure at the residential address level. The timeline of the Newark crisis, wherein residents remained largely unaware of the full scope of the lead contamination for at least the first one to two years post-contamination, allows us to trace out dynamics of the health effects of lead exposure in the presence of stress responses and possible, but likely insufficient, avoidance or compensatory behaviors aimed at mitigating the health risk. Finally, we note that while Newark provides the natural experiment in this study, the nature of the water contamination (corrosion of lead service lines) is common, with many community water systems in the United States being in violation of the EPA's MCL for lead. The estimates from our study can be used to inform a cost-benefit calculus of public investments in eliminating exposure from lead pipes, an initiative supported by the EPA and many localities, and we provide such calculus in the case of Newark.

3 Empirical Framework

3.1 Data

We use the restricted version of the birth certificate data from the New Jersey Department of Health (NJDOH) for this study. The data include all live births that occurred in New Jersey between 2011 and 2019. In addition to the information typically reported in vital statistics data, such as birth outcomes and mothers' demographic characteristics, which are publicly available through, for example, the U.S. National Center for Health Statistics (NCHS), the NJDOH data we obtained contain information on mothers' home addresses, geocoded by latitudes and longitudes.[29] This information allows us to include residential address fixed effects in our estimation, to disentangle the effect of elevated levels of lead in drinking water from the effect of having lead pipes at home, and also identify mothers in the impacted and non-impacted parts of the city. We limit our analysis to singleton births (about 96 percent of the NJDOH birth data), to avoid confounding factors causing adverse birth outcomes that are specifically related to carrying multiple fetuses in one pregnancy. There are 838,337 singleton live births in New Jersey over our analysis period, with 36,173 singleton live births occurring in Newark. Among the total number of singleton live births that occurred in Newark between 2011 and 2019 used in our regression analysis (which is 34,276),[30] the number of unique residential addresses is 2,810. Note that each address is geocoded by a pair of latitude and longitude coordinates, and this pair of coordinates can apply to multiple people who live, for example, in the same structure such as an apartment building or multi-family dwelling.[31]

Appendix Table A1 compares Newark to the U.S. as a whole and to similar sized cities, based on 2016–2019 data from the American Community Surveys (ACS), on key socioeconomic characteristics.[32] Median household income in Newark is $44,000 (39 percent lower than the U.S. average), and the poverty rate in the city (27.1 percent) is roughly double. Newark is predominantly black (50.2 percent) and low-educated (63.7 percent of residents

[29] In contrast, the lowest level of geography identified in the vital statistics data available at the U.S. NCHS is the county and city (for cities with at least 100,000 population).

[30] Out of 36,173 total number of singleton live births, our estimation sample comprises 34,276 births for which we have non-missing information for all of the variables used in the regression models.

[31] In our estimation sample that includes birth years 2011–2019, only 241 observations (i.e., 0.7% of the sample) do not have repeated pairs of latitudes and longitudes, and these observations do not contribute to the identifying variation in the regression model that controls for residential address fixed effects.

[32] Identification of cities in the ACS data is incomplete; statistics in the last column of Appendix Table A1 correspond to the following cities identified in the ACS with a population within +/- 10% of the population in Newark: Anchorage, AK; Buffalo, NY; Jersey City, NJ; Laredo, TX; Lincoln, NE; Pittsburgh, PA; Saint Louis, MO; Saint Paul, MN; Toledo, OH.

ages 24+ have at most a high school degree). A significantly higher share of residents are uninsured than the U.S. average, partly due to the high share of immigrants, Hispanic individuals, and minorities. While Newark is closer to other similarly medium-sized cities with respect to socioeconomics relative to the U.S. as a whole, its population is still relatively more disadvantaged and more diverse.[33] The housing stock in the city is relatively old, with over 71 percent built prior to 1980 and thus likely to contain lead plumbing fixtures in addition to being serviced by the lead pipelines. Finally, it is notable that the rate of renters in Newark is one of the highest among any major city (73.8 percent). Renters, compared with homeowners, are probably less likely to investigate the presence of lead service lines when making residential decisions or making major investments in their rental dwelling.

3.2 Identification Strategy

We employ a generalized difference-in-differences (DID) research design to identify causal effects of exposure to lead in drinking water—the "treatment"—on fetal health. We rely on the specific situation arising from Newark's water treatment and the ensuing natural experiment, which resulted in higher levels of lead exposure among some parts of the city but not others, to define the treatment and control groups as well as the pre- and post-treatment periods. In Section 4.3 we give detailed discussions regarding the plausibility of the common-trend assumption needed for the DID to allow causal inference. From the standpoint of minimizing the influence of confounding factors, it would be ideal to focus on mothers living right at the boundary between the two water plants' service areas, where mothers are plausibly separated randomly (unless there is residential sorting based on knowledge of the exact location of the boundary). However, in our empirical setting the population density very close to the boundary is relatively low, which restricts our statistical power to detect a treatment effect. We provide more discussion on this point in Section 4.5, in which we

[33] Comparing Newark to Flint, Michigan, median household income in Newark is about 48% higher and the poverty rate is about 10 percentage points lower relative to Flint; Newark also has a smaller black population and larger immigrant population compared to Flint.

conduct a near-border, rather than a sample-intensive at-border, analysis.

As noted earlier, water supplied to Newark is treated by two plants: the Pequannock plant and the Wanaque plant. The unique situation in Newark's water treatment is that the two plants rely on different chemical agents for corrosion control: Pequannock uses sodium silicate (City of Newark, 2018), while Wanaque uses orthophosphate (City of Newark, 2019). Both chemicals are approved by the EPA and effective in preventing dissolution of lead into the water by forming a protective layer (i.e., a diffusion barrier) on the interior surface of a lead pipe, although orthophosphate is more commonly used for corrosion control than sodium silicate (EPA, 2016b).

Pequannock's decision to use sodium silicate resulted from a corrosion optimization study conducted by the city of Newark in 1994. In that study both orthophosphate and sodium silicate proved to be effective corrosion inhibitors, but it was found that using orthophosphate could have negative environmental impact because of a specific situation of the water treated by that plant: water treated by Pequannock flows downstream into an uncovered, open-air reservoir, and this open body of water provides a conducive environment for orthophosphate to trigger algae growth (which is harmful) when that chemical gets into that reservoir (City of Newark, 2018).[34]

Pequannock started using sodium silicate for corrosion control in 1997, and the chemical had been effective since then. In 2016, however, the sodium silicate used by Pequannock was found to have become ineffective: the protective layer of lead service lines formed by the use of that chemical sloughed off. This was due to the pH in the water treated by Pequannock falling out of the range needed for sodium silicate to be an effective corrosion inhibitor (City of Newark, 2018). The lowered pH resulted from a deliberate decision made by Pequannock in 2015, to increase the acidity level (i.e., lowering the pH) of its treated water with the purpose of reducing disinfection byproduct formation (City of Newark, 2018).[35] While previously the

[34] When getting into an open body of water, orthophosphate can cause phosphorus concentrations. With phosphorus being a nutrient for algae, this can cause algal blooms.

[35] This was in response to a 2012 EPA rule that had strengthened monitoring of carcinogenic disinfectant byproducts.

pH of water treated by Pequannock was maintained around 8.0, it dropped to approximately 6.9 to 7.3 between 2015 and 2018 (see Figure 3 Panel A),[36] triggering a rapid release of lead from the pipes into the water (City of Newark, 2018).[37]

Our study uses the change in pH in the water treated by Pequannock (resulting from the plant's decision made in 2015), with the first indication of elevated lead levels in Newark being discovered in March 2016, as a natural experiment. In our study the treatment group comprises mothers living in the area serviced by Pequannock, and the control group includes mothers living in the area serviced by Wanaque. These two plants service the entire city separately: Pequannock services the western part of the city, and Wanaque services the eastern part of the city (City of Newark, 2018; City of Newark, 2019). The two service areas, delineated using the georeferencing method, are shown in the map depicted in Figure 1.[38]

We define birth years 2011–2015 as the pre-treatment period and birth years 2016–2019 as the post-treatment period. Figure 3 (Panel B) shows trends in lead contamination in water sampled across the Pequannock (treated) and Wanaque (control) service areas. Prior to 2016, tests from both service areas indicated compliance with the EPA's LCR (i.e., lower than 10 percent of samples testing above 15 ppb).[39] However, after 2016, there was a significant run-up in lead in tap water sampled from residences in the Pequannock service area. Through

[36] Note that the pH scale uses decimal logarithm, and therefore a decrease in pH by one unit indicates a 10-fold increase in the acidity of the water.

[37] Newark's corrosion control program had been tested in water with a very high pH (8.5 to 9.0), and optimal effectiveness is achieved with a pH of 8 to 9. In fact, the EPA (2016b) generally recommends a target pH of 8.8 to 10. Any anti-corrosion benefits of the silicates are lost when pH is adjusted below 7.5 (Thompson et al., 1997). Using historical data, Clay, Troesken and Haines (2014) show that a pH below 7.3 potentially could trigger a rapid increase in lead leaching into water, based on which they identify a causal effect of lead exposure on infant mortality in American cities during the period of 1900–1920.

[38] The authors produced this map in ArcGIS Pro 2.8.3. The shapefile delineating the boundary of Newark (New Jersey) was obtained from the City of Newark Open Data portal (https://data.ci.newark.nj.us/dataset/wards, accessed in September 2021). The authors used ArcGIS 2.8.3 to georeference the image showing the service areas of the two water treatment plants, and this image is shown in Appendix Figure A1.

[39] Note that prior to 2016, Newark was on a triennial monitoring cycle; hence, citywide lead tests of drinking water are not available for every year, and were not conducted in 2016. However, as noted earlier, the first indication of lead seeping into drinking water came from tests conducted in Newark public schools, which found 30 (out of 66) schools testing positive for elevated lead levels in 2016. This is consistent with the timing of the reduction in the water pH and ineffectiveness of sodium silicate as a corrosion inhibitor.

the end of 2019, tests indicated that this part of Newark had been in consecutive non-compliance since the start of the frequent biannual monitoring cycle. In contrast, trends in lead levels remained flat and in compliance in all periods in the Wanaque service area. As this figure shows, the risk of prenatal exposure to lead in drinking water significantly increased for babies born post-2016 and born to mothers living in the areas serviced by Pequannock, because of the unintended consequence of the plant's decision that eventually made the corrosion inhibitor (sodium silicate) it had been using ineffective. In contrast, the corrosion inhibitor (orthophosphate) used by Wanaque remained effective between 2016 and 2017; more detailed information is provided in City of Newark (2018).

Note that it takes time for lead to leach from the pipes into the water (following the failure of the corrosion inhibitor). As a result, increases in lead levels in the water will materialize with a time lag, which may explain the only modest uptick in lead levels in drinking water treated by Pequannock in 2015 (Figure 3 Panel B), when there was a rapid drop in pH of the treated water (Figure 3 Panel A). Despite this small uptick, the water treated by the impacted plant remained in full compliance with the EPA standards in 2015. We therefore include births that occurred in 2015 as part of the pre-treatment period. Any impact of prenatal exposure to lead for these births in 2015 would be nil to minor, and if there were any such adverse effects, we are erring on the side of providing a conservative estimate of the treatment effect.[40]

3.3 Econometric Specification

Our empirical approach proceeds in a stepwise manner to address specific issues that arise and to leverage the plausibly exogenous variation underlying the natural experiment. We

[40] Note that we only have the information on when elevated lead levels in Newark's drinking water were first discovered, based on the city's monitoring cycle, and also reported by media (i.e., in March 2016). We do not know exactly when elevated lead levels in Newark's drinking water actually occurred (since the city was on a triennial testing cycle prior to the crisis), or the date when the Pequannock plant increased the acidity level of its treated water. As a result, we are not able to use the year of conception, which is measured as the mother's (first day of the) last menstrual period (i.e., the earliest possible day of conception), to define the pre-treatment and post-treatment periods.

start with the following baseline DID specification, which can be interpreted as a reduced-form production function of infant health linking birth outcomes to prenatal exposure to lead in drinking water (Corman, Dave and Reichman, 2018; Dave and Yang, 2022):

$$y_{i,jt} = \alpha_0 + \alpha_1 treat_i T_t + \mathbf{x}'_{i,jt}\alpha_2 + \gamma_j + \lambda_t + \epsilon_{i,jt} \tag{1}$$

In equation (1), $y_{i,jt}$ denotes a specific birth outcome (e.g., LBW) of an infant born to mother i living at address j who gave birth in a year-month indexed by t.[41] The variable *treat* is binary, equal to one for the treatment group, and equal to zero for the control group. The variable T is also binary, equal to one for the post-treatment period, and equal to zero for the pre-treatment period. The key parameter of interest in our study is α_1, an intention-to-treat parameter which captures the effect of in utero exposure to elevated lead levels in drinking water on fetal health, operating through all reinforcing and mitigating mechanisms—that is, through biological, stress-induced, and compensatory self-protective pathways.

We include maternal residential address fixed effects (γ_j), in order to disentangle the effect of elevated levels of lead in drinking water (from differential acute exposure post-crisis) from the effect of just living in an old home that may contain lead pipes or lead paint, as well as controlling for any persistent environmental exposures at the residential address level.[42] Since the residential address fixed effects accommodate fixed effects that are based on broader geographic scopes (e.g., zip code fixed effects), we are also controlling for unobserved time-invariant neighborhood factors, such as local infrastructure, built environment, and access to health care. The residential address fixed effects allow us to disentangle the impact that was specifically driven by the exogenous variation in lead exposure (e.g., more lead seeping into

[41] We use a comma between the subscripts i and jt to emphasize that our data are not longitudinal in i: in the birth data we obtained from the NJDOH there is no unique identifier for each mother, which precludes the use of mother fixed effects.

[42] Note that in regression models using fixed effects to estimate effects of interventions, there is generally a reweighting of the estimates based on sizes of groups to which fixed effects are applied (Miller, Shenhav and Grosz, forthcoming). Our regression model is based on a DID design, in which we use residential address fixed effects as finer controls to replace the dummy variable indicating the treatment/control group, implicitly imparting a larger weight to larger families/households and/or resident mothers in larger dwelling units.

the tap water) from other longer-term and persistent environmental exposures associated with the dwelling and its neighborhood.

To control for any seasonality effects that exist in pregnancy or birth outcomes, as well as common shocks affecting mothers during the sample period, we include year-and-month of birth fixed effects (λ_t). Also included in this model is a vector of individual level control variables ($\mathbf{x}$): the sex of the baby; the mother's age, race and ethnicity, educational attainment, marital status, parity, the number of prenatal visits, as well as smoking status;[43] and whether or not the mother has had a previous preterm birth, which we use as a proxy for unobserved maternal health endowment at the time of pregnancy. We estimate all models by ordinary least squares (OLS), with standard errors clustered by year and month of birth.

We extend the baseline specification in several ways to address additional issues. We leverage precise information on the spatial boundary separating residents within Newark, serviced by the two water treatment plants, and implement a near-border analysis comparing mothers who live relatively close to the border. While our preferred specification relies on comparing impacted mothers with non-impacted mothers within Newark, drawing on the Wanaque service area to form the counterfactual, we show that our results are not sensitive to alternate controls that draw on mothers residing in cities and towns neighboring Newark which did not experience elevated lead levels in their drinking water during our sample period, both in the DID framework and in analyses that leverage the spatial border of Newark from adjacent towns. We further assess whether our effects on birth outcomes are driven by compositional shifts in the sample of mothers giving birth across the treated and control areas, or whether driven by changes in fertility or miscarriages.

A critical assumption needed for the DID research design to credibly identify the causal effect is that, in the absence of the water contamination, trends among mothers residing in the Wanaque service area are a valid counterfactual for trends among mothers living in the

[43] In the NJDOH birth data, maternal smoking status is measured by a binary response (yes/no) to the following question: "Did mother smoke cigarettes before or during pregnancy?" As a result, the maternal smoking status measured by this response can capture the status for two different periods—(1) before pregnancy and (2) during pregnancy—not necessarily for the latter exclusively.

Pequannock service area. In order to assess the validity of the counterfactual, we conduct a fully-specified conditional event study based on the following specification and disentangle the timing of the response:

$$y_{i,jt} = \beta_0 + \sum_{l=2011,\neq 2015}^{2019} \beta_{1,l} treat_i D_l + \mathbf{x}'_{i,jt}\beta_2 + \gamma_j + \lambda_t + \varepsilon_{i,jt} \tag{2}$$

In equation (2), D_l is a dummy variable, equal to one if birth year is l (where $l = 2011, \cdots, 2019$ except 2015) and equal to zero otherwise. Here, without loss of generality we use birth year 2015, which directly predated the water contamination, as the reference category.

The event study framework serves two purposes. First, it allows us to directly test for differential pre-crisis trends by evaluating the magnitude and statistical significance of the lead coefficient (i.e., $\beta_{1,l}$ where $2011 \leqslant l < 2015$). Second, the event study allows us to trace out the dynamics of the main DID effect from equation (1) across each period over the post-crisis window. This allows us to gauge effect dynamics as the crisis unfolded, from the first post-crisis year when residents remained largely uninformed of the true scope of the lead contamination, to later years when reports became more widespread. As information regarding the water contamination diffused across residents, this would also be expected to elicit self-protective behavioral and/or stress-related responses among pregnant women. While we are limited in the prenatal behaviors we can observe, we assess whether mothers responded to the crisis by increasing their contact with physicians (prenatal visits) that may have mitigated the effects of lead exposure on their pregnancy outcomes.

4 Results

4.1 Descriptive Statistics

Table 1 presents the summary statistics for sub-samples defined jointly by treatment status and treatment periods (columns 1–6) and for the full sample (column 7). Comparison of the means in Table 1 underscores two points. First, the rates of LBW and preterm births are significantly higher in the treatment group relative to the control group. These differences are driven by the treatment group including mothers who are relatively more disadvantaged (more likely to be unmarried and African American, for instance), although educational attainment appears to be somewhat similar between the treatment and control groups in the pre-treatment period. Second, a simple DID calculation (without covariate adjustment) shows an increase in the LBW rate by 1.4 percentage points,[44] for the treatment group and during the 2016–2017 period; in this period the LBW rate in the control group remains constant. However, when we expand the post-treatment period to include later years of 2018 and 2019, we see that the LBW rate in the control group also experiences some increase. This pattern could be explained by information spillovers, which we will discuss in more detail below.

Table 2 repeats the analyses done in Table 1 but focusing on mothers living within one mile of the boundary between the areas serviced by the two water treatment plants. Here, we confirmed the same pattern: a simple DID calculation (without covariate adjustment) shows an increase in the LBW rate by 1.4 percentage points,[45] for the treatment group and during the 2016–2017 period; with later years of 2018 and 2019 added to the post-treatment period, we noticed an increase in the LBW rate in the control group whereas the LBW rate remains relatively constant in the treatment group. Stability in the pattern of results in the descriptive statistics is noteworthy even though the sample size in Table 2 is on average 35% smaller than the sample size in Table 1. Another noticeable pattern in Table 2 is that pre-existing differences in mothers' race and ethnicity between the treatment and control groups are considerably narrowed than those in Table 1. This pattern suggests that residential sorting based on race and ethnicity, while being salient for the whole city, appears less significant but still existent near the boundary between the two plants' service areas.

[44] For LBW: (0.095 - 0.081) - (0.054 - 0.054) = 0.014.
[45] For LBW: (0.092 - 0.077) - (0.057 - 0.056) = 0.014.

4.2 Main Analysis

The remarkable similarity in the simple DID estimates we calculated from Tables 1 and 2 suggests the presence of an exogenous variation in lead exposure that is linked to the two water treatment plants' service areas. We rely on this exogenous variation to identify a causal effect of lead exposure. From the standpoint of controlling for confounding factors, it seems preferable to identify that causal effect at the boundary, where residential sorting could be viewed as continuous at the boundary and there is a discontinuous change in the lead exposure. However, Figure 1 reveals that such an identification is not really feasible in our empirical setting given that population density very close to the boundary (e.g., within 0.5 miles) is relatively low, especially for the control group. As a result, our main analysis uses the full sample to implement the DID, and we give detailed discussions on the plausibility of the common-trend assumption needed for the DID in the next section. Furthermore, we conduct a near- (rather than an at-) border analysis with results reported in a following section.

Table 3 reports the results of our main analysis based on the DID specification described by equation (1). For each birth outcome we use three different ending years for the post-treatment period. One important observation is that the adverse fetal health impact appears to be concentrated at the beginning part of the post-treatment period, especially for birth weight and LBW. We find an increase of 1.47 percentage points or 18 percent[46] in the likelihood of LBW among babies born in 2016–2017 and born to mothers living in Pequannock's service area, who were exposed to increased levels of lead in drinking water during pregnancy.[47] That the adverse health effect on continuous birth weight is weaker (approximately

[46] Here, $0.0147/0.081 \approx 18\%$, where 0.081 is the average LBW rate for the treatment group in the pre-treatment period (reported in Table 1).

[47] In Appendix Table A2 we report the full set of estimates. Our estimates are also robust to controlling for zip code-specific linear time trends (i.e., each zip code interacted with a linear time trend of birth year and month), which parametrically account for unobserved time-varying factors that may differ across parts of the city. Results are reported in Appendix Table A3. This robustness suggests that omitted variables bias that comes from zip code-level time-varying unobserved heterogeneities may not be a major concern once we control for residential address fixed effects and year-and-month of birth fixed effects.

a 31-gram decrease) indicates that the adverse impact is largely concentrated in the lower tail of the birth weight distribution. Similar to, although not as salient as, the pattern observed for birth weight and LBW, we find the effects on gestational length and preterm births appear to be stronger closer to the beginning part of the post-treatment period. Specifically, the likelihood of a preterm birth is predicted to increase by 1.91 percentage points or 19 percent,[48] among babies born in 2016–2017 and born to mothers in the treatment group.[49]

Possible reasons for the diminishing adverse effects over time that are revealed in Table 3 include information spillovers and maternal behavioral responses, which may emerge as the lead crisis unfolded (during the post-treatment period). We discuss and assess these possible channels below.

4.3 Checks on the Identification Assumptions

We conduct multiple checks on the identification assumptions needed for our causal inference, specifically with respect to the common trend, selection into birth and migration. We first check the plausibility of the common-trend assumption needed for the DID, and we report the results in Table 4. Here, we use the presence of a common trend between the treatment and the control groups in the pre-treatment period to support the assumption of a common trend in the post-treatment period in the absence of the treatment. Specifically, we run the regression model described by equation (1) only on the pre-treatment period, which is divided into two parts: one part is a true pre-treatment period; the other part is used as a false post-treatment period. In this analysis we confirm that there are no pseudo-treatment-induced differences in birth outcomes (e.g., LBW and preterm births) between the treatment and control groups in periods preceding the crisis. The flexible event study analyses discussed later also confirm that birth outcomes trended virtually identically in both parts of Newark

[48] Here, $0.0191/0.098 \approx 19\%$, where 0.098 is the average rate of preterm births for the treatment group in the pre-treatment period (reported in Table 1).

[49] We also use c-section as a crude proxy for any birth complications, and we use it as an additional dependent variable for the regression analyses done in Table 3. Here we find the coefficient on c-section to be statistically insignificant and the point estimates are close to zero. These results are available upon requests.

prior to the crisis.

Next, we assess potential selection into births. The water crisis in affected parts of Newark may have altered (or could be confounded with changes in) the composition of women giving birth.[50] If so, then the adverse effects we uncover may conflate a potential shift in the composition of mothers who tend to give birth to less healthy babies even in the absence of exposure to lead. To assess this possibility, we analyze the demographic characteristics of the mothers giving birth, and the results are reported in Figure 4.[51] Overall, for the entire sample period (2011–2019) there is some evidence suggesting a higher likelihood of babies born to mothers of higher socioeconomic status, which is indicated by an increased likelihood of the mother being white, in the treatment group and in the post-treatment period (Case A of Figure 4). The same pattern is confirmed among mothers living within one mile of the service area boundary for the period of 2011–2017 (Case B of Figure 4) during which the treatment effects are found to be most salient (Table 3). We do not find any other statistically significant or economically meaningful shift in maternal characteristics associated with the crisis. With the differential shift towards births observed among white mothers, if our treatment effect estimate is biased, then the bias would be more likely to result in an underestimate since higher socioeconomic status is usually associated with better

[50] Selection into births may also be driven by delays or failures of conception, or miscarriages. If less healthy fetuses are more likely to miscarry resulting in relatively healthier births, then adverse effects on birth outcomes from lead exposure would be understated. While we do not have information on delayed or failed conception, or miscarriages in the NJDOH birth data, we assess the importance of these selection pathways indirectly, using various specifications and for the period of 2011–2017 when adverse health effects are found in multiple birth outcomes (Table 3). Results are reported in Appendix Table A4. In column (1) we assess the impact on the likelihood of a female birth. The biological fragility of the male fetus to negative health shocks is often used to indirectly test for miscarriages, which would result in a greater likelihood of observing a female birth. We do not find any statistically significant effect of the Newark water crisis on this proxy measure. In column (2) we assess the impact on total births at the zip code level. We find that impact to be statistically insignificant and the point estimates to be small in magnitude. We interpret this finding as suggestive evidence that the adverse effects of Newark's lead-in-water crisis on fetal health are not likely to be driven by pregnancy behaviors or miscarriages. The live births included in the sample used for our estimation seem unlikely to be "survival of the fittest" who were exposed to lead in utero. One explanation for the lack of culling in utero could be that the exposure to lead did not reach a level high enough to trigger culling.

[51] In this figure point estimates and the associated 95% confidence intervals are reported (in the "rope ladder" plots), which are obtained from the DID estimation based on the equation (1) using each demographic characteristic (listed in Figure 4) as the dependent variable.

health. We also report on race-specific estimates below to bypass this racial compositional selection.[52]

The shift in composition of mothers giving birth may be also related to selective migration, for instance, if Newark's water crisis induces some mothers to move out of the city. With the vast majority of Newark residents (74%) being renters, for whom migration rates tend to be higher in general than for homeowners, it is possible that some residents move out of the city in response to higher perceived costs of staying (due to reduced water quality). On the other hand, the population in Newark is also largely of low socioeconomic status (see Appendix Table A1), for whom the costs of moving tend to be relatively higher.[53] To assess whether trends in migration patterns in the affected areas may have diverged after the onset of the water crisis, we turn to data from the ACS of 2011–2019 (covering our sample period). Migration in the ACS is observed across and within public use microdata areas (PUMAs).[54] Using this information, we define two outcomes: 1) an indicator for moving across PUMAs; and 2) an indicator for moving within a PUMA. We estimate DID models of these two outcomes for adult women (aged 18 or older), with the treated group comprising individuals who resided in Essex County (with Newark being its largest city and constituting its county seat) in the past year (and the control group including the rest of individuals living in New Jersey but outside Essex County).[55] For adult women overall, there is no

[52] In Appendix Figure A2 we repeat the analyses done in Figure 4 but focusing only on the pre-treatment period. Here, we find a pattern that is similar to the one revealed in Tables 1 and 2: the differences in mothers' demographic characteristics between the treatment and control groups in the pre-treatment period are smaller among mothers living near the boundary between the service areas of the two water treatment plants. But, only except for educational attainment, those differences are still statistically significant, suggesting the presence of residential sorting even near the service area boundary.

[53] In fact, the propensity to move has been found to be lower among individuals with less educational attainment, though largely similar across income groups, and also found to be lower among blacks, Hispanics, and foreign-born individuals (Molloy, Smith and Wozniak, 2011).

[54] PUMAs are geographic units with a population of at least 100,000 individuals and that are fully nested within states. There are 73 PUMAs defined for New Jersey, which are available in the ACS for current residence. However, for questions on migration, several of these are combined together. Hence, the lowest level of geographic information surrounding Newark that is available for residence prior to migration is for Essex County. Notably, Newark is the largest city in Essex County, making up over 35% of the county population.

[55] For within-PUMA migration, because we focus on moving out of Newark compared with not moving, our treatment group excludes women who currently reside in Newark if they reported moving in the past year. If a woman reported moving within Essex County, but currently lives in Newark, she may have moved

indication of any effects of the water crisis on migration, either out of the county or within the county; effects are close to zero and statistically insignificant. When we restrict the analyses to lower-educated women (high school or below), we find a marginally significant increase in the likelihood of moving out of the county, and moving within the county; the effect magnitude is small, about 0.6 percentage points (8% relative to the mean migration rate in Essex County for the sample). For higher-educated women, we find a decrease in within-county migration (0.9 percentage points), with no significant effect on moving out of Essex County. These effects are sensitive to area-specific trends, and become even weaker and less significant, suggesting that these patterns for Essex County could be more related to longer-term secular trends in migration rather than the water contamination in Newark per se. We find no other statistically significant or meaningful differences for other sub-populations.[56] We interpret this evidence as indicating that systematic migration responses may be nil, and if they are present then if anything the pattern of possible out-migration of lower-educated women would only serve to attenuate the estimated treatment effects.

4.4 Robustness Checks

In this section we conduct robustness checks on our main findings using the method proposed by Oster (2019). Specifically, we evaluate the robustness of our estimated treatment effect—an increase in the likelihood of LBW by 0.0147 (significant at the 10% level) in the treated area and in the post-treatment period over the sample period of 2011–2017 (reported in column 1 of Table 3). We examine how robust this estimate can be in the presence of

within the city, or she may have moved from out of Newark to Newark.

[56] Results are available from the authors upon request. We also separately explored effects on in-migration to Newark, based on an indicator of whether the ACS respondents reported moving into their residences in the past 12 months. Again, this is by no means an ideal measure since a respondent may have moved from one part of Newark to another. We find a decline in this proxy of in-migration (effect magnitude of about 2 percentage points, off a mean of 8.1%). While negative for both groups, the effect is slightly larger for lower-educated women. Interestingly, the effect for lower-educated women is similar for both the eastern and western parts of Newark, a detail in the PUMA we are able to observe when we explore in-migration, and again suggesting that these migration patterns may be part of Newark's longer-term development trend. As with our proxies for out-migration, the effects become weaker and largely insignificant with controls for PUMA-specific linear trends.

selection on unobservables, once we control for the residential address fixed effects. Results of this robustness check are reported in Figure 5. In both panels the x-axis represents what Oster (2019) refers to as the maximal R-squared. It is the R-squared obtained from a hypothetical regression model that includes all factors affecting the outcome variable (which is LBW in our case). These factors include those pre-determined (i.e., determined prior to the occurrence of the treatment) and those affected by the treatment. A key insight of Oster's method of linking selection bias due to unobservables with the stability of the treatment-effect estimates is the consideration of this maximal R-squared.

In theory, the maximal R-squared can equal one, if all contributing factors mentioned above are included in the regression model and the outcome variable is also measured without any error. Though, this is unlikely to happen in practice, and therefore choosing a specific maximal R-squared will be subjective and must be guided by empirical regularity. Here, we use a rule of thumb proposed by Oster (2019, p. 189), which is choosing a maximal R-squared that is 1.3 times the R-squared obtained from the regression model that controls for residential address fixed effects (which is 0.1403). Based on this, we use a maximal R-squared of 0.2 (i.e., $0.1403 \times 1.3 = 0.18239 \approx 0.2$). Using this maximal R-squared, Oster's method produces a bias-adjusted treatment-effect estimate of 0.0135 (shown in Panel A). In comparison, we hereafter refer to the treatment-effect estimate obtained from the regression model controlling for residential address fixed effect, which is 0.0147, as the original estimate. According to Oster (2019), assuming selection on unobservables is as important as selection on observables and if the interval formed by the original estimate and the bias-adjusted estimate excludes zero, then the original estimate will be deemed as robust. In our case the interval is [0.0135, 0.0147], which excludes zero. Therefore, we deem our original estimate as robust. This robustness can also be justified if the bias-adjusted estimate falls into the confidence interval of the original estimate. Panel A shows the bias-adjusted estimate (0.0135) falls into that interval, indicating that our original estimate is robust to selection on unobservables.[57] Panel A also shows that this robustness can be justified for a wide range of maximal R-squared, going from 0.2 to 0.46.[58]

In Panel B we conduct another robustness check also proposed by Oster (2019). Specifically, this method calculates the degree of selection on unobservables relative to observables, which Oster (2019) refers to as the coefficient of proportionality.[59] This calculation assumes a value for the maximal R-squared and also assumes the true treatment effect is zero. The calculated coefficient shows how large selection on unobservables has to be in order to produce a treatment effect of zero (i.e., completely eliminating the observed effect of prenatal lead exposure on LBW). For example, in the case of a maximal R-squared set to be 0.2, the calculated coefficient of proportionality turns out to be 5.6167, meaning that the unobservables would need to be five to six times as important as the observables in order to produce a treatment effect of zero. Oster (2019) recommends that the equal selection (i.e., the coefficient of proportionality being equal to one) should be used as the upper bound on the coefficient of proportionality. Based on this upper bound, the observed effect will be viewed as robust if the coefficient of proportionality is greater than one. In Panel B and in the range of maximal R-squared previously discussed (regarding the results shown in Panel A), we see that all calculated coefficients are greater than one. This result again suggests that the original estimate could be viewed as robust to selection on unobservables.

[57] Here, we choose the 90% confidence interval because the original estimate (0.0147) is significant at the 10% level.

[58] Note that the R-squared in the regression model that does not control for residential address fixed effects (i.e., replacing those fixed effects with a treatment dummy variable) is 0.0392. This R-squared increases to 0.1403 by about 258%, once the model controls for those fixed effects. Results in Panel A also indicates that the original estimate can be viewed as robust to the inclusion of unobservables into the regression model that raises the R-squared from 0.1403 to 0.46 by about 228%.

[59] In Oster (2019), this coefficient of proportionality is referred to as "delta," which is the ratio of selection on unobservables over selection on observables. In that paper, selection on observables (or unobservables) is defined by the covariance between the treatment dummy variable and observables (or unobservables) divided by the variance of observables (or unobservables).

4.5 Near-Border Analysis

While the findings from the robustness checks discussed above are reassuring, it may still be preferable to focus on mothers living exactly on the service area boundary where selection bias arising from residential sorting could be minimal. We begin this analysis with Figure 6, focusing on LBW and the period during which the effect on LBW is found to be most salient (results reported in Table 3). The pattern we observe in Panel A is consistent with the one observed in Table 1. There is a marked increase in the prevalence of LBW pre- and post-crisis in the treatment group relative to the control group. Moreover, the spatial discontinuity is far more pronounced and has noticeably widened at the boundary (between the two service areas) in the post-period (Panel A), though caution needs to be warranted in this inference since there is an uncertainty in estimating the LBW rate exactly at the boundary.

To accommodate such an uncertainty, in Panel B we present the binned sample means and the associated 95% confidence intervals by treatment status and period, for areas that are within one mile of the boundary. As in Panel A, results in Panel B also indicate the presence of a widened gap near the discontinuity in the post-crisis period. However, when confidence intervals are taken into account, there appears to be no significant difference in the LBW rate between the treatment and control groups in areas very close to the boundary. One explanation for this finding is that in our empirical setting the population density very close to the boundary is low (Figure 1), which limits the statistical power to detect a treatment effect. The effect magnitude at the spatial discontinuity (Panel A), or that based on the difference-in-discontinuity between the pre-crisis and the post-crisis period (Panel B), is on the order of about one to 1.5 percentage points, similar to the treatment effects reported in Table 3 (and in Table 5).

Overall, Figure 6 suggests that our empirical setting lacks sufficient density right at the boundary to be suitable for a regression discontinuity (or difference-in-discontinuity) design. In addition, estimating the LBW rate at the spatial boundary without bias requires the identification of an optimal bandwidth for the running variable (i.e., the signed distance to the boundary, positive for the treatment group and negative for the control group), and this optimal bandwidth is typically used as the upper bound for other bandwidths chosen for robustness checks. For our empirical setting the optimal bandwidth for the running variable is calculated to be about 0.5 miles.[60] As Figure 1 shows, sample size matched to this optimal bandwidth is small, particularly for analyzing LBW (which is a small-probability event).

Because of this limitation, we conduct a near- (rather than an at-) border analysis using alternate bandwidths of one and two miles with a uniform kernel, with estimates reported in Table 5. Using specifications based on equation (1) but allowing for non-linearities in the effects of the running variable and its interaction with the treatment status, we find the treatment effect estimates to be robust across those specifications, and especially salient for LBW either within one mile or within two miles of the boundary. In particular, consistent with the pattern observed in Tables 1 and 2, the treatment effect estimate for LBW among mothers living within one mile of the boundary (columns 1 through 4 of Panel A of Table 5; 1-mile bandwidth with a uniform kernel) is very similar to the treatment effect estimate (0.0147) obtained from the DID estimation for the full sample (column 1 of Table 3). The robustness in the treatment effect magnitude across the various estimation strategies validates the exogeneity of the variation in lead exposure linked to the water treatment plant's service area, and the common-trend assumption for both the full sample of mothers across the treated and control areas and for the sample near the boundary.

4.6 Event Study Analysis

Results reported in Table 3, for alternate post-treatment windows, indicated that the adverse effects were most pronounced during the early periods of the crisis before attenuating. We further assess these dynamics in the response as Newark's water crisis unfolded with a

[60] Specifically, we obtained the optimal bandwidth using the "rdrobust" package (Stata code downloaded from https://rdpackages.github.io/, accessed in September 2021) developed by Calonico et al. (2017) and Calonico, Cattaneo and Titiunik (2014). Results are available from the authors upon request.

flexible event study analysis (2). Results (coefficient estimates of the time-varying treatment effects and the associated 95% confidence intervals) are reported in Figure 7. Here, we find no differential pre-treatment levels or trends between the treated and control groups conditional on the observables included in the regression model, suggesting that the pre-existing level differences in birth outcomes between the groups (Table 1) are fully explained by the socioeconomic differences and also adding to the plausibility of the common trend.

Over the post-treatment period, the year-by-year comparison between the treated and control groups, while lacking some statistical power, appears to confirm the same pattern revealed in Table 3: the treatment effect is muted over time, with a marked increase in LBW in the treatment group relative to the control group that occurred about one year after Pequannock's decision to reduce the pH level of its treated water, which made the corrosion inhibitor in the water become ineffective. That decision was made in 2015 with no media reports or coverage prior to 2016 about elevated lead levels in drinking water,[61] and the marked increase in LBW corresponds to babies born in 2016. The downward trend in the treatment effect in the post-treatment period may reflect effects of information spillovers as well as avoidance and compensatory behaviors, which we discuss in the next section.

4.7 Information Spillovers and Behavioral Responses

Trends in Google search queries shown in Figure 2 indicate that there was substantial public interest after reports of elevated lead levels found in drinking water in Newark public schools surfaced in March 2016; relative to this initial spike, interest quickly subsided but continued till 2019, spiking again towards late 2018 with the publicity surrounding the city's release of its engineering report and the decision to distribute filters to affected residents, and then spiking again in the fall of 2019 due to reports on elevated lead levels found in houses even with filters installed. As a result, it is possible that as the water crisis continues, mothers

[61] In fact, during our sample period (2011–2019), 2016 is the first year in which media reported the existence of elevated lead levels (exceeding the EPA's threshold of 15 ppb for taking regulatory actions) found in Newark's drinking water.

living in the control area can become "treated" due to stress induced by the ongoing water crisis; consequently, higher LBW rates may be observed in the control group given the well-documented association between maternal stress during pregnancy and adverse birth outcomes (e.g., studies summarized in Bussières et al., 2015). This partial treatment among the control group may in part explain the muted treatment effects over time in the post-treatment period shown in Figure 7.

We conduct further investigations to assess the possibility of information spillovers within the city of Newark by using regions that are outside Newark, and the results are reported in Table 6. In Panel A we use mothers living in cities and towns that are east of the area serviced by the Wanaque plant to form the control group, for which and during our sample period there were no known reports on lead in drinking water. Focusing on LBW, we repeat the analyses done in Table 3, but contrary to the results in Table 3, here we find the adverse effects on LBW to be persistent over time in the entire post-treatment period. This result suggests that information spillovers may be present and only within the city of Newark, and are driving some of the attenuation in the treatment effects over time.

In Panels B through D we use mothers living in the area west of the area of Newark serviced by the Pequannock plant (shown in Figure 8) to form the control group. In Panel B the control group includes mothers living within one mile of the city's western border. In this case we do not find any statistically significant treatment effect, which may be due to two reasons. First, information-spillover-induced stress response may be present in areas that are immediately outside the western part of the city (where the water crisis occurred). Second, we have precise information on the boundary between the service areas of the two water treatment plants within Newark; however, the city's western border may not follow exactly the boundary of the treated Pequannock plant's service area. As a result, mothers living very close to (within one mile of) the city's western border outside of Newark may be partially treated by being exposed to the contaminated water supply, which can attenuate the treatment effect estimate.

As a result, we carve out and exclude mothers living in the control area (west of the Newark city border) but living within one mile of this border. With this exclusion, both the treatment group and the control group include mothers living within three miles (in Panel C) or five miles (in Panel D) of the border. Overall, results in Panels C and D show that, similar to what we find in Panel A, the adverse effect on LBW appears to be persistent over time. This finding is in contrast to what we find in Table 3, with the difference explained by the use of two different control groups. These two control groups cover areas inside or outside the city of Newark, respectively. Taken together, results in Table 6 and Table 3 suggest that within Newark propagation of the information regarding the water crisis that is linked to the Pequannock plant could have affected residents living in the same the city but serviced by the Wanaque plant.

Another related issue is the possible presence of avoidance or compensatory behaviors triggered by the water crisis,[62] which we examine indirectly (due to data limitation) by assessing whether there is greater contact with the medical care community among pregnant women during the water crisis. Results are reported in Table 7. Both columns use the same pre-treatment period (2011–2015), focusing on the maternal behavioral responses of the treatment group relative to that of the control group. In Panel A, column (1) reports the estimated treatment effect on the number of prenatal visits for the entire post-treatment period (2016–2019), while column (2) reports the estimated treatment effect that varies by each year of the post-treatment period. Overall, we find an increase in prenatal care utilization in the treatment group during the post-treatment period; in particular, the increase appears to be most substantial in 2018, which could in part explain why we previously find a muted treatment effect on LBW (or birth weight) once we expand the post-treatment period from 2016–2017 into 2018. Although the overall effect of an increase in the number of prenatal visits (by 0.2131, or by about 2.4% relative to the average of 8.985 reported in Table

[62] For more information on ways of reducing exposure to lead in drinking water, see the EPA's website https://www.epa.gov/ground-water-and-drinking-water/basic-information-about-lead-drinking-water#reducehome (accessed in December 2019).

1) is small, we interpret this finding as evidence of the presence, not the likely magnitude, of behavioral responses aimed at mitigating health risks caused by the water crisis.[63] The literature on the effects of prenatal care on birth outcomes in general has produced mixed and nuanced findings (Corman, Dave and Reichman, 2019). In the context of the water crisis in Newark, however, physicians may provide valuable information to pregnant women on how to avoid ingestion and absorption, for instance by using bottled or filtered water, letting the faucet run prior to drinking to flush out the lead, and increasing the intake of calcium, iron and vitamin C, which can inhibit lead absorption and help to get rid of lead from the body.[64]

In Panel B of Table 7, we repeat the estimations done in Panel A but now for maternal smoking. One limitation of the smoking measure in the NJDOH data is that it conflates both smoking prior to as well as during pregnancy, and we therefore interpret these results with caution. We find a statistically significant reduction (about 1.4 percentage points) in maternal smoking among mothers living in the treatment area over the entire post-treatment period (2011–2019); consistent with the pattern observed in Panel A, this reduction appears to happen mostly in 2018, an eventful year with the unfolding of the water crisis (explained in

[63] We also conduct the estimations by mother's race and ethnicity (white, black, and Hispanic), and we find that the significant effect on the number of prenatal visits reported in Panel A column (1) of Table 7 appears to be driven by mothers who are black, not by mothers who are white or Hispanic. These results are available upon request.

[64] In our study, we find that an increase in the number of prenatal visits is associated with a decrease in the likelihood of LBW, suggesting a beneficial effect of prenatal care utilization on fetal health. This result is reported in Appendix Table A2. In that table we also observe a slightly smaller magnitude of the estimated effect of maternal lead exposure on LBW based on the regression model that excludes variables on prenatal care and smoking (column 1). This pattern is consistent with the presence of protection behaviors indicated by greater use of prenatal care and less smoking (results reported in Table 7), as well as the negative association between prenatal care and LBW (column 2 of Appendix Table A2) and the positive association between maternal smoking and LBW (column 2 of Appendix Table A2).

Section 2.2).[65] Since most smokers have initiated smoking prior to age 18,[66] the decrease in maternal smoking likely reflects an increase in cessation, either among women of childbearing age prior to becoming pregnant or among pregnant women. The decline in smoking may also be due to greater contact with physicians (increased prenatal care; Wehby, Dave and Kaestner, 2020) or reflect a form of compensatory behavior to counteract the adverse health impact of greater lead exposure.

4.8 Heterogeneous Treatment Effects

While it is important to investigate the heterogeneity in the impact of the prenatal lead exposure on fetal health, identifying possible disparities, our ability to do so is limited because of the sample size used for each dimension of the heterogeneity analysis. Nevertheless, one dimension that imposes less restriction is fetal sex, since sample sizes for the two sexes are largely balanced (not lopsided). Here, we estimate the treatment effect by fetal sex, given that male and female fetuses may respond differently to a compromised intrauterine environment. Results are reported in Table 8. One robust and striking pattern is that the adverse impact of prenatal lead exposure on fetal health appears to be concentrated among male fetuses, which is consistent with the fragile male hypothesis (Eriksson et al., 2010; Kraemer, 2000). Coefficient magnitudes for female fetuses appear to suggest a much smaller response, and these treatment effects do not achieve statistical significance at conventional levels. This interpretation should also be qualified by noting that a smaller average effect could mask

[65] Danagoulian and Jenkins (2021) find an increase in smoking among mothers exposed to the Flint water crisis. The increase in smoking (reduction in cessation rates among pregnant women) is consistent with a stress response induced by the water crisis. The Newark crisis differs from the Flint crisis, notably in regards to information dissemination and public knowledge. Despite the finding of elevated lead levels in drinking water in Newark public schools in March 2016, and further violations of the EPA standards in 2017, the 2017 Water Quality Report downplayed the violations. The public was subject to conflicting information, and remained largely unaware of the scope of the water contamination. Public perceptions of the water contamination in Flint were more immediate and pronounced, with Flint switching its water source in April 2014, city residents soon thereafter complaining about the color, taste and smell of their water, the city issuing a boil advisory in August 2014, and General Motors announcing that it was discontinuing the use of Flint tap water due to high levels of chlorine and corrosion.

[66] Data from the 2018 National Survey of Drug Use and Health indicate that over 80 percent of smokers have initiated smoking by age 18, and over 68 percent have initiated smoking by age 17.

significant variation in the individual specific treatment effect across the population of female births.[67]

5 Conclusion

We provide the first study of the effects of the lead crisis in Newark, informing how prenatal exposure to lead through tap water impacts birth outcomes. Quantifying these effects is important for several reasons. First, the crisis in Newark is not singular, but rather emblematic of the nation's aging water infrastructure as many other cities in the U.S. face lead contamination in their water supply. High lead levels have been found in the tap water in Baltimore, Chicago, Detroit, Milwaukee, New York, Pittsburgh, and Washington D.C., and between 2018 and 2020 nearly 30 million people in the nation received their drinking water from community water systems that were in violation of the EPA's Lead and Copper Rule (Fedinick, 2021; Mulvihill, 2021). The American Society of Civil Engineers in its 2017 report card rated the nation's drinking water system a D grade, underscoring the aging pipes and emerging problems with contaminants such as lead.[68] The urgency of replacing all lead pipes in the U.S. water systems was finally recognized in the Infrastructure Investment and Jobs Act enacted by the U.S. Congress in November 2021 that includes funding of $15 billion for lead pipe replacement.[69]

[67] In Appendix Table A5 we investigate the heterogeneity in the treatment effect along other dimensions. Because of sample size limitations, we are not able to reject the null of similar adverse effects across the sub-groups. However, patterns across coefficient magnitudes largely point to the adverse impact of the water crisis being concentrated among pregnant women of lower socioeconomic status (characterized by educational attainment and marital status). Women of lower socioeconomic status have been found to have higher rates of calcium deficiency (Wallace, Reider and Fulgoni, 2013), which leads to greater absorption of ingested lead for a given level of exposure. Women of lower socioeconomic status may also be less likely to engage in avoidance and mitigation behaviors, which may otherwise have counteracted the effects of exposure for more educated and married mothers.

[68] Source: https://www.infrastructurereportcard.org/cat-item/drinking_water/ (accessed on October 7, 2020).

[69] See https://www.wsj.com/articles/how-the-1-trillion-infrastructure-bill-aims-to-affect-americans-lives-11636173786 for details (accessed on November 10, 2021). This final legislated amount is below the originally proposed funding of $45 billion to eliminate lead service lines nationwide, and, by some amounts, falls well below the estimated cost of nationwide replacement ($28–$47 billion). For more details, see https://www.brookings.edu/blog/up-front/2021/05/13/what-would-it-cost-to-replace-all-the-nations-lead-water-pipes/, accessed

Second, much of our understanding of the health effects of lead comes from potentially endogenous associations between blood lead levels and health outcomes. The water crisis in Newark provides a plausibly exogenous source of variation in lead exposure, allowing us to identify a causal effect of prenatal exposure on fetal health—an effect that is salient because it captures the overall impact in the population, operating through all channels (biological and behavioral, including any avoidance behaviors) when water systems fail. The effect we estimated is also of immediate policy interest since our estimate reflects the presence of behavioral responses that are insufficient for eliminating the health risk due to lead exposure. Moreover, population blood lead levels have declined considerably over the past five decades. Our study therefore captures the effects of an increase in exposure relative to a current low baseline.

Third, many public health advocates and experts on water systems have called for full replacement of the nation's estimated 6 to 10 million lead service lines, with some communities already having implemented successful replacement programs and other cities developing plans to do so.[70] A comprehensive evaluation of the cost implications of such public investments requires estimates of the public health impact of lead exposure.

We find robust and consistent evidence that the increased in utero exposure to lead through water contamination in Newark significantly increased the prevalence of infants being born with LBW or preterm. There is little evidence to suggest that these effects are driven by selection into births. Our estimates indicate an approximately 1.5 percentage-point (or 18 percent) increase in the likelihood of LBW, and an approximately 1.9 percentage-point (or 19 percent) increase in the likelihood of a preterm birth. These are intention-to-treat effects of residing during pregnancy in areas with increased lead levels in tap water, since not every resident here is being exposed to high levels of lead. As part of the city's lead service

on February 14, 2022).

[70] For instance, Framingham (MA), Lansing (MI), Madison (WI), Medford (OR), Sioux Falls (SD), Springfield (MA), and Spokane (WA) have fully removed lead service lines in their communities. Subsequent to the water crisis, Flint initiated a full replacement program of its lead water service lines in 2016, which is currently underway. Recently, Chicago rolled out a plan for lead service line replacement, fully subsidizing costs for eligible low-income households and waiving permit fees for other homeowner-initiated replacement.

line inventory, approximately 61 percent of the city service lines were constructed of lead.[71] Inflating the treatment effects by this "exposure" probability implies effect sizes between 2.5 and 3.1 percentage points, with respect to higher levels of lead exposure.[72] Our estimates imply an increase of about 27 to 49 LBW or preterm births in a given year attributed to the lead-in-water crisis in Newark.[73]

In March 2019, Newark commenced a program to remove and replace all of the city's lead service lines in the water system at no cost to the homeowner, at a projected public cost of \$90–\$180 million.[74] With the lifetime societal economic burden of a preterm birth estimated to be approximately \$67,532 (Institute of Medicine, 2007),[75] the annual societal cost of the lead crisis in Newark amounts to \$1.82–\$3.31 million from the estimated increase in worse infant health linked to the heightened lead exposure each year.[76] Assuming a discount rate for public policy of 2% based on the social rate of time preference (Council of Economic Advisers, 2017), societal cost savings from averting this adverse fetal health could be between \$91 and \$166 million.[77] These calculations suggest that the benefits are

[71] The city's lead service line inventory, undertaken in response to its EPA's LCR violations, shows 18,406 (out of 29,938) of its service pipes were lead (source: https://www.nj.com/essex/2019/08/newarks-handing-out-bottled-water-what-you-need-to-know-about-the-citys-lead-crisis.html, accessed on September 9, 2019).

[72] To place these magnitudes in context, this increase in low birth weight, while large and clinically significant, is about half the magnitude of the effect due to maternal cigarette smoking during pregnancy (Evans and Ringel, 1999; Lien and Evans, 2005).

[73] There were 5,210 (10,645) births in the treated part of Newark over the post-treatment period of 2016–2017 (2016–2019). Combining these bases with the treatment effects for LBW and preterm births from Tables 3, 5 and 6, and normalizing to an annual basis, yields a range of about 27 to 49 LBW or preterm births.

[74] Source: https://www.newarkleadserviceline.com/replacement (accessed on October 14, 2020).

[75] The Institute of Medicine (2007) estimated the societal burden of a preterm birth to be \$51,589 in 2005 dollar. We inflate this estimate to 2019 dollars.

[76] Here is the calculation: 27 (or 49) preterm births $\times$\$67,532 per preterm births$\approx$\$1.82 million (or \$3.31 million).

[77] There is some debate as to the appropriate discount rate to apply for public policy (see for instance, Council of Economic Advisers, 2017; Li and Pizer, 2021) depending on the social rate of time preference or the social opportunity cost of capital, and the length of the time horizon under consideration. The U.S. federal guidance requires agencies to use both a 3% and a 7% real discount rate in regulatory cost-benefit analyses. Under this guidance, the annual societal cost savings of averting the adverse fetal health would be between \$61 million and \$110 million (social discount rate of 0.03) and between \$26 million and \$47 million (social discount rate of 0.07). Clearly, the cost implications are sensitive to the discount rate employed. With long-term real interest rates decreasing substantially over the past decade, a recent issue brief by the Council of Economic Advisers (2017) recommends lowering the estimate of the social discount rate in applications to public policy cost-benefit calculus.

roughly the same order of magnitude as the intervention costs, offsetting much, if not all, of the public sector cost of lead abatement through service line replacement.[78]

That the public values such investments to improve the water system infrastructure in the nation is apparent from the considerable engagement in avoidance behaviors when contaminant violations in water systems are disclosed to the public (Graff Zivin, Neidell and Schlenker, 2011). According to McCarthy (2017), drinking water pollution worries are also at their highest levels since 2001, with 63% of the public reporting that they are worried a great deal about this issue; concern is even higher among low-income individuals (75%) and non-whites (80%). The cost-saving estimates we previously discussed are likely to be lower-bound estimates given that the focus of this study is specifically on adverse fetal health effects; lead exposure among children has also been found to independently and adversely impact their development, increasing anti-social and criminal behaviors and reducing achievement in school.[79]

We qualify this discussion that since our estimates are derived from a city composed of a population of lower socioeconomic status (SES) compared to the U.S. as a whole (or even an average similarly-sized city), the effects may not necessarily generalize due to variation in media exposure, information processing, and mitigation behaviors. However, Newark is emblematic of another pattern that speaks to the significance and external validity of our estimates; low-income and high-minority communities have, in particular, been found to face disproportionately higher pollutant exposures, and especially greater exposure to drinking water contamination, for various reasons (Schaider et al., 2019). Thus, for communities that are experiencing issues with lead-related water contamination, which tend to be low-SES and minority-prevalent communities, the estimates from this study are highly salient and

[78] We ignore here the possibility that replacement of lead service lines, due to partial replacement or disturbing the pipes, may potentially increase exposure risk temporarily. Though, Gazze and Heissel (2021) find no such increase in risk for lead service line disturbances in Chicago. Moreover, since Newark commenced its lead service line replacement program in March 2019, and secured full financing in August 2019, any short-term exposure risk from the start of the replacement program is likely to be small over our sample period (2011–2019) and would not impact our treatment effect estimates.

[79] See, for instance, Aizer and Currie (2019), Aizer et al. (2018), and Billings and Schnepel (2018).

would have greater external validity.

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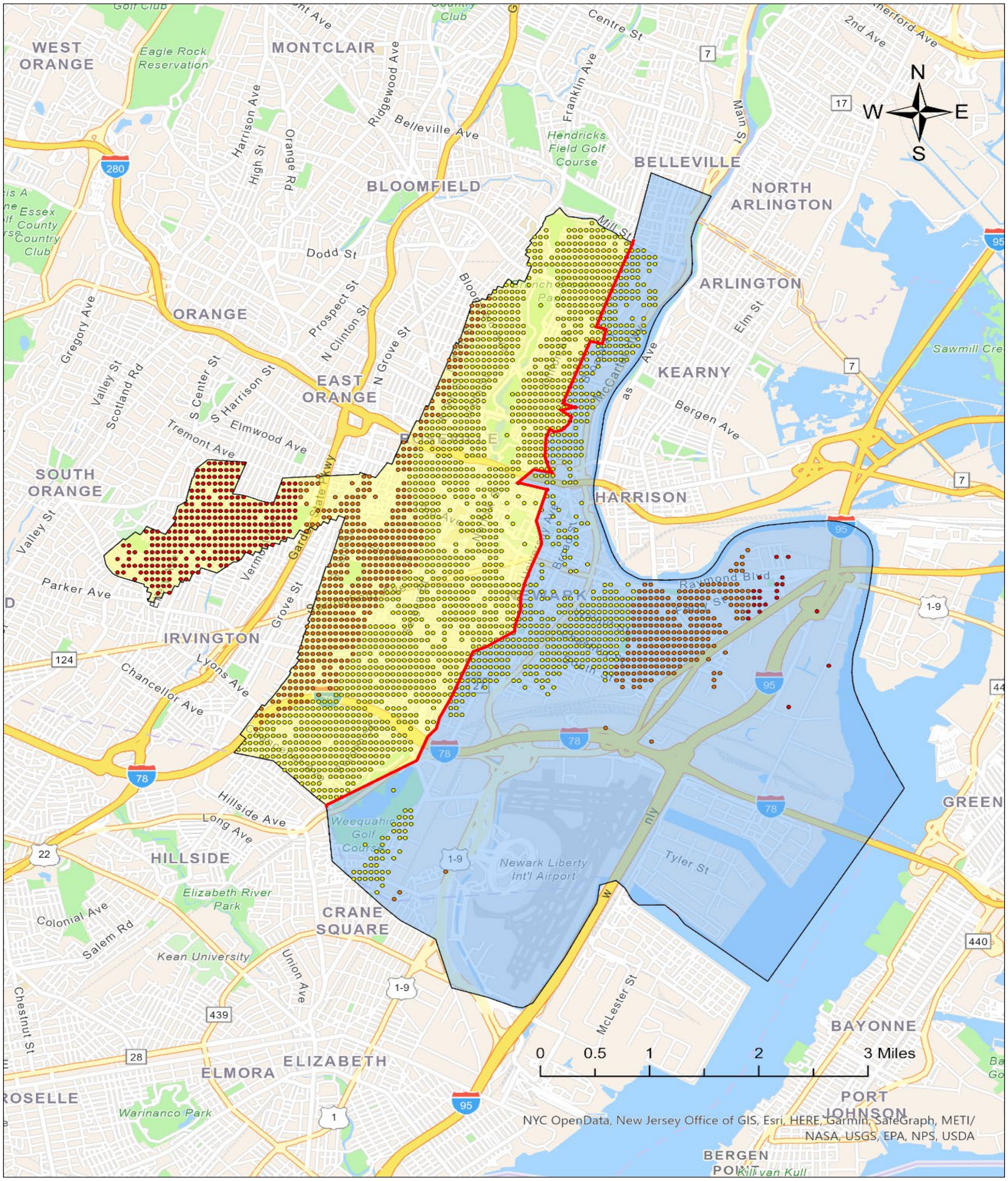
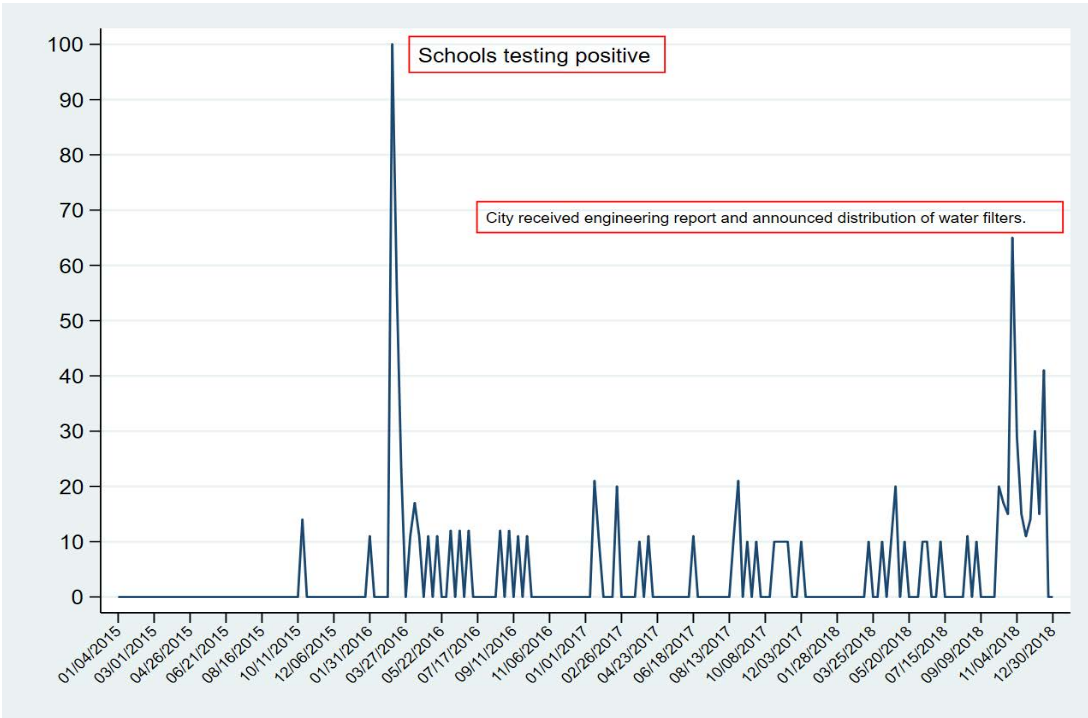


Figure 1: City of Newark and the Areas Serviced by the Two Water Treatment Plants

Notes: The authors produced this map in ArcGIS Pro 2.8.3. The shapefile delineating the boundary of Newark (New Jersey) was obtained from the City of Newark Open Data portal (https://data.ci.newark.nj.us/dataset/wards, accessed in September 2021). The authors used ArcGIS 2.8.3 to georeference the image showing the service areas of the two water treatment plants (obtained from https://www.nj.com/essex/2018/11/newarks_now_under_a_national_spotlight_for_lead_in.html, accessed in November 2019). The georeferenced image is shown on the map: the area shaded in blue indicates the area of Newark serviced by the Wanaque plant; the area shaded in yellow indicates the area of Newark serviced by the Pequannock plant; the red line shows the boundary between the two service areas. The yellow dots represent home addresses that are within 1 mile of the boundary; the orange dots represent home addresses that are between 1 and 2 miles of the boundary; and the red dots represent home addresses that are between 2 and 3.3 miles (which is the maximum distance in our data) of the boundary.

Panel A



Panel B

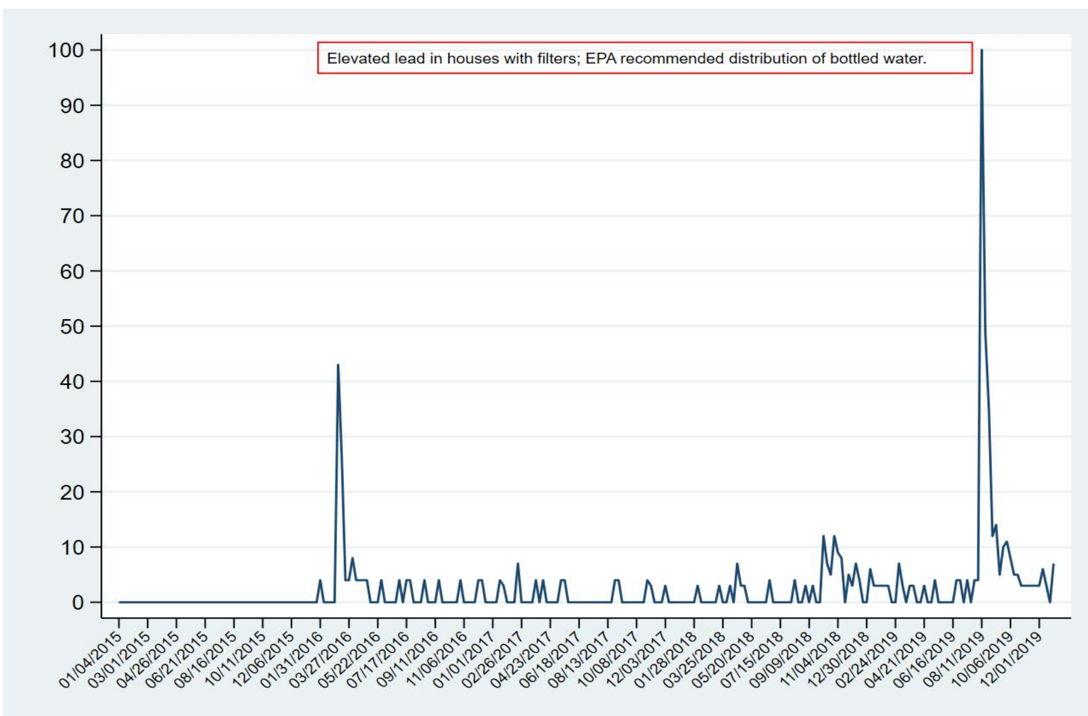
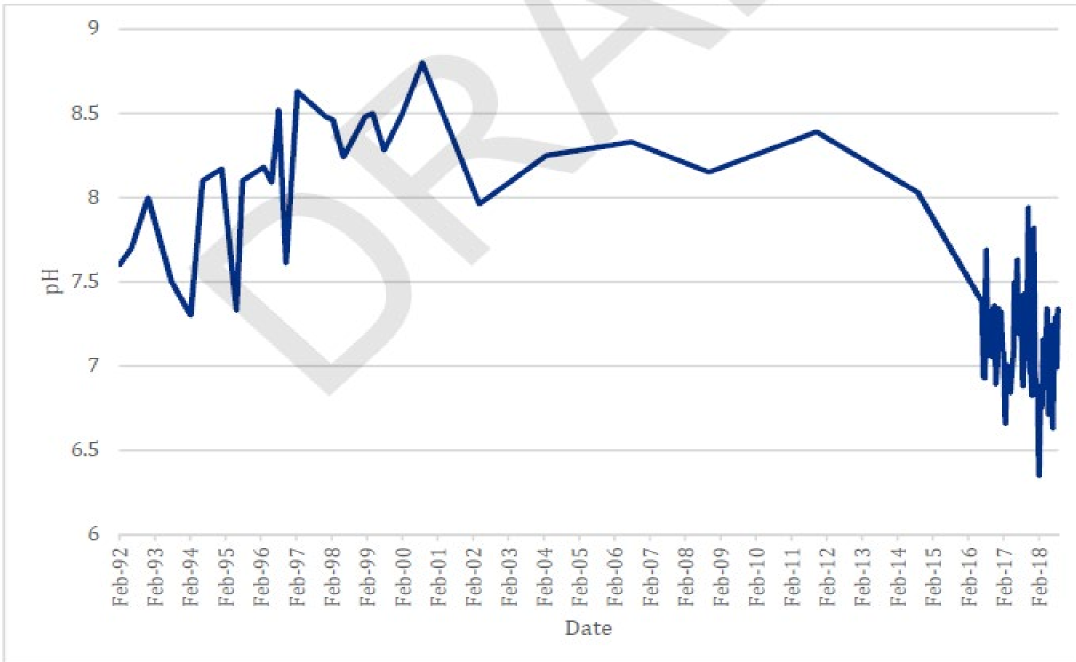


Figure 2: Google Trends Based on Query Terms "Lead + Water + Newark"

Notes: Panels A and B are for 2015–2018 and 2015–2019, respectively. Google trends do not provide the absolute number of queries. In each period (2015–2018 or 2015–2019), the day with the largest number of queries is indexed to be 100 (the maximum), and queries on all other days are measured relative to this maximum.

Panel A



Panel B

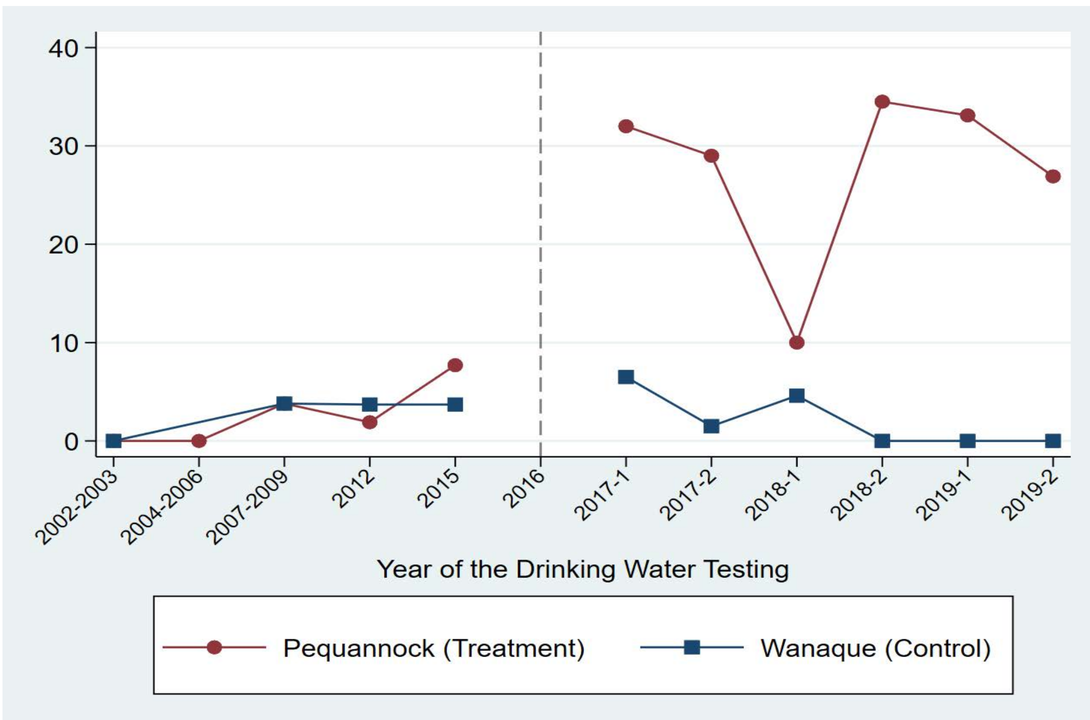


Figure 3: pH Levels of Water Delivered by the Pequannock Plant (Panel A) and Percent of Drinking Water Testing Samples with Results Showing Lead Levels in Drinking Water > 15 ppb (Panel B)

Notes: The figure in Panel A is reproduced from the report by the CDM Smith titled "Pequannock WTP Corrosion Control Review and Recommendations—Draft. City of Newark Lead and Copper Rule Compliance Study" (City of Newark, 2018). The original figure in the report is denoted "Figure ES-3 – Historic Pequannock WTP Delivered Water pH". The authors have received permission from the CDM Smith to include this figure in this study. In Panel B calculations were based on data from City of Newark (2018) and New Jersey Drinking Water Watch from the New Jersey Department of Environmental Protection (https://www9.state.nj.us/DEP_WaterWatch_public/index.jsp, accessed in February 2020).

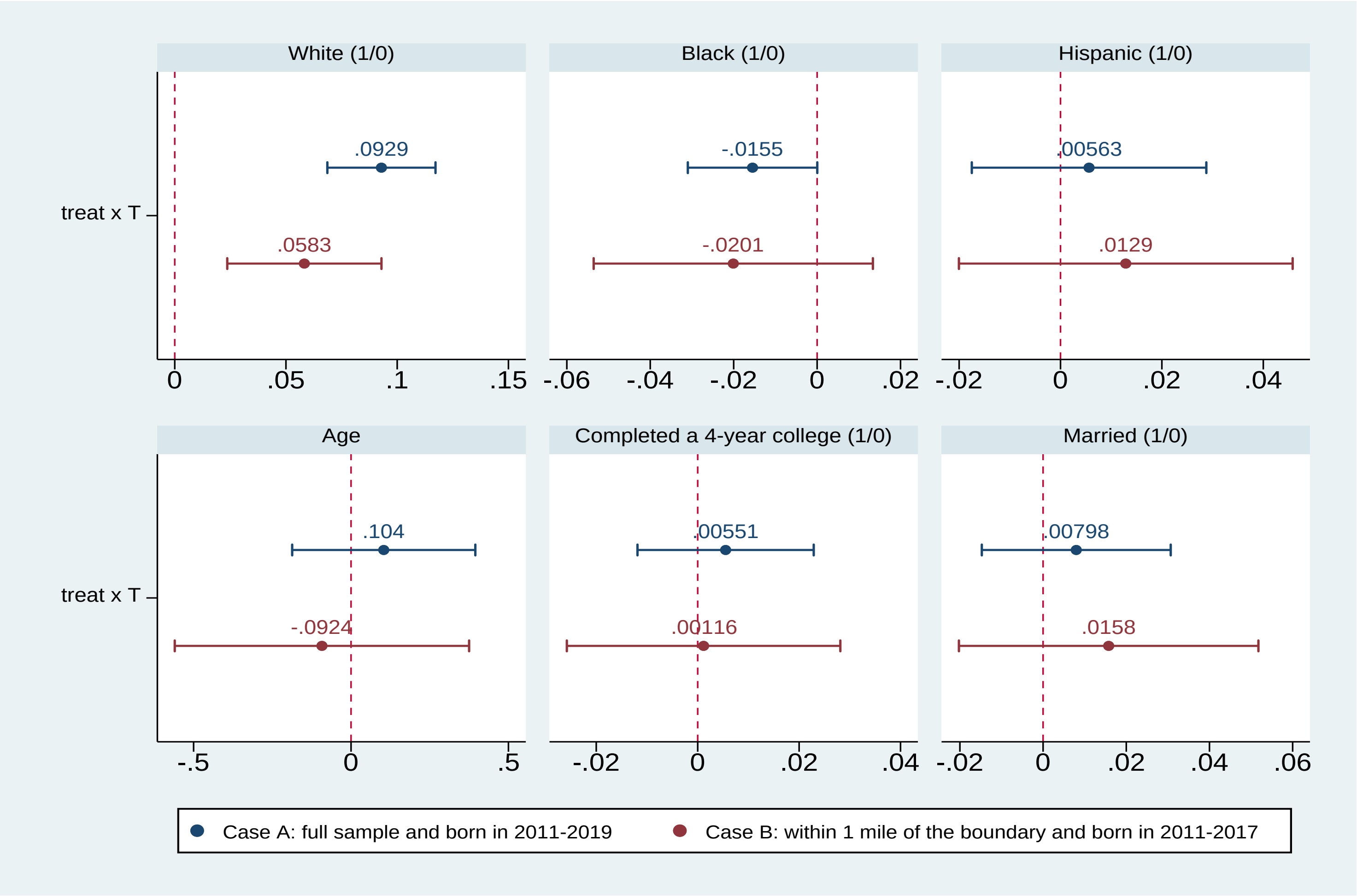
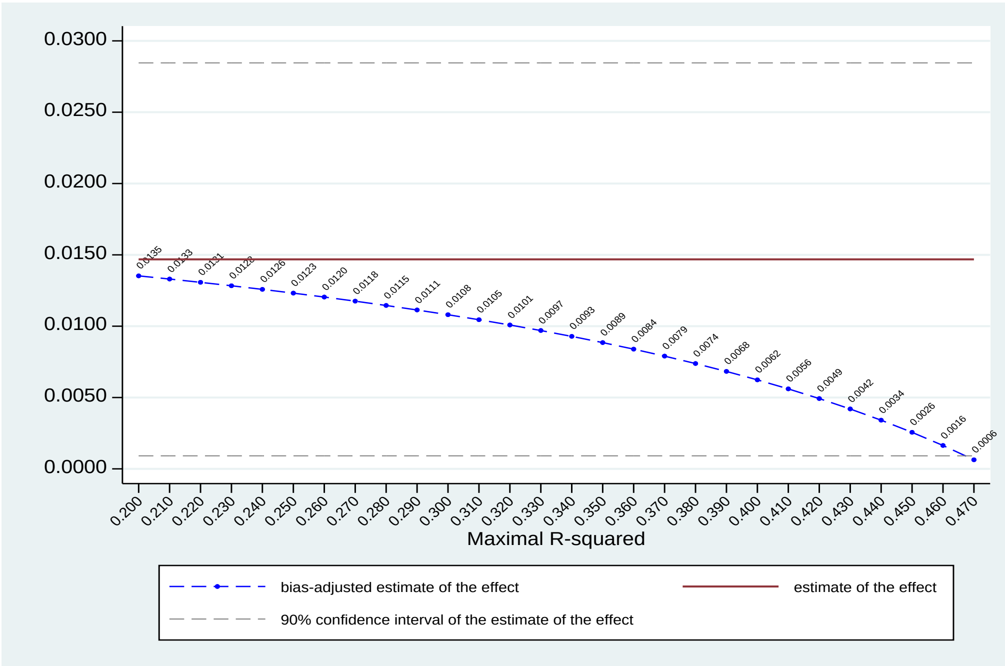


Figure 4: Comparisons of Maternal Demographic Characteristics between the Treatment Group and the Control Group

Notes: The estimation samples include live and singleton births among mothers who live in Newark, New Jersey (Case A), or who live in Newark, New Jersey, and within 1 mile of the boundary (Case B) between the areas serviced by the two water treatment plants (shown in Figure 1). The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2019 (Case A) or between 2016 and 2017 (Case B). Each subgraph contains two “rope ladder” plots for Cases A and B, showing the point estimates and the associated 95% confidence intervals of the coefficient on “treat × T” of an ordinary least squares (OLS) regression of a maternal demographic characteristic on an intercept and “treat × T”, together with the year-month of birth (i.e., monthly) fixed effects and the residential address fixed effects. Standard errors in all OLS regressions are clustered by the year-month (i.e., monthly) of birth. The numbers of observations in Cases A and B are 34,276 and 17,684, respectively.

Panel A



Panel B

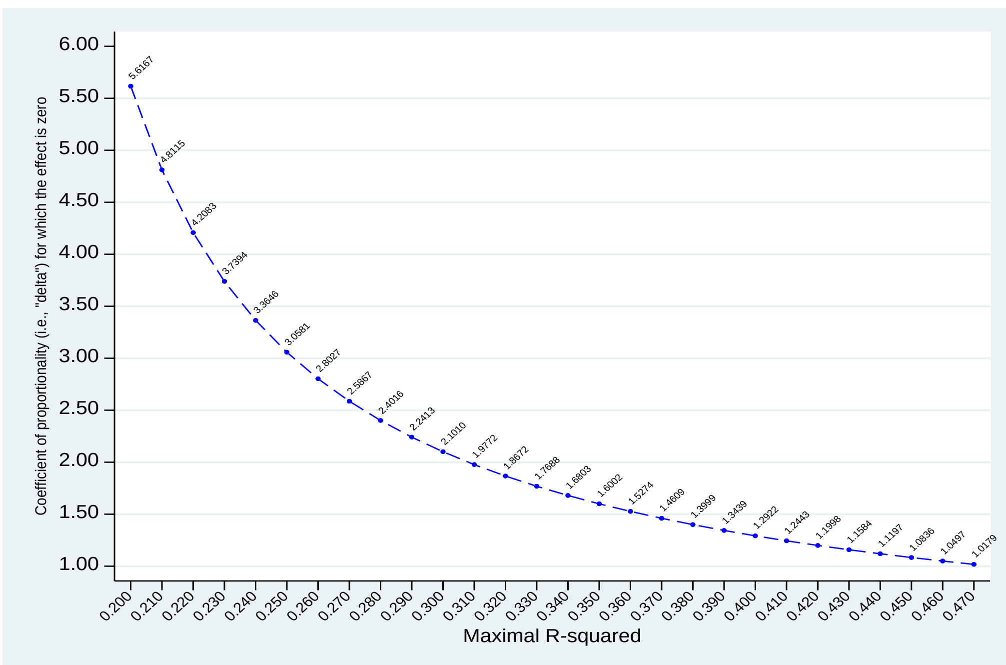
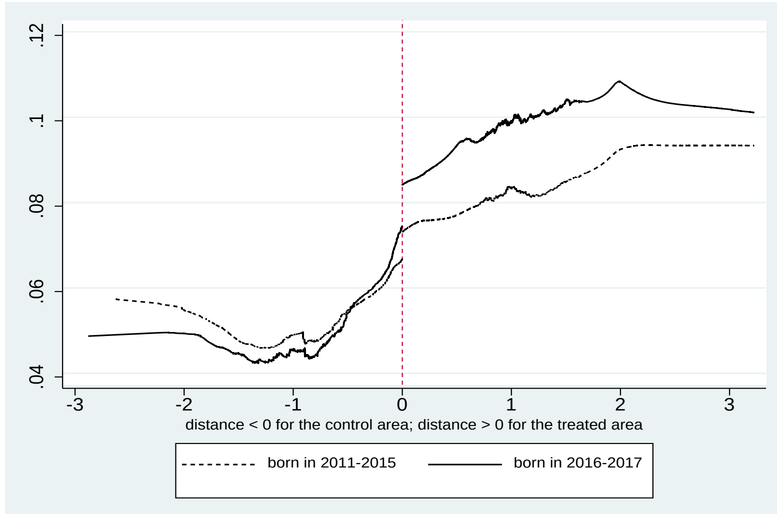


Figure 5: Robustness Checks on the Estimates of the Effect of Prenatal Exposure to Lead on Low Birth Weight

Notes: In Panel A the red line represents the estimate of the effect, which is 0.0147 and statistically significant at the 10% level (reported in column 1 of Table 3). The associated confidence interval at the 90% level is also reported in Panel A. The bias-adjusted estimate of the effect is referred to as "beta star" in Oster (2019). Also in Oster (2019) the coefficient of proportionality is referred to as "delta," which is the ratio of selection on unobservables over selection on observables. In Oster (2019) selection on observables (or unobservables) is defined by the covariance between the treatment dummy variable and observables (or unobservables) divided by the variance of observables (or unobservables).

Panel A



Panel B

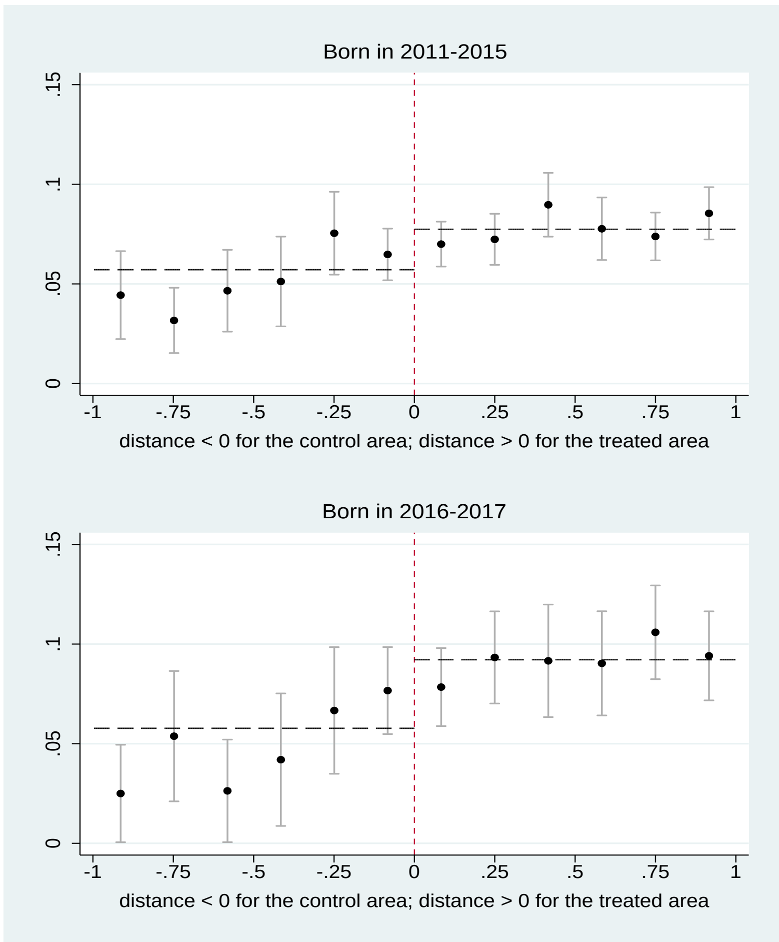


Figure 6: Low Birth Weight Rate and Distance to the Boundary between the Areas Serviced by the Two Water Treatment Plants

Notes: This figure shows the relationship between the low birth weight (LBW, birth weight < 2,500 grams) rate among singleton births and the distance (measured in miles) of each home address to the boundary between the areas serviced by the two water treatment plants (shown in Figure 1). The treated area is the area serviced by the Pequannock plant; the control area is the area serviced by the Wanaque plant. In Panel A this relationship is represented by smoothed lines, produced by Stata's "lowess" smoothing estimator with the default bandwidth of 0.8. In Panel B this relationship is represented by binned sample means and the associated 95% confidence intervals for home addresses that are within 1 mile of the boundary; dashed horizontal lines represent the average LBW rates by treatment status and treatment period. The two plots in Panel B were produced by the "rdplot" package (Stata code downloaded from https://rdpackages.github.io/, accessed in September 2021) developed by Calonico et al. (2017) and Calonico, Cattaneo and Titiunik (2014).

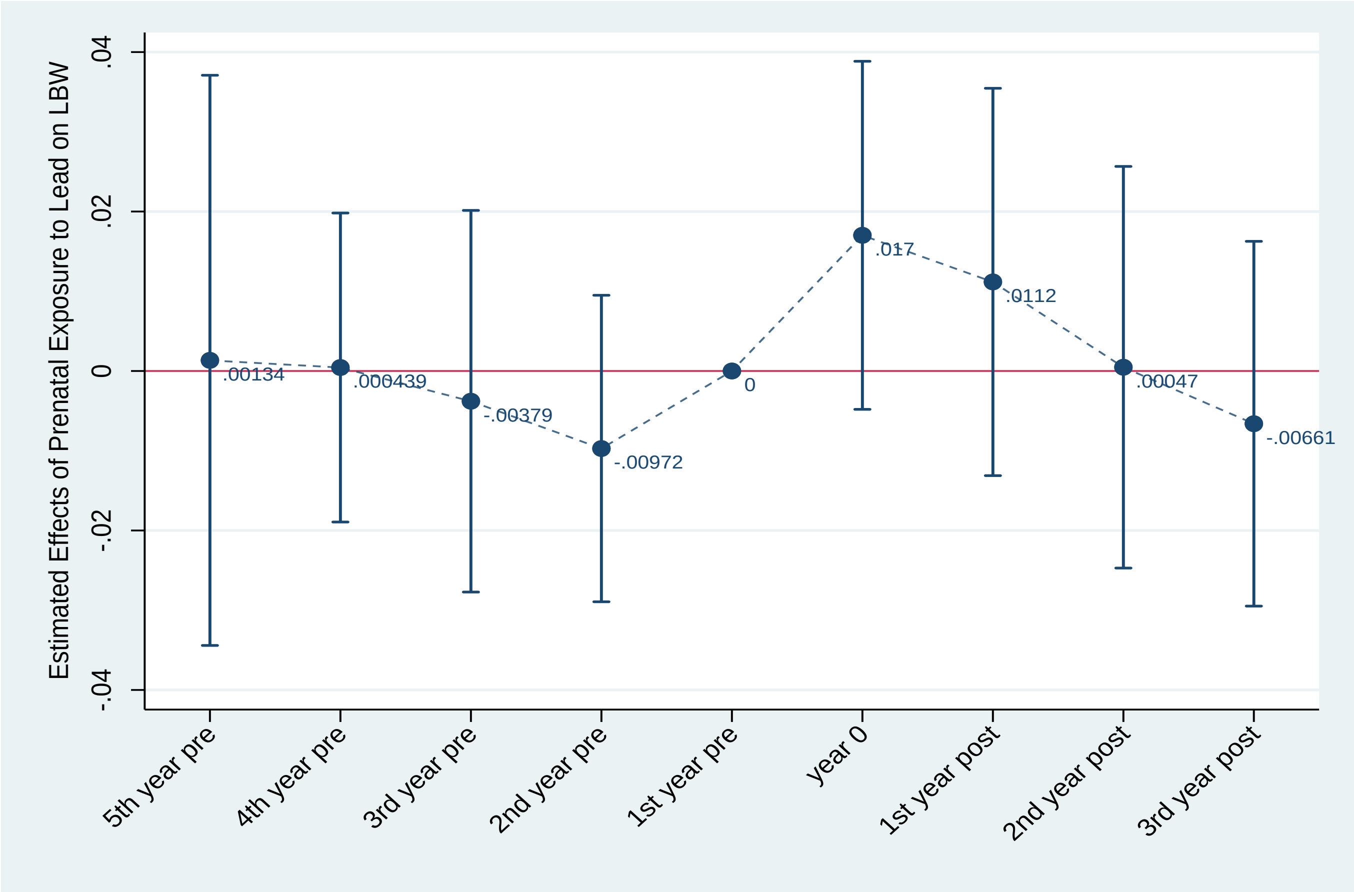


Figure 7: Event Study of Effects of Prenatal Exposure to Lead on Low Birth Weight

Notes: The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The outcome variable is low birth weight (LBW), a binary variable equal to one for birth weight < 2,500 grams, and equal to zero otherwise. The intervals reported (in the "rope ladder" plots) are constructed at the 95% confidence level. Birth years are from 2011 to 2019. Birth year 2016 is defined as year 0, when the treatment was present. The reference category is birth year 2015 (i.e., the year before the treatment was present). The treatment group includes mothers living in the area serviced by the Pequannock plant. The control group includes mothers living in the area serviced by the Wanaque plant. Standard errors are clustered by the year-month (i.e., monthly) of birth. The number of observations in the estimation sample is 34,276.

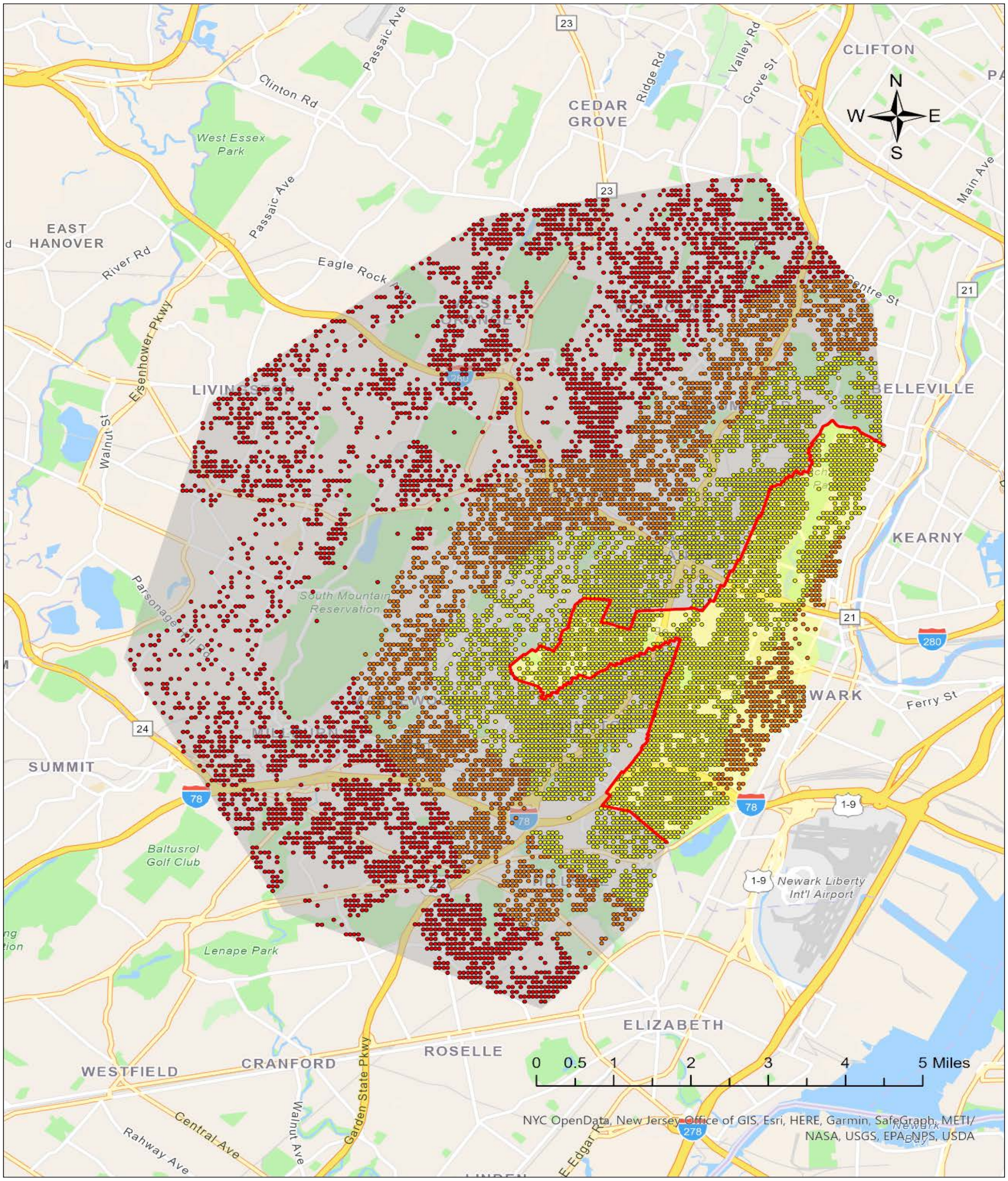
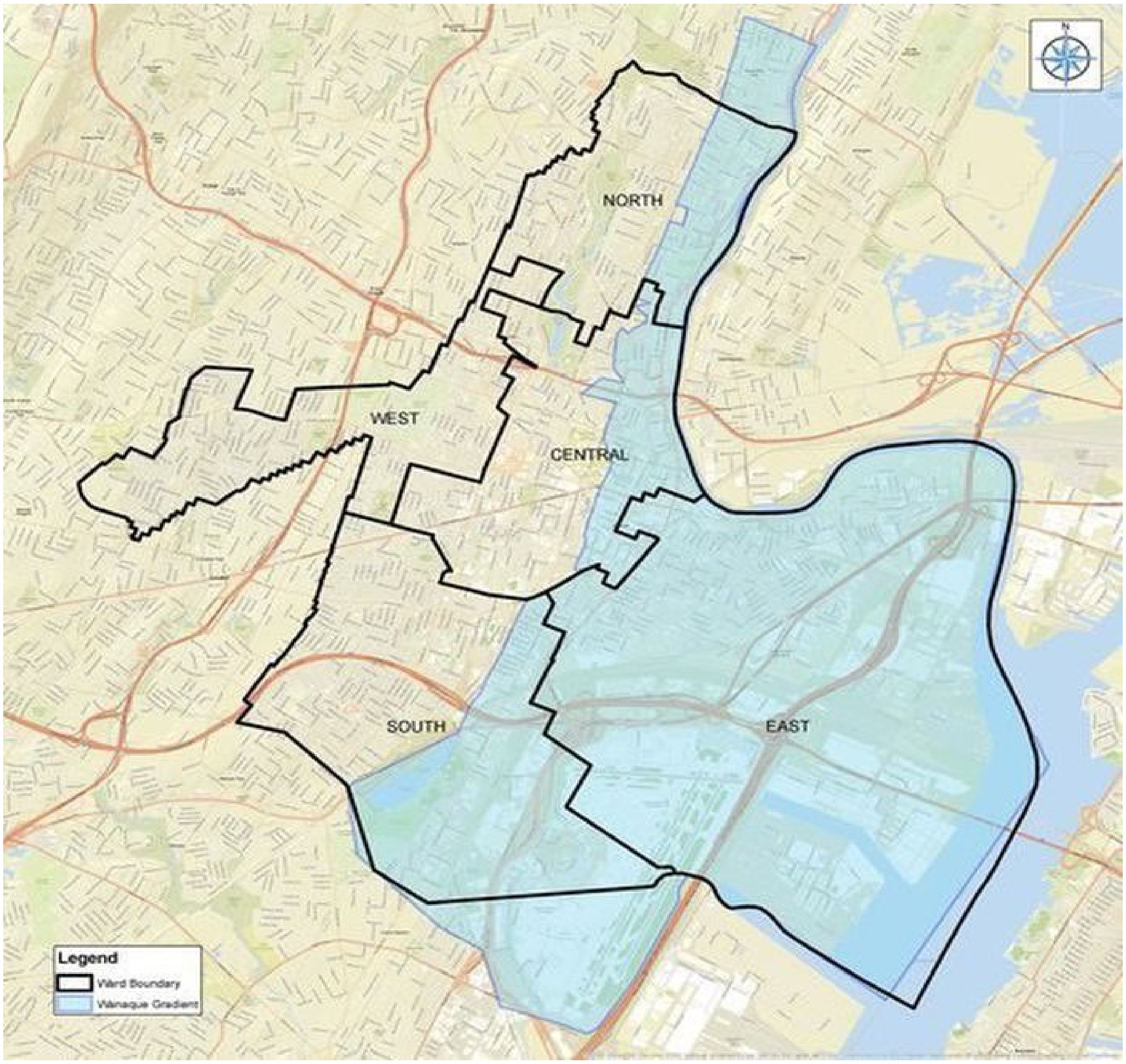


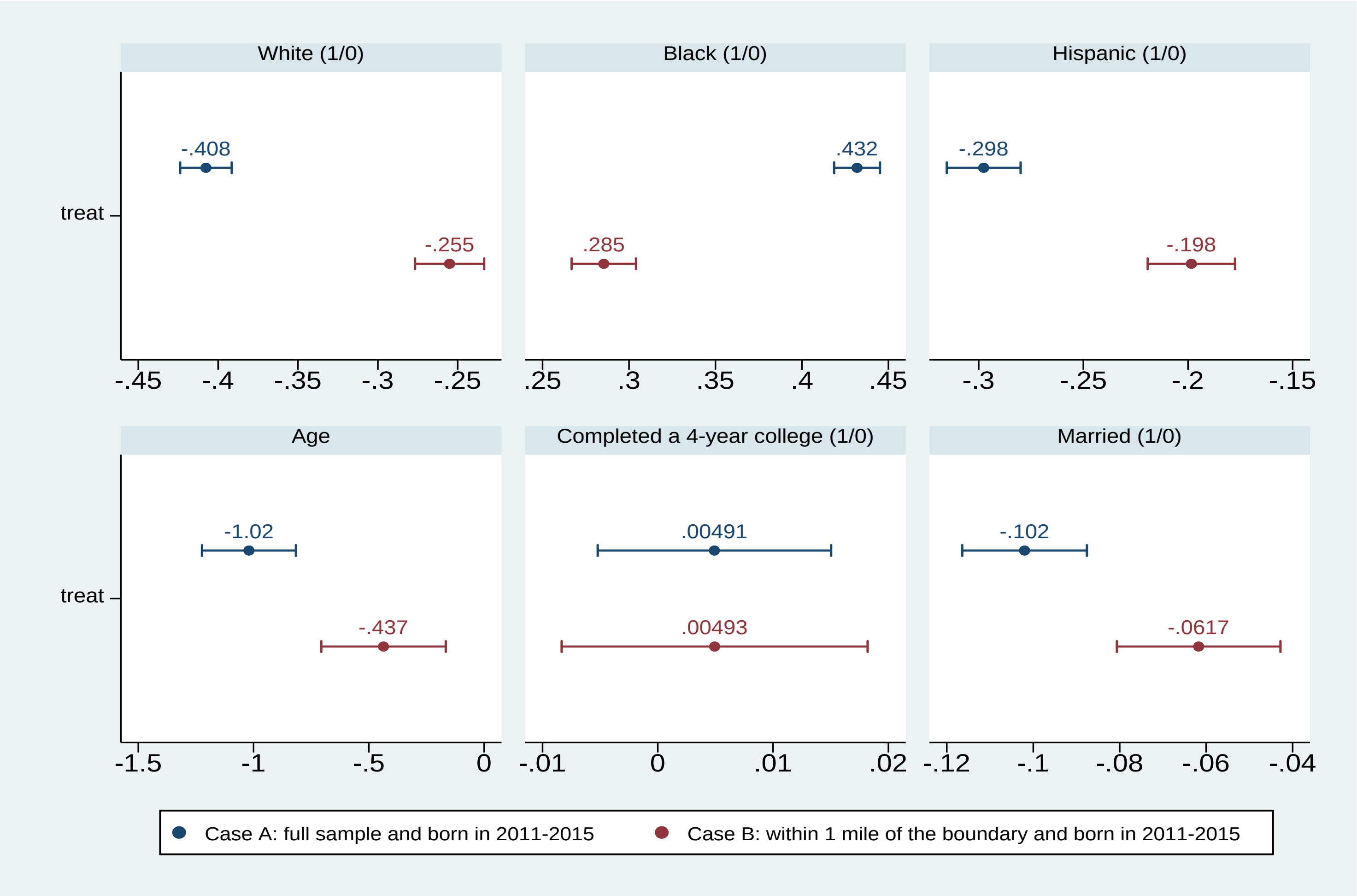
Figure 8: Area of Newark Serviced by the Pequannock Plant and the Area West of Newark

Notes: The georeferenced image is shown on the map. Explanations about the georeferencing were given in Figure 1's notes. The area shaded in yellow indicates the area of Newark serviced by the Pequannock plant; the red line shows that area's border excluding the boundary (shown in Figure 1) between the service area of the Pequannock plant and the service area of the Wanaque plant. The area shaded in gray indicates the area west of the area of Newark serviced by the Pequannock plant. The yellow dots represent home addresses that are within 1 mile of the border; the orange dots represent home addresses that are between 1 and 2 miles of the border; and the red dots represent home addresses that are between 2 and 5.5 miles (which is the maximum distance in our data) of the border.



Appendix Figure A1: Service Areas of the Two Water Treatment Plants of Newark, New Jersey

Notes: This figure shows the image of a map of the City of Newark (which is divided into five wards) and the areas served by the two water treatment plants. The image was obtained from the following source: https://www.nj.com/essex/2018/11/newarks_now_under_a_national_spotlight_for_lead_in.html (accessed in November 2019). In this image the area shaded in blue is served by the Wanaque plant; the area of the rest of the city (i.e., the unshaded area within the city's border) is served by the Pequannock plant.



Appendix Figure A2: Comparisons of Maternal Demographic Characteristics between the Treatment Group and the Control Group for the Pre-Treatment Period

Notes: The estimation samples include live and singleton births that occurred between 2011 and 2015 (the pre-treatment period), and among mothers who live in Newark, New Jersey (Case A), or who live in Newark, New Jersey, and within 1 mile of the boundary (Case B) between the areas serviced by the two water treatment plants (shown in Figure 1). The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. Each subgraph contains two "rope ladder" plots for Cases A and B, showing the point estimates and the associated 95% confidence intervals of the coefficient on the "treat" dummy variable of an ordinary least squares (OLS) regression of a maternal demographic characteristic on an intercept and the "treat" dummy variable, together with the year-month of birth (i.e., monthly) fixed effects. Standard errors in all OLS regressions are clustered by the year-month (i.e., monthly) of birth. The numbers of observations in Cases A and B are 19,284 and 12,816, respectively.

Table 1: Summary Statistics

Samples:	Control group			Treatment group			Full
Treatment periods:	Pre	Post	Post	Pre	Post	Post	sample
Birth years:	2011-2015	2016-2017	2016-2019	2011-2015	2016-2017	2016-2019	2011-2019
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Birth weight (in grams), among singleton births	3307.991	3295.058	3267.412	3217.152	3178.111	3182.602	3227.627
	[515.709]	[535.797]	[551.023]	[548.341]	[590.489]	[595.238]	[560.304]
Low birth weight (1/0): birth weight < 2,500 grams, among singleton births	0.054	0.054	0.065	0.081	0.095	0.095	0.079
	[0.226]	[0.225]	[0.247]	[0.273]	[0.294]	[0.293]	[0.269]
Gestational length (in weeks), among singleton births*	38.723	39.083	38.978	38.484	38.752	38.752	38.667
	[2.005]	[1.742]	[1.892]	[2.290]	[2.203]	[2.239]	[2.189]
Preterm (1/0): gestational length < 37 weeks, among singleton births*	0.077	0.055	0.064	0.098	0.089	0.086	0.087
	[0.267]	[0.229]	[0.244]	[0.297]	[0.284]	[0.281]	[0.281]
Female baby (1/0)	0.505	0.489	0.496	0.491	0.491	0.494	0.495
	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]
Mother's age	28.629	29.486	29.598	27.601	28.552	28.711	28.367
	[6.021]	[6.064]	[6.083]	[6.187]	[6.101]	[6.143]	[6.172]
Mother being White (1/0)	0.710	0.594	0.543	0.303	0.273	0.237	0.379
	[0.454]	[0.491]	[0.498]	[0.459]	[0.446]	[0.425]	[0.485]
Mother being Black (1/0)	0.211	0.203	0.203	0.643	0.606	0.610	0.506
	[0.408]	[0.402]	[0.402]	[0.479]	[0.489]	[0.488]	[0.500]
Mother being Hispanic (1/0)	0.657	0.674	0.662	0.360	0.386	0.379	0.453
	[0.475]	[0.469]	[0.473]	[0.480]	[0.487]	[0.485]	[0.498]
Mother having completed a four-year college or higher (1/0)	0.136	0.117	0.128	0.141	0.128	0.138	0.137
	[0.342]	[0.321]	[0.335]	[0.348]	[0.334]	[0.345]	[0.344]
Mother being married (1/0)	0.358	0.394	0.400	0.256	0.299	0.315	0.309
	[0.480]	[0.489]	[0.490]	[0.437]	[0.458]	[0.465]	[0.462]
Number of previous live births the mother had	1.081	1.102	1.143	1.236	1.283	1.281	1.213
	[1.194]	[1.206]	[1.223]	[1.372]	[1.373]	[1.377]	[1.330]
Mother having previous preterm birth (1/0)	0.010	0.021	0.024	0.013	0.036	0.032	0.020
	[0.100]	[0.143]	[0.154]	[0.114]	[0.186]	[0.177]	[0.140]
Number of prenatal visits	9.725	9.932	9.556	8.886	9.232	8.985	9.139
	[3.552]	[3.903]	[3.949]	[3.806]	[3.941]	[4.047]	[3.875]
Mother having smoked before or during pregnancy (1/0)	0.043	0.049	0.045	0.080	0.075	0.067	0.066
	[0.204]	[0.216]	[0.208]	[0.271]	[0.263]	[0.251]	[0.247]
Number of observations	5,596	2,216	4,347	13,688	5,210	10,645	34,276

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The means and standard deviations (in brackets) reported in the table are based on the estimation sample including live and singleton births among mothers who live in Newark, New Jersey. The treatment group includes mothers living in the area serviced by the Pequannock plant. The control group includes mothers living the area serviced by the Wanaque plant. *: The numbers of observations are 5,497; 2,132; 4,190; 13,489; 4,987; 10,171; 33,347 for columns 1 through 7, respectively.

Table 2: Summary Statistics, within 1-Mile of the Boundary between the Areas Serviced by the Two Water Treatment Plants

Samples:	Control group			Treatment group			Full
Treatment periods:	Pre	Post	Post	Pre	Post	Post	sample
Birth years:	2011-2015	2016-2017	2016-2019	2011-2015	2016-2017	2016-2019	2011-2019
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Birth weight (in grams), among singleton births	3296.502	3275.951	3243.062	3230.507	3189.932	3192.229	3230.414
	[513.024]	[551.206]	[561.163]	[541.946]	[578.428]	[591.952]	[557.046]
Low birth weight (1/0): birth weight < 2,500 grams, among singleton births	0.056	0.057	0.069	0.077	0.092	0.092	0.077
	[0.230]	[0.232]	[0.254]	[0.266]	[0.290]	[0.289]	[0.267]
Gestational length (in weeks), among singleton births*	38.694	39.036	38.901	38.493	38.770	38.755	38.654
	[2.031]	[1.816]	[2.004]	[2.258]	[2.105]	[2.214]	[2.185]
Preterm (1/0): gestational length < 37 weeks, among singleton births*	0.078	0.063	0.071	0.093	0.086	0.085	0.085
	[0.268]	[0.243]	[0.257]	[0.290]	[0.280]	[0.279]	[0.280]
Female baby (1/0)	0.501	0.486	0.492	0.491	0.491	0.495	0.494
	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]	[0.500]
Mother's age	28.050	28.945	29.106	27.600	28.401	28.632	28.176
	[6.041]	[6.139]	[6.089]	[6.180]	[6.119]	[6.142]	[6.160]
Mother being White (1/0)	0.632	0.540	0.485	0.378	0.342	0.295	0.405
	[0.482]	[0.499]	[0.500]	[0.485]	[0.474]	[0.456]	[0.491]
Mother being Black (1/0)	0.282	0.257	0.265	0.568	0.522	0.531	0.475
	[0.450]	[0.437]	[0.441]	[0.495]	[0.500]	[0.499]	[0.499]
Mother being Hispanic (1/0)	0.644	0.661	0.641	0.446	0.475	0.465	0.507
	[0.479]	[0.473]	[0.480]	[0.497]	[0.499]	[0.499]	[0.500]
Mother having completed a four-year college or higher (1/0)	0.135	0.115	0.126	0.139	0.121	0.128	0.133
	[0.341]	[0.319]	[0.332]	[0.346]	[0.326]	[0.334]	[0.340]
Mother being married (1/0)	0.322	0.353	0.360	0.260	0.307	0.315	0.299
	[0.467]	[0.478]	[0.480]	[0.438]	[0.461]	[0.465]	[0.458]
Number of previous live births the mother had	1.090	1.135	1.186	1.219	1.233	1.258	1.207
	[1.237]	[1.267]	[1.283]	[1.345]	[1.322]	[1.350]	[1.324]
Mother having previous preterm birth (1/0)	0.011	0.019	0.024	0.014	0.034	0.032	0.020
	[0.103]	[0.136]	[0.154]	[0.116]	[0.182]	[0.175]	[0.140]
Number of prenatal visits	9.603	9.876	9.444	9.049	9.395	9.127	9.208
	[3.592]	[3.924]	[3.954]	[3.761]	[3.891]	[4.023]	[3.848]
Mother having smoked before or during pregnancy (1/0)	0.048	0.053	0.050	0.078	0.068	0.064	0.065
	[0.213]	[0.224]	[0.218]	[0.268]	[0.251]	[0.245]	[0.247]
Number of observations	3,514	1,423	2,792	9,302	3,445	7,029	22,637

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The means and standard deviations (in brackets) reported in the table are based on the estimation sample including live and singleton births among mothers who live in Newark, New Jersey, and within 1 mile of the boundary between the areas serviced by the two water treatment plants (shown in Figure 1). The treatment group includes mothers living in the area serviced by the Pequannock plant. The control group includes mothers living the area serviced by the Wanaque plant. *: The numbers of observations are 3,451; 1,366; 2,682; 9,170; 3,305; 6,719; 22,022 for columns 1 through 7, respectively.

Table 3: Effects of Prenatal Exposure to Lead on Birth Outcomes

Dependent variables:	Low birth weight (1/0, equal to 1 if birth weight < 2,500 grams)			Preterm (1/0, equal to 1 if gestational length < 37 weeks)			Birth weight (in grams)			Gestational length (in weeks)		
Birth years used in the estimation:	2011-2017	2011-2018	2011-2019	2011-2017	2011-2018	2011-2019	2011-2017	2011-2018	2011-2019	2011-2017	2011-2018	2011-2019
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Treat × T	0.0147*	0.0110	0.0089	0.0191**	0.0162**	0.0125**	-30.7860**	-19.4990	-10.0914	-0.1638***	-0.1136*	-0.0828
	(0.0083)	(0.0071)	(0.0064)	(0.0080)	(0.0067)	(0.0063)	(15.2933)	(13.9825)	(13.1544)	(0.0602)	(0.0580)	(0.0526)
Number of observations	26,710	30,645	34,276	26,105	29,868	33,347	26,710	30,645	34,276	26,105	29,868	33,347
R-squared	0.1403	0.1334	0.1279	0.1612	0.1516	0.1444	0.1764	0.1689	0.1627	0.1787	0.1711	0.1644
Control variables												
Individual level demographic variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Residential address fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2017 (in columns 1, 4, 7, 10), between 2016 and 2018 (in columns 2, 5, 8, 11), or between 2016 and 2019 (in columns 3, 6, 9, 12). Individual level demographic variables controlled for are infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * p -value < 0.1; ** p -value < 0.05; *** p -value < 0.01.

Table 4: Checks on the Common Pre-Treatment Trend Using Fake Post-Treatment Periods

Dependent variables:	Low birth weight (1/0, equal to 1 if birth weight < 2,500 grams)				Preterm (1/0, equal to 1 if gestational length < 37 weeks)			
Birth years used for the fake post-treatment period:	2015	2014-2015	2013-2015	2012-2015	2015	2014-2015	2013-2015	2012-2015
Birth years used for the pre-treatment period:	2011-2014	2011-2013	2011-2012	2011	2011-2014	2011-2013	2011-2012	2011
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Treat × T	0.0042	-0.0027	0.0005	0.0267	0.0040	0.0050	-0.0097	0.0026
	(0.0087)	(0.0090)	(0.0095)	(0.0178)	(0.0100)	(0.0098)	(0.0095)	(0.0218)
Number of observations	19,284	19,284	19,284	19,284	18,986	18,986	18,986	18,986
Control variables								
Individual level demographic variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Residential address fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The definitions of the pre-treatment period (T = 0) and the fake post-treatment period (T = 1) are given in the table. Individual level demographic variables controlled for are infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * *p* -value < 0.1; ** *p* -value < 0.05; *** *p* -value < 0.01.

Table 5: Effects of Prenatal Exposure to Lead on Birth Outcomes near the Boundary between the Areas Serviced by the Two Water Treatment Plants

Dependent variables:	Low birth weight (1/0, equal to 1 if birth weight < 2,500 grams)				Preterm (1/0, equal to 1 if gestational length < 37 weeks)			
Birth years used in the estimation: 2011-2017	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Sample includes home addresses that are within 1 mile of the boundary between the areas serviced by the two water treatment plants.								
Treat × T	0.0147*	0.0145*	0.0146*	0.0146*	0.0077	0.0076	0.0076	0.0077
	(0.0086)	(0.0086)	(0.0086)	(0.0086)	(0.0087)	(0.0087)	(0.0087)	(0.0087)
Treat	-0.0036	-0.0069	-0.0068	-0.0036	-0.0028	-0.0045	-0.0050	-0.0025
	(0.0074)	(0.0073)	(0.0074)	(0.0098)	(0.0075)	(0.0076)	(0.0077)	(0.0104)
Number of observations	17,684	17,684	17,684	17,684	17,292	17,292	17,292	17,292
Panel B: Sample includes home addresses that are within 2 miles of the boundary between the areas serviced by the two water treatment plants.								
Treat × T	0.0163**	0.0163**	0.0163**	0.0164**	0.0139*	0.0139*	0.0139*	0.0139*
	(0.0071)	(0.0071)	(0.0071)	(0.0071)	(0.0075)	(0.0075)	(0.0075)	(0.0075)
Treat	0.0033	0.0031	0.0028	-0.0070	-0.0018	-0.0017	-0.0019	-0.0086
	(0.0056)	(0.0055)	(0.0055)	(0.0080)	(0.0060)	(0.0060)	(0.0060)	(0.0087)
Number of observations	24,050	24,050	24,050	24,050	23,506	23,506	23,506	23,506
Control variables used in Panels A and B								
Individual level demographic variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Distance (signed)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Distance (signed) squared	No	Yes	No	Yes	No	Yes	No	Yes
Treat × Distance (signed)	No	No	Yes	Yes	No	No	Yes	Yes
Treat × Distance (signed) squared	No	No	No	Yes	No	No	No	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2017. Distance (signed) is the distance (measured in miles) between a home address and the boundary between the areas serviced by the two water treatment plants (shown in Figure 1): the signed distance is positive for the treatment group and negative for the control group. Individual level demographic variables controlled for are infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * p -value < 0.1; ** p -value < 0.05; *** p -value < 0.01.

Table 6: Effects of Prenatal Exposure to Lead on Low Birth Weight Based on Alternative Control Group Definitions

Birth years used in the estimation:	2011-2017	2011-2018	2011-2019
	(1)	(2)	(3)
Panel A: The control group includes mothers living in the area east of the area serviced by the Wanaque plant, including the following New Jersey cities and towns: Bayonne, Elizabeth, Harrison, Jersey City, and Kearny.			
Treat × T	0.0161**	0.0131***	0.0134***
	(0.0061)	(0.0049)	(0.0043)
Number of observations	64,160	73,596	82,656
Residential address fixed effects	Yes	Yes	Yes
Panel B: The control group includes mothers living in the area west of the area of Newark serviced by the Pequannock plant. The estimation sample includes home addresses that are within 1 mile of the boundary (shown in Figure 8).			
Treat × T	0.0038	-0.0008	-0.0014
	(0.0065)	(0.0059)	(0.0054)
Number of observations	31,321	36,052	40,474
Distance (in miles) and treat (1/0)	Yes	Yes	Yes
Panel C: The control group includes mothers living in the area west of the area of Newark serviced by the Pequannock plant. The estimation sample includes home addresses that are within 3 miles of the boundary (shown in Figure 8) but excluding home addresses in the control group that are within 1 mile of that boundary.			
Treat × T	0.0153**	0.0108*	0.0111**
	(0.0069)	(0.0061)	(0.0055)
Number of observations	33,615	38,584	43,388
Residential address fixed effects	Yes	Yes	Yes
Panel D: The control group includes mothers living in the area west of the area of Newark serviced by the Pequannock plant. The estimation sample includes home addresses that are within 5 miles of the boundary (shown in Figure 8) but excluding home addresses in the control group that are within 1 mile of that boundary.			
Treat × T	0.0162**	0.0097	0.0096*
	(0.0066)	(0.0061)	(0.0054)
Number of observations	38,014	43,621	49,067
Residential address fixed effects	Yes	Yes	Yes
Control variables used in Panels A through D			
Individual level demographic variables	Yes	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. Low birth weight is a dummy variable, equal to one if birth weight is less than 2,500 grams and equal to zero otherwise. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The definitions of alternative control groups (treat = 0) are given in the table. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2017 (in column 1), between 2016 and 2018 (in column 2), or between 2016 and 2019 (in column 3). Individual level demographic variables controlled for are infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0). In Panel B residential address fixed effects are replaced by controlling for treat (1/0) and the distance (measured in miles) between a home address and the boundary (shown in Figure 8). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * p-value < 0.1; ** p-value < 0.05; *** p-value < 0.01.

Table 7: Effects of Prenatal Exposure to Lead on Prenatal Visits and Maternal Smoking

	(1)	(2)
Panel A: Dependent variable is the number of prenatal visits.		
Treat × T	0.2131**	
	(0.1021)	
Treat × 3rd year post-treatment (i.e., 2019)		0.2510
		(0.1874)
Treat × 2nd year post-treatment (i.e., 2018)		0.4097**
		(0.1708)
Treat × 1st year post-treatment (i.e., 2017)		0.2302
		(0.1604)
Treat × year 0 (i.e., 2016)		-0.0303
		(0.1317)
Number of observations	34,276	34,276
Panel B: Dependent variable is maternal smoking before or during pregnancy (1/0)		
Treat × T	-0.0142**	
	(0.0057)	
Treat × 3rd year post-treatment (i.e., 2019)		-0.0141
		(0.0100)
Treat × 2nd year post-treatment (i.e., 2018)		-0.0192**
		(0.0084)
Treat × 1st year post-treatment (i.e., 2017)		-0.0130
		(0.0087)
Treat × year 0 (i.e., 2016)		-0.0104
		(0.0082)
Number of observations	34,276	34,276
Control variables used in Panels A and B		
Individual level demographic variables	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes
Residential address fixed effects	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2019. Individual level demographic variables controlled for are infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, and mother having previous preterm birth (1/0). In column (2) the "treat" dummy variable is interacted with dummy variables for each year of the post-treatment period (i.e., 2016 through 2019). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * p-value < 0.1; ** p-value < 0.05; *** p-value < 0.01.

Table 8: Effects of Prenatal Exposure to Lead on Birth Outcomes by Sex

	Infant's sex	
Estimation by subsample:	Male	Female
	(1)	(2)
Panel A: Dependent variable is low birth weight (1/0, equal to 1 if birth weight < 2,500 grams).		
Treat × T	0.0212**	0.0150
	(0.0104)	(0.0130)
Number of observations	13,520	13,190
Panel B: Dependent variable is preterm (1/0, equal to 1 if gestational length < 37 weeks).		
Treat × T	0.0344***	0.0119
	(0.0124)	(0.0124)
Number of observations	13,204	12,901
Panel C: Dependent variable is birth weight (in grams).		
Treat × T	-47.2849*	-30.5808
	(25.5924)	(25.2247)
Number of observations	13,520	13,190
Panel D: Dependent variable is gestational length (in weeks).		
Treat × T	-0.3267***	-0.0671
	(0.0910)	(0.0877)
Number of observations	13,204	12,901
Control variables used in Panels A through D		
Individual level demographic variables	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes
Residential address fixed effects	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The birth years used in the estimation are 2011 through 2017. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2017. Individual level demographic variables controlled for are mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * *p* -value < 0.1; ** *p* -value < 0.05; *** *p* -value < 0.01.

Appendix Table A1: Demographics of Newark, NJ and Other Cities

	Newark, NJ	U.S.	Identified cities in the ACS with population +/- 10% of Newark, NJ
Median household income ($)	44,000	72,500	60,400
% Below poverty	27.1	13.4	19.8
% High school educated or below (ages 24+)	63.7	44.2	41.2
% College educated or above	14.6	32.3	35.9
% White	29.1	72.3	62.4
% Black	50.2	12.7	20.0
% Hispanic	36.6	18.1	19.0
% Married	33.0	50.0	40.2
% Immigrant	37.8	15.2	16.8
% Uninsured	17.3	9.1	10.3
% Rent	73.8	34.1	47.6
% Homes built ≤ year 1979	71.2	49.3	67.9
Population	283,000	326,000,000	287,000

Notes: Statistics are based on the 2016–2019 American Community Surveys (ACS). Identification of cities in the ACS is incomplete; the following cities are identified with a population within +/- 10% of the population in Newark: Anchorage, AK; Buffalo, NY; Jersey City, NJ; Laredo, TX; Lincoln, NE; Pittsburgh, PA; Saint Louis, MO; Saint Paul, MN; Toledo, OH.

Appendix Table A2: Effects of Prenatal Exposure to Lead on Low Birth Weight, Full Set of Coefficient Estimates

Birth years used in the estimation:	2011-2017 For column (1) of Table 3, without controlling for prenatal visits and smoking (1)	2011-2017 For column (1) Table 3 (2)
Treat × T	0.0127	0.0147*
	(0.0087)	(0.0083)
Female baby (1/0)	0.0112***	0.0123***
	(0.0034)	(0.0034)
Mother's age	0.0017***	0.0026***
	(0.0004)	(0.0004)
Mother being White (1/0)	-0.0127*	-0.0107
	(0.0066)	(0.0067)
Mother being Black (1/0)	0.0072	0.0074
	(0.0077)	(0.0075)
Mother being Hispanic (1/0)	-0.0192***	-0.0121**
	(0.0049)	(0.0049)
Mother having completed a four-year college or higher (1/0)	-0.0140**	-0.0116**
	(0.0055)	(0.0056)
Mother being married (1/0)	-0.0173***	-0.0112***
	(0.0040)	(0.0040)
Number of previous live births the mother had	-0.0055***	-0.0106***
	(0.0017)	(0.0017)
Mother having previous preterm birth (1/0)	0.1524***	0.1556***
	(0.0209)	(0.0204)
Number of prenatal visits		-0.0093***
		(0.0005)
Mother having smoked before or during pregnancy (1/0)		0.0524***
		(0.0093)
Number of observations	26,710	26,710
R-squared	0.1227	0.1403
Control variables		
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes
Residential address fixed effects	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. Low birth weight is a dummy variable, equal to one if birth weight is less than 2,500 grams and equal to zero otherwise. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2017. Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * *p* -value < 0.1; ** *p* -value < 0.05; *** *p* -value < 0.01.

Appendix Table A3: Effects of Prenatal Exposure to Lead on Birth Outcomes, Controlling for Zip Code Specific Time Trend

Dependent variables:	Low birth weight (1/0, equal to 1 if birth weight < 2,500 grams)			Preterm (1/0, equal to 1 if gestational length < 37 weeks)			Birth weight (in grams)			Gestational length (in weeks)		
Birth years used in the estimation:	2011-2017	2011-2018	2011-2019	2011-2017	2011-2018	2011-2019	2011-2017	2011-2018	2011-2019	2011-2017	2011-2018	2011-2019
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Treat × T	0.0150*	0.0112	0.0083	0.0189**	0.0160**	0.0117*	-30.5986**	-18.6975	-8.0556	-0.1595**	-0.1088*	-0.0740
	(0.0083)	(0.0074)	(0.0067)	(0.0082)	(0.0071)	(0.0066)	(15.1592)	(13.9807)	(13.3057)	(0.0619)	(0.0598)	(0.0552)
Number of observations	26,710	30,645	34,276	26,105	29,868	33,347	26,710	30,645	34,276	26,105	29,868	33,347
R-squared	0.1424	0.1350	0.1292	0.1631	0.1528	0.1456	0.1780	0.1702	0.1638	0.1800	0.1721	0.1654
Control variables												
Individual level demographic variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Residential address fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Zip code specific linear time trend of year-month of birth	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2017 (in columns 1, 4, 7, 10), between 2016 and 2018 (in columns 2, 5, 8, 11), or between 2016 and 2019 (in columns 3, 6, 9, 12). Individual level demographic variables controlled for are infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * p -value < 0.1; ** p -value < 0.05; *** p -value < 0.01.

Appendix Table A4: Effects of Prenatal Exposure to Lead on Whether the Birth Is Female and on the Number of Births

Birth years used in the estimation:	2011-2017	2011-2017
Dependent variables:	Female (1/0): individual-level analysis	Number of births for each zip code-year and month of birth pair
	(1)	(2)
Panel A:		
Treat × T	0.0031	-1.2496
	(0.0162)	(1.2985)
Number of observations	26,710	756
Panel B:		
Treat × T	-0.0134	-0.8089
	(0.0134)	(0.6087)
Number of observations	33,615	2,108
Panel C:		
Treat × T	-0.0045	-0.9193
	(0.0113)	(0.5952)
Number of observations	38,014	2,566
Control variables used in Panels A, B and C		
Individual level demographic variables averaged at the zip code-monthly level	No	Yes
Individual level demographic variables	Yes	No
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes
Zip code fixed effects	No	Yes
Residential address fixed effects	Yes	No

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. In all four panels the pre-treatment period (T = 0) includes births that occurred between 2011 and 2015, and the post-treatment period (T = 1) includes births that occurred between 2016 and 2017.

In Panel A the treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant.

In Panel B the treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living in the area west of the area of Newark serviced by the Pequannock plant. The estimation sample includes home addresses that are within 3 miles of the boundary (shown in Figure 8) but excluding home addresses in the control group that are within 1 mile of that boundary.

In Panel C the treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living in the area west of the area of Newark serviced by the Pequannock plant. The estimation sample includes home addresses that are within 5 miles of the boundary (shown in Figure 8) but excluding home addresses in the control group that are within 1 mile of that boundary.

In column (1), estimations use individual level data. The dependent variable is the newborn being female (1/0). Individual level demographic variables controlled for are mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0).

In column (2), the dependent variable is the total number of births within each cell defined by the mother's residential zip code and her year and month of childbirth. Here, the "treat" variable is the proportion of observations that are in the treatment group for each cell defined by the mother's residential zip code and her year and month of childbirth. This "treat" variable is included as a control variable in the regression model. Individual level demographic variables are averaged over each cell defined by the mother's residential zip code and her year and month of childbirth. These individual level demographic variables include infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0).

In both columns, estimations use standard errors (reported in parentheses) that are clustered by the year-month (i.e., monthly) of birth year. * p -value < 0.1; ** p -value < 0.05; *** p -value < 0.01.

Appendix Table A5: Effects of Prenatal Exposure to Lead on Birth Outcomes by Maternal Characteristics

	Mother's race and ethnicity					Mother having completed a four-year college education or higher		Mother's marital status	
Estimation by subsample:	White	White and Non-Hispanic	White and Hispanic	Hispanic	Black	Yes	No	Married	Not married
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: Dependent variable is low birth weight (1/0, equal to 1 if birth weight < 2,500 grams).									
Treat × T	0.0146	-0.0532	0.0115	0.0121	0.0122	-0.0342	0.0148	0.0052	0.0191*
	(0.0106)	(0.0677)	(0.0126)	(0.0108)	(0.0210)	(0.0285)	(0.0096)	(0.0127)	(0.0102)
Number of observations	10,852	1,539	9,313	12,107	13,586	3,610	23,100	7,942	18,768
Panel B: Dependent variable is preterm (1/0, equal to 1 if gestational length < 37 weeks).									
Treat × T	0.0276**	0.0675	0.0232**	0.0255***	0.0050	-0.0101	0.0181*	0.0157	0.0177
	(0.0107)	(0.0737)	(0.0114)	(0.0094)	(0.0241)	(0.0349)	(0.0096)	(0.0136)	(0.0109)
Number of observations	10,575	1,498	9,077	11,799	13,323	3,551	22,554	7,762	18,343
Panel C: Dependent variable is birth weight (in grams).									
Treat × T	-34.2122	-132.9173	-16.9295	-36.7191*	5.2194	-43.7037	-29.8218	-42.2028	-26.2058
	(20.9072)	(152.7994)	(25.0801)	(21.5254)	(38.0367)	(77.7710)	(18.3357)	(25.4825)	(20.1201)
Number of observations	10,852	1,539	9,313	12,107	13,586	3,610	23,100	7,942	18,768
Panel D: Dependent variable is gestational length (in weeks).									
Treat × T	-0.1538**	-0.5294	-0.1255	-0.1655**	-0.0443	0.2039	-0.1598**	-0.0933	-0.1792**
	(0.0760)	(0.4668)	(0.0766)	(0.0698)	(0.1932)	(0.2660)	(0.0713)	(0.1011)	(0.0839)
Number of observations	10,575	1,498	9,077	11,799	13,323	3,551	22,554	7,762	18,343
Control variables used in Panels A through D									
Individual level demographic variables	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year-month of birth (i.e., monthly) fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Residential address fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: Data are from the New Jersey birth records on all live births collected by the New Jersey Department of Health. The estimation sample includes live and singleton births among mothers who live in Newark, New Jersey. The birth years used in the estimation are 2011 through 2017. The treatment group (treat = 1) includes mothers living in the area serviced by the Pequannock plant. The control group (treat = 0) includes mothers living the area serviced by the Wanaque plant. The pre-treatment period (T = 0) includes births that occurred between 2011 and 2015. The post-treatment period (T = 1) includes births that occurred between 2016 and 2017. Except those used as the conditioning variables shown in columns (1) through (9), individual level demographic variables controlled for are infant being female (1/0), mother's age, mother's race and ethnicity (1/0 dummy variables for White, Black, and Hispanic), mother having completed a four-year college education or higher (1/0), mother being married (1/0), number of previous live births the mother had, mother having previous preterm birth (1/0), number of prenatal visits, and mother having smoked before or during pregnancy (1/0). Standard errors (reported in parentheses) are clustered by the year-month (i.e., monthly) of birth. * *p* -value < 0.1; ** *p* -value < 0.05; *** *p* -value < 0.01.