Where Have all the Young Men Gone? Using Gender Ratios to Measure the Effect of Pollution on Fetal Death Rates*

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Abstract

We estimate the causal impact of ambient prenatal pollution exposure on fetal deaths. Since a complete census of true fetal deaths is impossible to obtain, we exploit the differential in fetal susceptibility to environmental stressors across genders to estimate this effect. Males are more vulnerable to maternal stress *in utero*, and thus are more likely to suffer fetal death due to pollution exposure. We use the Clean Air Act Amendments of 1970 (CAAA) as a source of exogenous variation in county-level ambient total suspended particulate matter (TSPs). We find that a one standard deviation increase in TSPs decreases the percentage of live births that are male by 0.9 percentage points. We then use the observed differences in neonatal and one-year mortality rates across genders in response to pollution exposure to estimate total fetal losses *in utero*. Our preferred calculations suggest the pollution reductions from the CAAA prevented approximately 36,000 fetal deaths in 1972.

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1 Introduction

Improvements in air quality have led to improvements in observable health outcomes such as birth weight and infant mortality, but little is known about fetal deaths. This is because fetal death data are rarely available, and when available are selectively measured, making it difficult to estimate the impacts of policy interventions. Policy choices based only on traditional infant health outcomes may not be made with full information, as they do not account for any fetal losses *in utero*. We introduce a metric to measure fetal death rates and use it to estimate the impacts of prenatal pollution exposure.

Greater information about fetal death effects would clearly be useful in effective policy construction. Unfortunately, examining policy effects on total birth rates cannot be used as an effective measure of fetal deaths as fertility choices such as frequency of intercourse or other behavioral decisions could also change in response to the policy intervention. Since total fertility may be a function of factors other than fetal deaths we instead examine the gender ratio at birth. We exploit the fact that males are more sensitive than females to negative health shocks *in utero* to estimate the impact of prenatal exposure to ambient total suspended particulate matter (TSPs) on fetal death. By using the observable gender differences in pollution-driven neonatal mortality rates as an estimate of relative gender sensitivity, we can convert gender ratio changes into an estimate of total fetal deaths caused by ambient TSPs.

The gender ratio for live births, unlike total fertility, is largely orthogonal to other fertility factors correlated with changes in pollution, and thus provides a less biased measure of fetal deaths. Unfortunately, relationships between the gender of live births and socioeconomic status make cross sectional analysis of pollution and gender ratios difficult (Almond and Edlund, 2007). We therefore employ a panel data, instrumental variables strategy, using the Clean Air Act Amendments of 1970 (CAAA), which imposed sanctions on firms in counties with pollution levels over a defined attainment threshold, as an exogenous driver of ambient TSP levels.[1] We use estimated CAAA

[1]This identification strategy was first used in Chay and Greenstone (2003a) and Chay, Greenstone, and Dobkin

attainment status as an instrument for changes in ambient pollution levels between 1970-1972, and then use a first-difference model to estimate the effect of pollution on the gender ratio. We find a statistically and economically significant association between ambient TSP levels and the fraction of live births that are male: a one unit increase in ambient TSP levels is associated with approximately a 0.033 percentage point change in the probability of a live birth being male (or a change in the gender ratio of males to females of 0.0014 with a baseline of 1.05), and a standard deviation increase in TSPs is associated with a 0.89 percentage point change (or a change in the gender ratio of 0.038).[2] These effects are larger when considering particularly vulnerable subgroups, such as less educated mothers, very young or very old mothers, and black children.

Using pollution changes from 1971-1972, Chay and Greenstone (2003a) (hereafter CG) find that the changes in TSPs associated with the CAAA reduced infant mortality rates. We expand on their findings by showing this effect differs by gender — the effect of pollution on neonatal mortality for males is approximately 3.6 times that for females. This observable differential pollution sensitivity between males and females after birth can be used as an estimate of the differential sensitivity *in utero*. Using this approximation, we estimate that a one-unit decrease in TSPs is associated with approximately 100 fewer male and 30 fewer female fetal deaths per 100,000 live births. This implies the changes in air quality between 1970 and 1972 caused by the CAAA in counties subject to its restrictions prevented approximately 36,000 fetal deaths in 1972, or a change of approximately 2% of live births in those counties. As a point of comparison, CG find the CAAA prevented approximately 1,300 infant deaths. This suggests a higher sensitivity to TSPs *in utero* than after birth, and that there are substantial health improvements due to reduced air pollution not currently quantified in the economics literature.

We also contribute to the literature on gender differences in response to external shocks. Prior research has focused largely on rare, one-time events, such as the "Killer Fog" in London dur-

(2003).

[2]Baseline values refer to the value in attainment counties in our sample in 1970.

ing December of 1952 (Lyster, 1974), earthquakes (Fukuda et al., 1998), radioactive fallout (Almond, Edlund, and Palme, 2007; Peterka, Peterková, and Likovský, 2007), famine (Almond et al., 2007), the collapse of the East German economy (Catalano, 2003), the French Revolutionary War (Kemkes, 2006), the September 11th attacks (Catalano, Bruckner, and Ahern, 2010), and other such events. Other work has examined more frequently experienced shocks including temperature (Lerchl, 1999; Catalano, Bruckner, and Smith, 2008; Helle, Helama, and Jokela, 2008), alcohol consumption (Nilsson, 2008), and job loss (Catalano et al., 2010). To our knowledge, this is the first paper to use a quasi-experimental, panel data design to consider the differential gender impacts caused by a common air pollutant.[3]

The remainder of this paper is organized as follows. Section 2 presents evidence of gender differences in susceptibility to external stress, and discusses the potential effects of pollution on fetal health. Section 3 provides some background on the CAAA. Section 4 outlines our identification strategy. Section 5 describes the data used in the analysis. Section 6 describes our main results. Section 7 presents several falsification tests and alternate specifications. Section 8 places our findings in context with prior work. Section 9 concludes.

2 Environmental stressors, fetal susceptibility, and gender effects

The health consequences of environmental externalities have received a good deal of attention in applied research as of late, much of it dedicated to the health impacts of lead, carbon monoxide, ozone, and particulate matter, four of the six "criteria pollutants" regulated by the EPA.[4] Lead has been linked to lowered IQ and increased aggression (Reyes, 2007; Nilsson, 2009) and increased infant mortality (Clay, Troesken, and Haines, 2010). Carbon monoxide has been linked to increased

[3]Note that our identification uses the drastic reduction in TSPs seen during the aftermath of the CAAA. Modern particulate levels are far lower in the United States. If effects are nonlinear, we may be estimating an upper bound of the effects likely to be seen today. However, in other currently industrializing countries, particulate levels are currently as high as they were during our period of analysis, if not higher.

[4]The term criteria pollutants refers to six commonly found air pollutants that are regulated by developing health-based and/or environmentally-based criteria for allowable levels. The current criteria pollutants are: particular matter, ground-level ozone, carbon monoxide, sulfur oxides, nitrogen oxides and lead.

infant mortality (Currie and Neidell, 2005), low birth weight and preterm birth (Ritz and Yu, 1999; Currie, Neidell, and Schmieder, 2009), and increased school absences in young children (Currie et al., 2009). Ozone has been linked to higher asthma rates and cardiac difficulties (Neidell, 2004, 2009; Lleras-Muney, 2010; Moretti and Neidell, 2011). Particulate matter has been found to increase infant mortality rates (Chay and Greenstone, 2003b,a; Knittel, Miller, and Sanders, 2009), as well as the incidence of low birth weight (Wang et al., 1997).

Studies on less pollutant-specific environmental factors have found negative fetal health effects. Currie and Walker (2011) find that decreased traffic pollution resulting from the introduction of EZ-Pass in New Jersey reduced the incidence of low birth weight and premature birth. Currie and Schmieder (2009) find negative effects of toxic releases for gestation, birth weight, and infant mortality rates, and Currie, Greenstone, and Moretti (2011) find that proximity to Superfund sites is associated with up to a 25% increase in the probability of congenital anomalies. Using the short-term closing of a Utah steel mill as an exogenous source of pollution variation, Parker, Mendola, and Woodruff (2008) find the plant closure period was accompanied by a reduction in preterm birth. A more comprehensive review of the fetal environmental literature is available in Currie (2011).

In this paper, we focus on TSPs as our pollutant of interest, the measure of airborne particulate matter used by the EPA during the timeframe of the CAAA. The term TSPs refers to all suspended, airborne liquid or solid particles smaller than 100 micrometers in size.[5] Suspended particulates can be naturally occurring (e.g., dust, dirt, and pollen) or a by-product of common economic activities such as fuel combustion (e.g., coal, gasoline and diesel), fires, and industrial activity. Particulates are the cause of a number of environmental problems, including decreased visibility, increased acidity of both water and soil, and plant death.

[5] As monitoring technology has advanced, regulatory attention has shifted to finer sizes of particulate matter, with much of the attention now on two size classifications: particulate matter smaller than 10 micrometers (PM10) and particulate matter smaller than 2.5 micrometers (PM2.5). Both of these size classifications are contained with the older TSP measure.

Exposure to particulate matter may impact fetal development in a number of ways. The mother's health may be compromised, which could indirectly harm fetal development. Inhaled particulates have been associated with health problems including difficulty breathing, decreased lung function, aggravated asthma, and cardiac difficulties, any of which could harm the mother and, in doing so, reduce the health capital available for the survival of the fetus. Smaller particulates can be transferred from the lungs into the bloodstream, causing internal damage for both the mother and the fetus directly.[6] Research supports that such direct fetal impacts are indeed possible — associations have been found between polycyclic aromatic hydrocarbons (PAHs), a type of particulate matter, and a number of pre- and early post-natal developmental problems including damage to the immune system, hindered neurological development, reduced birth weight and smaller head circumference, and impairment of neuron behavior associated with long-term memory formation.[7]

Research also suggests that negative environmental externalities can have damaging, long-run effects on a developing fetus. Elevated prenatal radiation exposure has been linked to lower test scores (Almond, Edlund, and Palme, 2009), and a medical study involving personal air monitoring systems during pregnancy found that higher prenatal pollution exposure was associated with lower IQ scores at age 5 (Perera et al., 2009). Sanders (2011) looks at the test scores of high school students in Texas and finds a negative relationship between *in utero* pollution and later test performance. The presence of such fetal damages further supports the hypothesis that TSP exposure can impact the fetus as well as infants. For further discussion of how negative fetal shocks (environmental and otherwise) can have lasting life effects, see Almond and Currie (2011).

Our use of the gender ratio as a measure of fetal deaths is based on the evolutionary theory that women in poor health are more likely to produce female children than male children. This hypothesis, first proposed by Trivers and Willard (1973), can be summarized as follows: carrying

[6]For greater discussion of particulate matter and health, see World Health Organization (1979), available at `http://www.inchem.org/documents/ehc/ehc/ehc008.htm`.

[7]For a brief review of findings on the potential consequences of PAH exposure, see Perera et al. (2009).

a fetus to term is costly and it is beneficial to ensure the ensuing child will produce grandchildren. Since a man can simultaneously father children with multiple women, men in good health could secure several mates, and men in poor health might secure none. For women the relationship between health and mating is less pronounced, as women in poor health can still secure mating opportunities with men in good health. If maternal health is an indicator for potential future infant health conditions, the Trivers-Willard hypothesis predicts that mothers are more likely to favor daughters when in poor health themselves, as this will maximize the mating opportunities for their children, and thus also maximize their chances of having grandchildren.

In our context, exposure to pollutants may compromise the health of the mother, sending a signal to the mother's systems that she is in poor health and that her offspring will also be born into poor health. The Trivers-Willard mechanism suggests this could lead to a lower probability of a live male birth. Such favoring could occur via male fetal loss, or shortly after conception via preventing implantation of male embryos.[8] Work in the medical and economics literature using research designs that can isolate stressors during gestation from those that occurred around the time of implantation suggests differential implantation cannot be the sole mechanism of altering gender ratios. For example, Cagnacci et al. (2004) find weight gain during pregnancy had negative impacts on the probability of bearing a male child. And Almond, Edlund, and Palme (2007) find that fallout from the Chernobyl disaster had significant negative impacts on the percentage of live births that are male for cohorts that were in their second trimester during the disaster. Nilsson (2008) finds lower alcohol prices decreased the percentage of male births among cohorts that had been conceived prior to the price decrease. Differential implantation rates by gender in response to pollution may be a contributor to gender ratio differences in the first (or potentially second) trimester. We examine pollution effects differently by trimester of gestation and find similar effects across all three trimesters. Effects are indistinguishable across trimesters, and we

[8]Research using rats has found such favoring could also take place after birth via differential food allotments (McClure, 1981).

conclude that differential implantation rates by gender in response to pollution are a small part of the total share. Henceforth, we use the term fetal death to encompass both failed implantations and post-implantation fetal deaths.

The Trivers-Willard hypothesis is consistent with a negative impact of pollution on maternal health, such as restricted oxygen intake caused by lung damage, driving male fetal loss. It may also be that male fetuses are more susceptible to toxins or chemicals that alter or hinder proper cellular development. Unfortunately, the direct mechanism through which TSPs might influence either maternal or fetal health is unknown. For the remainder of the analysis we focus on the causal relationship between higher pollution rates and fetal death but do not attempt to identify the mechanism through which this effect operates.

3 The Clean Air Act Amendments of 1970 and ambient pollution

The original Clean Air Act, enacted in 1963, established funding for the study and cleaning of air pollution. In 1967, Congress passed the Air Quality Act, which allowed the Secretary of Health, Education, and Welfare to establish "air quality regions," though air quality standards remained a responsibility of the state. By 1970 it was clear that little was being done — no state had yet established a full pollution control program (Rogers, 1990). In response, on December 31, 1970 President Richard Nixon signed the first round of Clean Air Act Amendments, which made air pollution a concern of the federal government. Around approximately the same time, the National Environmental Policy Act established the Environmental Protection Agency (EPA), which was placed in charge of enforcing the various regulations of the CAAA.

The amendments of 1970 established National Ambient Air Quality Standards (NAAQS) and placed federal limits on pollution levels for six "criteria pollutants:" carbon monoxide, nitrogen dioxide, ozone, sulfur dioxide, total suspended particulate matter, and lead. States were further divided into Air Quality Control Regions (AQCR) consisting of multiple counties. As part of its attempt to reduce ambient pollution, the federal government classified regions as being in "attain-

ment" or "nonattainment" based on regulatory caps on the various pollutants. Regions found to be in nonattainment were subject to more stringent regulation as a result — states were required to establish plant level controls, set emissions caps, and install abatement technologies (for more information on the CAAA and plant response, see Greenstone (2002)). Following CG, we assign attainment status at the county level, assuming that states placed their regulatory attention on individual counties within an AQCR when deciding how to best lower ambient air measures.

After the announcement of the NAAQS, states were required to prepare and submit State Implementation Plans (SIPs) which provided information as to how the states were planning on controlling and regulating ambient pollution. The first SIPs were due to the EPA in January, 1972. In a given year, areas were deemed to be in nonattainment for TSPs if they violated either of two conditions: (1) the annual geometric mean was greater than 75 μg/m^3, or (2) the second highest reading for the year was greater than 260 μg/m^3. We use this nonattainment status as an instrument for pollution changes within counties, where counties that received the "treatment" of being classified as nonattainment are anticipated to see greater decreases in pollution. Figure 1 shows the distribution of 1970 pollution levels for counties in our sample. The regulatory cutoff point of 75 μg/m^3 is indicated with a vertical dashed line. In our preferred specification, we examine the change in pollution levels between 1970 and 1972 to span a period prior to and just following the enactment of the CAAA. As a robustness check we also consider a slightly longer 1969-1972 difference, and a shorter 1971-1972 difference, and we cannot reject that these results are equal to each other at standard confidence levels. This is discussed further in Section 7.

4 Estimation strategy

Using panel data at the county-by-year level, we utilize a quasi-experimental strategy that exploits variation in pollution levels across counties and over time. For some county c in year t, the relationship between a county level outcome of interest y and ambient pollution can be expressed

as

$$y_{c,t} = \beta TSP_{c,t} + \delta X_{c,t} + \lambda_c + \gamma_{s,t} + \epsilon_{c,t} \tag{1}$$

where β is the coefficient of interest (the marginal impact of TSPs), $X_{c,t}$ is a vector of aggregated individual demographic covariates and county-level economic covariates, λ_c is a time-invariant county level fixed effect, $\gamma_{s,t}$ is a state-by-year fixed effect, and $\epsilon_{c,t}$ is the error term. An analog to the fixed-effects model is the "first-difference" model, where changes in y are expressed as functions of changes in TSP and other covariates. Let $\Delta y_c = y_{c,1972} - y_{c,1970}$, with similar notation for TSP, X, and ϵ. Then,

$$\Delta y_c = \beta \Delta TSP_{c,} + \delta \Delta X_c + \gamma_s + \Delta \epsilon_c. \tag{2}$$

Time-invariant factors such as λ_c have been eliminated with the difference, and the state-by-year fixed effects become simply state fixed effects. The remaining error may still have period-specific, county-level unobserved factors. This will contribute to bias in the ordinary least squares (OLS) estimates if such unobserved factors are correlated with the estimate of interest even after controlling for covariates, i.e.,

$$E[\Delta TSP_c, \Delta \epsilon_c | \Delta X_c] \neq 0. \tag{3}$$

OLS results can also suffer from measurement error, which, if classical, will bias results toward zero. Pollution is assigned at the county level, an inherently noisy measure of true individual exposure. In addition, we are considering prenatal effects, and the exact exposure timeframe is unknown.[9] Any fixed-effects type model will accentuate existing measurement error, as such models

[9]Even for live births, reported gestation length information is imprecise. We test to see if exposure calculated using daily pollution data over an estimated gestation yields different results in Section 6 and find results consistent with our main specification.

remove some true variation while doing nothing to eliminate random noise, increasing the noise-to-signal ratio.

In order to obtain unbiased estimates, we use estimated 1970 county-level attainment status as an instrument for changes in pollution, similar to Chay and Greenstone (2003a).

$$\Delta y_c = \beta \Delta \widehat{TSP}_c + \delta \Delta X_c + \gamma_s + \Delta \epsilon_c \tag{4}$$

$$\Delta TSP_c = \mathbf{1}(\text{geometric mean}_{c,70} > 75 | 2^{\text{nd}} \text{ highest}_{c,70} > 260) + \phi \Delta X_c + \gamma_s + \eta_c \tag{5}$$

where $\mathbf{1}(\bullet)$ is an indicator function equal to 1 if either condition detailed in Section 3 is true and η is the first stage error term. Data on actual attainment status in the early 1970s are unavailable. To construct our instrument we must estimate which regions were most likely to be classified as in attainment or not based on the available pollution data. We use TSP monitor data from 1970 to assign likely attainment status at the county level for 1972 (following CG), and when we discuss counties as being in attainment or nonattainment in 1972, we are referring to a status calculated using data from 1970 levels. This assumes that, in order to write their SIPs in time for the January 1972 deadline, states would have needed to use pollution information from 1970, as 1971 data were not yet available.

Figure 2 illustrates the difference in pollution reduction between counties estimated to be in attainment and those that were not. There is a general trend of declining pollution levels over the period of interest.[10] Between 1970 and 1972 air pollution in attainment counties increased slightly, while the declines in nonattainment counties were dramatic. Panel A of Table 2 shows the strength of the first stage of the 2SLS estimation numerically. Counties classified as being in nonattainment saw pollution drops that were around 17 μg/m^3 greater on average than attainment counties. This relationship is illustrated visually in Figure 3, which plots county-level 1970-1972 changes in the arithmetic mean as a function of the 1970 geometric mean and fits a locally estimated polynomial

[10] While pollution sensor data are available prior to 1968, they are available for only a very small number of counties and are omitted here.

on either side of the regulatory cutoff (indicated with a dashed vertical line). In all instrumental variables regressions that follow, we show the standard F-statistics for first stage regressions as a demonstration of the strength of the first stage.

We assume that attainment status is uncorrelated with the errors in the second stage. This correlation could be present if, for example, pollution decreases are driven by mean reversion rather than attainment status. In order to control for this potential confounder, we explore specifications that add the running variable used to calculate attainment status (the 1970 geometric mean), linearly and with higher order polynomials, as explicit controls. The remaining variation in pollution is driven entirely by the predicted discontinuous change in attainment status around the threshold specified in the 1970 CAAA, and is independent of 1970 pollution levels beyond CAAA cutoff violation. These results are similar to our main specification and are discussed in Section 7.

5 Data

To examine the effects of pollution exposure on fetal and infant death, we combine data from birth records, death records, ambient TSP measurements, and local economic indicators. As noted in Section 4, we conduct our analysis primarily on the changes that occurred between 1970-1972, though we also examined the 1969-1972 and 1971-1972 periods. In each case, we used a balanced panel of counties with data from the beginning, ending, and intermediary years for each sample.

Birth data come from the National Center for Health Statistics Vital Statistics Micro-data. Data begin in 1968, and from 1968-1972 represent a 50% sample of all birth certificates in the United States (weighted up to represent the full population of births). The data feature a large amount of information on demographic covariates such as county and day of birth, race and gender of the child, characteristics of the mother and father, and health indicators for observed births. We use county and year of birth to match birth cohorts to their relevant ambient pollution levels. In some specifications, we expand on this by using daily pollution data to calculate exposure levels based on day of birth and then again collapse data to the county-by-year level.

We limit the covariates used in our study to those that are least frequently missing in our time period. These include the child's race (white, black, and other), whether the birth was in a hospital, whether a physician was present, birth parity, and mother's age. In some specifications, as noted, we include mother's education, though this reduces the number of observed births (and counties) available for estimation. When considering joint effects on both genders, we conduct estimation at the county-by-year of birth level and weight all regressions by the number of births in each cell. When considering differential effects by gender, estimation is done at the county-by-year of birth-by-gender level and weighted accordingly. We use a similar strategy when considering differences by race, age, and education.

Infant death data, used to examine infant mortality rates, are from the full census of deaths from the National Center for Health Statistics National Vital Statistics System Multiple Cause of Death Files. Demographic variables include race, gender, and age at death.[11] We construct the neonatal infant mortality rate for year Y by dividing the number of infants born in year Y that died within 28 days by the number of live births in year Y by gender and race. The one-year mortality rate is constructed similarly. After 1982, limited fetal death data are available in the Vital Statistics Fetal Death Detail Record. In Section 8, we discuss these data as potential measure of the fetal sensitivity differences across genders.

Pollution data are from the EPA Air Quality Database. The EPA has air-monitoring stations located around the country measuring a variety of air pollutants. We focus on stations that measure TSPs (pollutant classification number 11101) using the 24-hour average. We collapse the arithmetic mean measurement of TSPs for all monitor observations by county to calculate the annual county pollution measure, using the number of valid observations as weights.[12] In order to closely approximate the regulations in the CAAA we estimate each county's attainment status using the

[11] We exclude deaths due to external causes (e.g., fractures, injuries, or adverse effects of medical agents) from our analysis. Such deaths are not causally linked with pollution levels (see Table 8 of CG).

[12] We have also explored using the geometric mean as the health-relevant pollution shock. Results are qualitatively similar and available on request.

geometric mean and second-highest daily measure from the highest reading monitor in the county of pollution data from 1970, a strategy identical to CG (see Section 4). This assumes that counties that may have been borderline nonattainment still felt pressure from the EPA to reduce pollution levels (Chay and Greenstone 2003a).[13]

Economic factors may be correlated with both air quality and fetal death rates. In order to control for such potential confounders, we use the Regional Economic Information System (REIS), provided by the Bureau of Economic Analysis to obtain county-level economic covariates. These data contain annual measures of per capita income, per capita net earnings, and several measures of total government transfer payments which we convert to per capita: total transfers, total medical transfers, public assistance medical payments, income maintenance, family assistance payments, food stamps payments, and unemployment insurance. We also control for county level employment (total employment divided by total population), employment in manufacturing (as the CAAA likely had differential impacts based on the size of the manufacturing sector), and total population. All dollar values control for inflation and are adjusted to 2009 dollars (using the 1982-1984 chained CPI from the Bureau of Labor Statistics).

Summary statistics by attainment status across our period of interest are shown in Table 1. In general, birth outcomes are similar for attainment counties and nonattainment counties over the two years of interest. The last two columns report the p-value from a regression of the variable of interest on the difference-in-difference estimate between attainment and nonattainment counties across 1970-1972. The second to last column reports the p-values from regressions using all counties. The last column reports the p-value from regressions restricted to counties with 1970 TSP levels between 60 and 90 μg/m^3. Nonattainment status was not randomly assigned, so we expect that high pollution counties will be different from low pollution counties, and they are on several measures. Nonattainment counties are generally more populous, have higher infant mortality rates,

[13]Using the average geometric mean across *all* monitors rather than the highest reading monitor yielded similar results.

and more a higher share of infants are of low birth weight. When we restrict the sample to counties that had similar levels of pollution in 1970, many of the statistically significant differences in changes in these measures disappear. The only statistically significantly different change between attainment and nonattainment counties occurred in the overall employment rate, which increased in attainment counties but decreased slightly in nonattainment counties. Given the potential link between employment and maternal and fetal health, we control directly employment in all of our preferred specifications. We also note that the decreased level of employment in nonattainment counties may result in higher maternal stress and a lower percentage of males in that birth cohort. This effect is in the opposite direction of the main effect we identify, and is unlikely to contribute to the result we find (see Section 7).

6 Results

In the absence of reliable data on fetal deaths, we must turn to alternative metrics to measure births that did not occur, but would have occurred in the absence of pollution exposure *in utero*. One potential metric is the number of total births in the population. If pollution has a positive effect on the probability of fetal death, then the decreases in pollution seen as a result of the CAAA could be associated with an increase in the average number of live births. There are, however, a number of reasons why the average birth rate may not be the correct metric to measure fetal deaths. Fertility rates may respond to a wide variety of economic factors potentially correlated with pollution levels. For example, Lindo (2010) finds that income shocks resulting from husband job displacements can both reduce total fertility and alter fertility timing, and Dehejia and Lleras-Muney (2004) show fertility responds to shifts in the business cycle. Even instrumenting for pollution will not remove all bias when using total births as a proxy for fetal deaths. Any change in the total number of births necessarily includes the health effect from the policy change as well as any accompanying fertility behavior changes.[14] Total fertility is thus the combined impact of the impact of pollution on fetal

[14]Instrumental variable estimates showed pollution levels had no identifiable effect on total births — effects were all small and statistically insignificant. A one-unit increase in TSP had an estimated effect of 0.89 additional births

health, and parental conception decisions and an inappropriate metric to examine if the outcome of interest is fetal health.

The gender ratio at birth presents an alternative measure of fetal health that has the advantage of being orthogonal to these parental conception decisions.[15] As discussed in Section 2, prior research suggests that the male fetus is more sensitive to external stressors, and thus more likely to suffer fetal death in the presence of negative health shocks. If pollution exposure has a positive impact on fetal death rates, and males are more likely to suffer fetal death, than one expected outcome of higher pollution levels is a decrease in the share of live births that are male. By considering changes in the gender ratio as they correlate to changes in ambient pollution, we can observe an indirect measure of the number of male births that did not occur but would have in the absence of air pollution exposure. This is not a precise measure of the true fetal death rate, as it does not consider any effects on females (and in fact treats the effect on females as zero). In Section 8, we expand on these findings to estimate the total fetal death effect.

A raw data comparison of the change in the fraction of live births that are male by attainment status is illustrated in Figure 4. The average change in the fraction of births that are male between 1970-1972 is larger for nonattainment counties than attainment counties. This is consistent with reductions in pollution stemming from the CAAA leading to increases in fetal health, though the graph does not control for any covariates. Panel B of Table 2 presents the reduced form numerically, allowing for the inclusion of controls to better identify the causal effect. Our preferred specification in column 4 indicates that the CAAA increased the fraction of births that are male by 0.547 percentage points. This result is significant at the 5% confidence level.

Table 3 shows the estimated relationship between the share of births that are male and ambient

per hundred thousand. Combining this zero, or slightly positive, result with our later estimates of total fetal deaths indicates frequency of intercourse may be positively correlated with pollution levels. These results are available upon request.

[15] Individuals may choose to engage in behavior that they believe impacts the gender of the child. We do not attempt to address whether such behaviors are effective or not. Unless individuals modify this behavior in response to the CAAA attainment status of their home county, such activities should have no impact on our results.

TSPs. The outcome variable is the probability of a live birth being male. Coefficients should be interpreted as percentage point changes. We weight each cell by the number of observed live births and standard errors are clustered on state. [16] Negative marginal impacts indicate higher pollution levels are correlated with a lower fraction of males among live births, which in turn suggests an increase in the fetal mortality rate among males (or a decrease in the number of conceived males, an alternative which we address in Section 8). Panel A shows OLS results, and Panel B shows IV results. Column 1 is the most sparsely specified model, while columns 2, 3, and 4 add natality controls for observed births, economic controls, and state fixed effects, respectively. Column 4 is our preferred specification.

OLS results are all negative, indicating increased ambient pollution levels decreases the share of live births that are male. After adding natality and economic controls, results are significant at 10%. Adding state fixed effects leaves the coefficients effectively unchanged but increases the standard errors enough to remove statistical significance. The coefficient in column 4 suggests that an increase in ambient pollution of 1 μg/m^3 results in an observed 0.007 percentage point decrease in the probability of a live birth being male, and a standard deviation increase in pollution (approximately 23 μg/m^3) is associated with an observed 0.20 percentage point change in the probability of a live birth being male. IV results are larger, and in our preferred specification, which is statistically significant at the 1% level, a standard deviation increase in pollution is associated with a 0.89 percentage point decrease in the probability of a live birth being male. Using the 1970 attainment county share of male births (51.28) as a baseline, this is a change of approximately 1.7%.

Measurement error may bias OLS toward zero, but it is unlikely to explain why our IV estimates are four times larger than our OLS estimates. Omitted variables bias could be influencing the OLS results if there is some omitted factor that is positively correlated with both pollution levels

[16] Results with standard errors clustered on county rather than state are negligibly different and are available on request.

and the fraction of live births that are male. For example, counties could be experiencing an economic downturn, which would cause declining pollution levels as well as economic hardship. The declining pollution level in the county would positively impact fetal health, but the economic hardship would negatively impact fetal health, and the OLS estimate of the relationship between pollution and fetal health would be an understatement of the true effect. Using attainment status as an instrument for the change in pollution will avoid this bias, provided that attainment status is independent of such confounding trends. We discuss background trends further in Section 7.

An additional possible explanation for the much larger IV estimates is that local average treatment effect (LATE) estimated by the IV specifications is much larger than the average treatment effect (ATE). OLS results use the entirety of the pollution distribution and identify the ATE. IV, however, identifies the LATE, driven by counties around the regulatory threshold that had predicted pollution decreases as a result of the CAAA and would not have otherwise. These counties near the regulatory point may have had stronger relationships between pollution and fetal deaths for a variety of reasons: effects may not be detectable at very low pollution rates, or counties with extremely high pollution rates may have other systematically confounding factors that are not controlled for using available covariates. As a possible check for OLS results being biased by such "extreme" counties, we estimate the OLS specifications using only those counties close to the cutoff, with 1970 geometric means between 60 and 90 μg/m^3 (we conduct a similar robustness check using the IV results in Section 7). In the preferred specification analogous to column 4, the result was 0.017 with a standard error of 0.018. While the result is noisy (likely due to the substantially smaller sample size around the cutoff of 165 counties), it is much closer to the IV results in magnitude, suggesting that some of the difference is caused by OLS being biased by extreme values in the 1970s TSP distribution. We also repeated the OLS analysis dropping only counties with low 1970 TSP levels (below 60 μg/m^3) and then only high 1970 TSP levels (above 90 μg/m^3). The estimated impact is larger in the case of removing the high 1970 TSP counties, further supporting that OLS results are downward biased by unobservable factors in the high TSP counties. These esults are

available in Table A-1 in the Appendix.

We next consider the effects of pollution on the gender ratio at birth by subgroups: mother's education, child's race, and mother's age. If pollution exposure impacts gender ratios though the fetal death mechanism, we expect to see larger impacts on subgroups that are more sensitive, either through lower availability of fetal damage abatement capital such as prenatal care and avoidance behavior, or because of lower baseline fitness and nonlinearities in health effects. Columns 1 and 2 of Table 4 show results for mothers with high school education or lower and greater than a high school education, respectively. There are a lower number of counties due to the lack of reliable mother's education data in the earlier natality data files. Results confirm our prior expectation — mothers with lower education levels (comprising approximately $\frac{3}{4}$ of our sample), a factor highly correlated with availability of fetal damage abatement capital, see a substantially larger impact on their gender ratios when exposed to higher pollution levels, and the result remains statistically significant at 1%. Differences in effect also appear when examining effects by race. Column 3 shows the effect for whites which, while still large and significant, is approximately $\frac{1}{3}$ the estimated effect for blacks. The National Center for Health Statistics reported that for live births in 1970, an estimated 72% of white mothers received prenatal care during the first trimester, compared to 44.2% of black mothers. 6.3% of whites either waited until the third trimester or received no prenatal care at all, compared to 16.6% of black mothers.[17] These noted differences in use of prenatal care across races further support our prior expectation — blacks have a lower use of prenatal damage abatement capital, and thus see a larger effect on fetal death. Finally, columns 5, 6, and 7 show results for mothers younger than 20, 20 to 34, and 35 years old and up, respectively. As expected, we see the greatest negative impacts of pollution for the more sensitive groups. Both younger and older mothers see larger effects, though all results are now significant at only 10% and the smallest effects are contained within two standard errors of the largest effects.

We also consider the possibility that pollution had larger effects depending on when during

[17]Table 5 on page 106 of *Health, United States, 2010* (National Center for Health Statistics, 2011).

development the fetus is exposed. Buckles and Hungerman (2008) have shown that mother's average socioeconomic status is lower during the winter. Since these seasonal differences may be related to maternal and fetal health, we begin by looking for different effects by quarter of birth. Table 5 shows effects by quarter of birth in Panel A and by exposure trimester in Panel B. Panel A consists of four separate regressions. Column 1 is limited to births that occurred between January and March, Column 2 is limited to births that occurred between April and June, and so forth. While there are positive effects across all four quarters of birth, the largest effects are for births in the third quarter of the year. Figure 5, which shows monthly TSP levels over time, may explain this finding. While pollution levels are generally declining, there is a cyclicality of pollution levels within each year. Levels are lowest during the fourth quarter and highest during the second quarter. One possible explanation for the higher third quarter effect is that the infants born in the third quarter were the only infants not exposed to the low pollution levels in the fourth quarter.

As noted in Section 5, our main specifications assign cohort fetal exposure using year of birth. We next calculate each infant's TSP exposure using daily pollution data. Using daily pollution data allows us to consider effects by trimester of exposure, but there is a tradeoff between this method and what we present for our main analysis. The advantage of this method is that each observed infant is only associated with the pollution exposure he or she was more likely to have experienced. However, note that "daily" does not mean each monitor has a reading for every day. Many monitors have pollution measures every six days, but some go weeks or months without an observation of pollution — our pollution data are a noisy measure of the true pollution level in the county. While an infant born in December may not have experienced the pollution levels measured in January of that year, January's levels may help to remove some of the noise from the measures in the subsequent quarters. The results are presented in Panel B of Table 5.

Without data on the exact date of conception, we assume each gestation was nine months in length, or would have been in the absence of a fetal death. We label the three months before birth the third trimester, the three months before that the second trimester, and the three months before

that the third trimester. We then calculate average pollution exposure over those dates. Columns 1 through 3 of Panel B in Table 5 show the results from these calculations by trimester. We cannot reject that these effects are equal (an F-test of coefficient equality yielded a p-value of 0.89), and thus can establish no support in the data for variation in exposure by trimester of gestation. Note we cannot controls for all three trimesters individually in the same regression, as we only have one available instrument. We have also tried to exploit the exact date of birth from the natality data to estimate pollution exposure for date-of-birth cohorts rather than overall year cohorts. The fourth column of Panel B shows the results when we use only the pollution data in the nine months before observed date of birth to calculate pollution exposure (rather than the average pollution level in the year of birth). The estimated 0.043 percentage point change we find when using only the pollution measurements from the nine months before birth is very close to the 0.033 we find in our main specification that uses all the pollution measurements in the calendar year of birth.

7 Further considerations

Our results suggest that there is a difference between the susceptibility of males and females to ambient pollution, both *in utero* and after birth. Further, the fetal death effects appear greatest for groups we expect to be most sensitive. Effects are largest for blacks, mothers with high school education or lower, and mothers younger than age 20 and age 35 and older. In order to better establish the validity of our results, we now consider potential confounders in our analysis such as regression to the mean in pollution and differential background trends between polluting and nonpolluting counties.

We first examine the sensitivity of our results with respect to the identifying assumptions of the discontinuity used in the first stage. Results are shown in Table 6. Panel A addresses the assumption that counties that fall short of the regulatory cutoff serve as good controls for those beyond the cutoff. This assumption becomes more likely to hold as we restrict the bandwidth around the cutoff for the counties to be used in the analysis — thus we begin by removing more

extreme counties. Columns 1-3 show how our results vary when we limit the data to counties with 1970 pollution levels within bandwidths of 35, 25, and 15 μg/m^3 on either side of the regulatory cutoff. There is a tradeoff here between the strength of assumption required for the instrument to be valid and the statistical power available to identify effects. It is more likely that attainment and nonattainment counties are similar on unobservables in this restricted sample than in the full sample we use for our main results. However, due to the smaller number of counties in the restricted sample we do not have the statistical power to precisely identify effects. The estimated impact of pollution on the share of male births remains negative for all bandwidths, and all are within two standard errors of the full sample estimate. This gives credence to the assumption that attainment status is excludable from our second stage in the full sample.

In Panel B we return to the full data set and add smooth functions of the running variable (geometric mean of pollution in year 1970) in the second stage. This check allows us to see if our identification is arising from a smooth change in pollution across the spectrum, i.e., regression to the mean in higher pollution counties over time, rather than the assumed discontinuity in pollution changes. Columns 1-3 control for linear, quadratic, and cubic forms of the running variable, adding subsequently higher order terms. As with the case of restricted bandwidth, the additional strain on the data reduces the predictive strength of the excluded variable, but results remain significant at 5% with a linear function and 10% using a quadratic. While results are no longer significant when controlling for a cubic function, the coefficient remains negative. In all three cases, the estimated effect has the same sign as and is similar in magnitude to our main effect. Based on the illustrated relationship between the geometric mean and changes in pollution seen in Figure 3, we believe the simple linear relationship is a relatively good approximation of the truth.[18]

We next investigate whether general background trends are driving our results. Nonattainment status may have been assigned to counties that already had a positive trend in the percent of each

[18]We have also allowed the running variable to have differential effects on either side of the cutoff by interacting the linear, quadratic, and cubic functions with the indicator for nonattainment. Since we lack the statistical power to identify these models the results are omitted, but are available upon request.

birth cohort that were male. To test for the existence of this confounder, ideally we would examine county-level trends in the fraction of births that were male prior to the CAAA. As noted in Section 5, detailed natality data do not exist prior to 1968. In the absence of data on births by gender and county from the pre-period we examine trends in the post-period. We repeat the analysis using the 1970-1972 pollution changes, but assign them to the controls and gender ratios of other two-year difference pairs, ranging from 1973-1975 up through 1985-1987. If a difference in trends between attainment and nonattainment counties is responsible for our findings rather than a true causal link between pollution and fetal deaths, this trend would likely continue in the years after our analysis. This would manifest as negative, statistically significant relationships between 1970-1972 pollution levels and the percentage of births that are male in subsequent years. Results are shown in Table 7. Column 1 repeats our main estimates (1970-1972), and columns 2-6 show the results using natality and economic covariate data from later two-year periods. The number of counties changes slightly across specifications due to availability of REIS data across periods. Results are not significant for any other two-year pair, and more importantly we can rule out point estimates for any of these relationships as large as the main effect, suggesting background trends are not driving our results.

We also tested to see if the choice of the two-year 1970-1972 difference is of importance. As noted in Section 3, we prefer to use this two-year window to allow for a "before" and "after" of the enactment of the CAAA. However, since there are other possible choices for a "pre" period, we have repeated our analysis using a three-year window spanning the CAAA (1969-1972) and a one-year window (1971-1972). Our results are robust to several valid choices of the before period — regardless of the window chosen, the estimates are similar, and we cannot reject equality of the estimates across year specifications (an F-test of coefficient equality yielded a p-value of 0.63).[19]

[19]Results available upon request.

7.1 Potential non-pollution impacts

There remains concern that our estimated effect is driven by some unobservable factor which remains even after utilizing an IV strategy. One such confounder is macroeconomic changes resulting from the CAAA. For example, Greenstone (2002) shows that the CAAA had substantial economic consequences, particularly for the manufacturing sector. The use of gender ratios as our measure of fetal death avoids the concern of economic factors resulting in selection into fertility. However, male fetuses are more susceptible to fetal death from stressors other than pollution, which could bias our findings if, for example, the CAAA led to job loss in nonattainment counties, which then led to decreased mother health, either though income loss or additional non-pollution stress. This would suggest that, as a byproduct of the non-pollution effects of the CAAA, the number of male births should decrease due to the increased stress levels. Similarly, if job loss as a result of the CAAA leads to lower levels of maternal nutrition, findings by Almond, Hoynes, and Schanzenbach (Forthcoming) indicate that the fraction of male births should decrease. We find the lower pollution caused by the CAAA is associated with an *increase* in the number of male births — both of these effects should exert pressure in the opposite direction of our main effect and bias our results toward zero, if at all.

A similar concern is that differences in human capital and stresses across socioeconomic status are correlated with lower male birth rates. In a cross-sectional comparison, Almond and Edlund (2007) find significant differences between gender ratios among socioeconomic groups. Specifically, single mothers with less than a high school education are 0.8% less likely to have males than married mothers with some college education. This could be problematic if the CAAA is associated with a change in the composition of mothers. Specifically, if in response to the CAAA policies, lower education mothers move out of nonattainment counties (or are more likely to avoid pregnancy), we might mistake the change in mother composition as evidence of changes in fetal death rates.[20] Looking at empirical data cannot answer this question, as the composition of moth-

[20]See Dehejia and Lleras-Muney (2004) for a discussion of motherhood composition changes and birth outcomes.

ers might change due to fetal deaths as well — the characteristics of mothers that never give birth are just as unobservable as births that never occur. However, we can place bounds on the potential bias. The reduced form result in Table 2 suggest that the CAAA led to a change in the probability of a male birth of 0.547 percentage points, which is an increase of approximately 1.1% from a baseline of 51.32. If, prior to the CAAA, every birth in nonattainment counties was to a single mother without a high school degree, and afterward every birth was to a married mother with some college, the implied change would be 0.8% — even in the most extreme case, a shift in the mother population cannot explain the entirety of our findings.

Finally, we note that the estimated effects need not necessarily be limited to pregnancies that suffered from fetal death after successful insemination. Research in the medical field has proposed that observed changes in the gender ratio in response to maternal stressors are the result of stressful situations favoring the implantation of female over male embryos (Cameron, 2004). Sperm carrying the Y chromosome that determines the male gender may be weaker than those that carry the X chromosome, or sperm carrying the Y chromosome may combine less efficiently with the egg, and maternal stress may disrupt zygote formation with "Y sperm" more than zygote formation with "X sperm" (Boklage, 2005). If pollution exposure can change the probability of a successful implantation in ways that vary across genders, or can weaken Y sperm in such a way as to reduce the relative probability of a male zygote, such changes in the male birth population would be interpreted in our findings as male fetal deaths. Our results may include within them not only fetal deaths, but avoided initial pregnancies, though effects on the sex ratio for pollution exposure in the estimated second and third trimesters suggest this is at most a small portion of the effect.

8 Discussion

The decrease in ambient pollution levels after the CAAA resulted in a decrease in male fetal deaths, as measured by changes in the probability of a live birth being male.[21] Of more interest to policy

[21] While we currently attribute our findings to changes in TSPs, other unobserved pollutants that are strongly correlated with TSPs could be included in our findings. Unfortunately, data on other pollutants are not available for our

makers, however, is the effect on fetal death for both genders. Our finding can be interpreted as a measure of total fetal deaths only if female fetuses are completely unresponsive to pollution, which is unlikely. We combine our findings, which calculate the difference in losses between males and females *in utero*, with several estimates of relative *in utero* sensitivities of males and females to estimate the total fetal losses. We use the relative causal impacts of pollution on neonatal deaths and the relative causal impacts of pollution on deaths within one year to provide a range of plausible estimates of the total *in utero* mortality effect of pollution levels. We also discuss the observed total male and female fetal deaths reported in the fetal death data between 1982 and 1989, though we note these are not causally linked to pollution.

Panels A and B of Table 8 present the causal impact of pollution on one year and neonatal mortality (death within 28 days of birth) separately for males (column 1) and females (column 2). Each of these four cells is a coefficient from a first-difference regression using changes from 1970-1972 and controlling for natality covariates, economic covariates, and state fixed effects. Female and male losses during both the one-year and neonatal periods are all positively signed, as were the results found in CG for the overall population. Consistent with our findings of differential fetal loss rates *in utero*, male live births have higher mortality than females in response to pollution shocks. Panel A estimates each additional unit of TSP leads to an additional 6.7 male neonatal deaths per 100,000 live male births and 2.0 female neonatal deaths per 100,000 live female births, an impact ratio of 3.36 to 1. Panel B presents similar findings for the one-year mortality rate: the increase in the male mortality rate is approximately 8.9 per 100,000 live male births, while females see a smaller increase of approximately 2.1 deaths per 100,000 live female births, a ratio of 4.28 to 1.

Column 4 of Table 8 shows the estimated total fetal impacts using these relative sensitivity estimates. Using neonatal mortality rates, the estimated change of 0.547 percentage points from Table 2 translates to a combined impact of 35,900 prevented fetal deaths as a result of the CAAA,

timeframe. Regardless of the pollutant, however, the reduced form estimate in Table 2 is identifying the impact of the additional CAAA regulation on fetal deaths, and is informative from the standpoint of policy evaluation.

or a change in the overall live birth rate of approximately 2.1% for nonattainment counties. Using one-year mortality rates, the total effect is estimated at around 31,200 prevented fetal deaths, which is approximately 1.8% of the total birth population in nonattainment counties.[22] As noted in Section 5, fetal death data are available beginning in 1982. These data on observed fetal deaths can also be used to construct a ratio of male to female fetal deaths, though the deaths are not causally linked to pollution. We prefer our estimates that use the causal impact of pollution on relative neonatal mortality rates as we believe those are the closest to the effects of pollution *in utero*, and are less likely to be biased by the selectivity of measured fetal deaths. However, for completeness we note that for all data available in the 1980s, a period somewhat close to our period of interest, the number of recorded fetal deaths was 133,706 males and 115,553 females, for a relative ratio of 1.16.[23]

Using our preferred relative gender susceptibilities, the above calculations translate to a one-unit drop impact of 111-128 fewer fetal deaths per 100,000 live births.[24] These effects may appear large when compared to the literature on pollution and post-natal infant deaths. A number of factors could explain this difference. It is likely that live births are more robust to stresses than a developing fetus, and abatement actions in the presence of health complications are more easily enacted with infants. For example, if air pollution causes an infant to display respiratory difficulty, the infant may be brought to a hospital, where active medical attention helps to offset the negative effects. No such effects can be easily observed with an injured fetus, and treating a fetus may be more difficult that providing medical treatment to an infant.

[22] These estimates can be obtained by noting that

$$\beta_{\text{CAAA}} = \frac{M}{M+F} - \frac{(M - maledeaths)}{(M - maledeaths) + \left(F - \frac{1}{\Omega} maledeaths\right)} \tag{6}$$

where M and F are the 885,759 male and 842,878 female births in all observed nonattainment counties in 1972 and Ω is the sensitivity of males relative to females, provided above.

[23] Using this relative fetal sensitivity yields a total estimated effect of 320,700 avoided fetal deaths, or 18.6% of the total birth population.

[24] Marginal impacts are calculated by dividing the estimated number of avoided deaths by the average TSP reduction caused by the CAAA as shown in Panel A of Table 2. This assumes a linear impact of TSPs.

It is also informative to consider our effects in the light of other studies that have found gender differences in the presence of other external stresses. Using the 1970 birth ratio for attainment counties as a baseline, our estimates put the change in the probability of a male birth at approximately 0.03% per unit of TSPs, with the total effect of the CAAA being a change of approximately 1.1%. The 2007 working paper version of Almond, Edlund, and Palme (2009) finds that exposure to the fallout from Chernobyl in Sweden resulted in a decrease in the probability of having a male of 1.6% in Sweden. Using similar identification, Peterka, Peterková, and Likovský (2004) find the radiation-induced change in the number of male births to be almost 4% for the Czech Republic. Almond, Hoynes, and Schanzenbach (Forthcoming) examine the effect of Food Stamp Program (FSP) rollout at the beginning of the third trimester in utero and find that it increases the fraction of births that are male among whites by 0.09% and blacks by 0.32%. Our effect is smaller than the effects observed for Chernobyl radioactive fallout, but larger than the maternal nutritional effects of the FSP. We are unaware of any conversion of health impacts from subclinical levels of radiation or nutritional deficiencies to the reduction in air pollution levels we examine, but we note that findings for other *in utero* health shocks are qualitatively similar to those we find for air pollution.

Finally, there is the challenge of quantifying the value of the change in gender ratios at birth. In the case of infant mortality, there are measures of the value of a statistical life that can be used to financially quantify the impacts. Fetal deaths, however, are more complicated. Some may occur without the knowledge of the mother, and one could argue the social costs of such foregone pregnancies are effectively zero, or at least no greater than the loss of births resulting from other medical factors such as impotency. Of the fetal deaths that occur in known pregnancies, the social cost should be less than that of an infant death — fewer resources have been invested, and given that the mother can become pregnant again, the largest costs may be a shifting of the pregnancy time frame and the psychological impacts. Of course, changes in fetal deaths are also indications of changes in maternal health, but converting changes in the gender ratio to the value of changes

in maternal health is difficult.[25] Given these complications, we cannot monetize our estimated impacts.

9 Conclusion

Measuring the impacts of policy on fetal health presents several challenges. Post-natal measures of fetal health are net of selection, as birth weight averages and mortality rates are only observed among those infants that survive to term. Fetal deaths themselves are rarely observed, recorded only for a selected subset of the population, and unavailable prior to 1982. Policy changes may have causal impacts on fertility choices as well as impacts on fetal health and separating the two can be difficult, making changes in total live births a potentially biased metric. Our solution to these complications is to use changes in the gender ratio of live births as a potential proxy for fetal deaths, which exploits the medical finding that male fetuses are more susceptible to death from external stresses. Such a measure has the advantage that gender determination is orthogonal to many traditional sources of fertility bias, and could be used to estimate the effects of other policy measures intended to improve maternal health and infant outcomes. The benefit of this metric is that it requires only that the researcher have information regarding, (1) the gender ratio of live births, and (2) the differential effects of the policy on a measurable post-birth outcome by gender, which can be used to extrapolate the total effect from the observed impact on male fetal losses relative to females.

We use the Clean Air Act Amendments of 1970 as an exogenous shock to ambient total suspended particulate pollution to examine the impacts of prenatal pollution exposure on the gender ratio of live births. In the absence of other confounders associated with the CAAA, a change in the gender ratio in the presence of decreased pollution can be interpreted as avoided male fetal deaths. We then scale our findings using TSP-driven neonatal and one-year infant mortality rates to approximate the impact on total fetal deaths. We find that a one-unit decrease in ambient TSP levels

[25] In the very long run, Angrist (2002) notes that excess females at marriage age lead to worse outcomes for females.

is associated with a 0.033 percentage point increase in the probability of a live birth being male, suggesting reducing pollution reduces male fetal deaths. Given the relative sensitivity of males to females in pollution-induced neonatal mortality, we estimate the total impact of a one-unit TSP reduction to be approximately 2,200 total avoided fetal deaths. This suggests that the pollution decreases seen as a result of the 1970 CAAA prevented almost 36,000 fetal deaths in nonattainment counties, a change of approximately 2.1% of overall births in those counties. As conventional estimates of the social cost of pollution include only observable infant health outcomes such as mortality and birth weight, such estimates are lower bounds of the true costs.

References

Almond, Douglas and Janet Currie. 2011. "Killing Me Softly: The Fetal Orgins Hypothesis." Mimeo.

Almond, Douglas and Lena Edlund. 2007. "Trivers-Willard at birth and one year: evidence from US natality data 1983-2001." *Proceedings of the Royal Society B: Biological Sciences* 274 (1624):2491–2496.

Almond, Douglas., Lena Edlund, Hongbin Li, and Junsen Zhang. 2007. "Long-term effects of the 1959-1961 China famine: Mainland China and Hong Kong." NBER Working Paper 13384.

Almond, Douglas, Lena Edlund, and Mårten Palme. 2007. "Chernobyl's Subclinical Legacy: Prenatal Exposure to Radioactive Fallout and School Outcomes in Sweden." NBER Working Paper 13347.

———. 2009. "Chernobyl's Subclinical Legacy: Prenatal Exposure to Radioactive Fallout and School Outcomes in Sweden*." *Quarterly Journal of Economics* 124 (4):1729–1772.

Almond, Douglas, Hilary W. Hoynes, and Diane Whitmore Schanzenbach. Forthcoming. "Inside the war on poverty: The impact of food stamps on birth outcomes." *Review of Economics and Statistics* .

Angrist, Josh. 2002. "How do Sex Ratios Affect Marriage and Labor Markets? Evidence from America's Second Generation." *Quarterly Journal of Economics* 117 (3):997–1038.

Boklage, Charles E. 2005. "The epigenetic environment: secondary sex ratio depends on differential survival in embryogenesis." *Human Reproduction* 20 (3):583.

Buckles, Kasey and Daniel M. Hungerman. 2008. "Season of birth and later outcomes: Old questions, new answers." NBER Working Paper 14573.

Cagnacci, A., A. Renzi, S. Arangino, C. Alessandrini, and A. Volpe. 2004. "Influences of maternal weight on the secondary sex ratio of human offspring." *Human Reproduction* 19 (2):442.

Cameron, Elissa. 2004. "Facultative adjustment of mammalian sex ratios in support of the Trivers-Willard hypothesis: evidence for a mechanism." *Proceedings of the Royal Society* 271 (1549):1723–1728.

Catalano, Ralph, Tim Bruckner, and Jennifer Ahern. 2010. "Male fetal loss in the US following the terrorist attacks of September 11, 2001." *BMC Public Health* 10 (273).

Catalano, Ralph, Claire E. Magerison Zilko, Katherine B. Saxton, and Tim Bruckner. 2010. "Selection in utero: A biological response to mass layoffs." *American Journal of Human Biology* 22 (3):396–400.

Catalano, Ralph A. 2003. "Sex ratios in the two Germanies: a test of the economic stress hypothesis." *Human Reproduction* 18 (9):1972.

Catalano, Ralph A., Tim Bruckner, and Kirk R. Smith. 2008. "Ambient temperature predicts sex ratios and male longevity." *Proceedings of the National Academy of Sciences* 105 (6):22–44.

Chay, Kenneth Y. and Michael Greenstone. 2003a. "Air quality, infant mortality, and the Clean Air Act of 1970." NBER Working Paper 10053.

———. 2003b. "The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession." *Quarterly Journal of Economics* 118 (3):1121–1167.

Chay, Kenneth Y., Michael Greenstone, and Carlos Dobkin. 2003. "The Clean Air Act of 1970 and adult mortality." *Journal of Risk and Uncertainty* 27 (3):279–300.

Clay, Karen, Werner Troesken, and Michael R. Haines. 2010. "Lead, Mortality, and Productivity." NBER Working Paper 16480.

Currie, Janet. 2011. "Inequality at birth: some causes and consequences." NBER Working Paper 16798.

Currie, Janet, Michael Greenstone, and Enrico Moretti. 2011. "Superfund Cleanups and Infant Health." NBER Working Paper 16844.

Currie, Janet, Eric A. Hanushek, E. Megan Kahn, Matthew Neidell, and Steven G. Rivkin. 2009. "Does Pollution Increase School Absences?" *The Review of Economics and Statistics* 91 (4):682–694.

Currie, Janet and Matthew Neidell. 2005. "Air Pollution and Infant Health: What Can We Learn From California's Recent Experience?" *Quarterly Journal of Economics* 120 (3):1003–1030.

Currie, Janet, Matthew Neidell, and Johannes F. Schmieder. 2009. "Air pollution and infant health: Lessons from New Jersey." *Journal of Health Economics* 28 (3):688–703.

Currie, Janet and Johannes F. Schmieder. 2009. "Fetal Exposures to Toxic Releases and Infant Health." *The American Economic Review* 99 (2):177–183.

Currie, Janet and Reed Walker. 2011. "Traffic Congestion and Infant Health: Evidence from E-ZPass." *American Economic Journal: Applied Economics* 3 (1):65–90.

Dehejia, Rajeev and Adriana Lleras-Muney. 2004. "Booms, Busts, and Babies' Health." *Quarterly Journal of Economics* 119 (3):1091–1130.

Fukuda, Misao, Kyomi Fukuda, Takashi Shimizu, and Henrick Møeller. 1998. "Decline in sex ratio at birth after Kobe earthquake." *Human reproduction* 13 (8):2321–2322.

Greenstone, Michael. 2002. "The impacts of environmental regulations on industrial activity: Evidence from the 1970 and 1977 clean air act amendments and the census of manufactures." *Journal of Political Economy* 110 (6):1175–1219.

Helle, Samuli, Samuli Helama, and Jukka Jokela. 2008. "Temperature-related birth sex ratio bias in historical Sami: warm years bring more sons." *Biology Letters* 4 (1):60.

Kemkes, Ariane. 2006. "Secondary sex ratio variation during stressful times: The impact of the French Revolutionary Wars on a German parish (1787–1802)." *American Journal of Human Biology* 18 (6):806–821.

Knittel, Christopher R., Douglas L. Miller, and Nicholas J. Sanders. 2009. "Caution, drivers! Children present. Traffic, pollution, and infant health." Mimeo.

Lerchl, Alexander. 1999. "Sex ratios at birth and environmental temperatures." *Naturwissenschaften* 86 (7):340–342.

Lindo, Jason M. 2010. "Are Children Really Inferior Goods? Evidence from Displacement-driven Income Shocks." *Journal of Human Resources* 45 (2):301–317.

Lleras-Muney, Adriana. 2010. "The needs of the Army: using compulsory relocation in the military to estimate the effect of air pollutants on children's health." *Journal of Human Resources* 45 (3):549.

Lyster, W. R. 1974. "Altered sex ratio after the London smog of 1952 and the Brisbane flood of 1965." *Journal of Obstetrics and Gynaecology* 81:626–631.

McClure, Polly Ann. 1981. "Sex-Biased Litter Reduction in Food-Restricted Wood Rats(Neotoma floridana)." *Science* 211 (4486):1058–1060.

Moretti, Enrico and Matthew Neidell. 2011. "Pollution, Health, and Avoidance Behavior: Evidence from the Ports of Los Angeles." *Journal of Human Resources* 46 (1):154–175.

National Center for Health Statistics. 2011. "Health, United States, 2010: With Special Feature on Death and Dying."

Neidell, Matthew. 2009. "Information, Avoidance Behavior, and Health: The Effect of Ozone on Asthma Hospitalizations." *Journal of Human Resources* 44 (2):450.

Neidell, Matthew J. 2004. "Air pollution, health, and socio-economic status: the effect of outdoor air quality on childhood asthma." *Journal of Health Economics* 23 (6):1209–1236.

Nilsson, J. Peter. 2008. "Does a Pint a Day Affect Your Child's Pay? Unintended and Permanent Consequences of a Temporary Alcohol Policy Experiment." CEMMAP Working Paper.

———. 2009. "The Long-term Effects of Early Childhood Lead Exposure: Evidence from the Phase-out of Leaded Gasoline." Mimeo.

Parker, Jennifer D., Pauline Mendola, and Tracey J. Woodruff. 2008. "Preterm birth after the Utah Valley Steel Mill closure: a natural experiment." *Epidemiology* 19 (6):820.

Perera, Frederica P., Zhigang Li, Robin Whyatt, Lori Hoepner, Shuang Wang, David Camann, and Virginia Rauh. 2009. "Prenatal Airborne Polycyclic Aromatic Hydrocarbon Exposure and Child IQ at Age 5 Years." *Pediatrics* 124 (2):e195–e202.

Peterka, Miroslav, Reneta Peterková, and Z. Likovský, Z. 2004. "Chernobyl: prenatal loss of four hundred male fetuses in the Czech Republic." *Reproductive Toxicology* 18 (1):75–79.

———. 2007. "Chernobyl: Relationship between the number of missing newborn boys and the level of radiation in the Czech regions." *Environmental Health Perspectives* 115 (12):1801.

Reyes, Jessica Wolpaw. 2007. "Environmental Policy as Social Policy? The Impact of Childhood Lead Exposure on Crime." *The BE Journal of Economic Analysis & Policy* 7 (1):51.

Ritz, Beate and Fei Yu. 1999. "The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993." *Environmental Health Perspectives* 107 (1):17.

Rogers, Paul G. 1990. "The Clean Air Act of 1970." EPA Journal, available online at `http://www.epa.gov/history/topics/caa70/11.htm`.

Sanders, Nicholas J. 2011. "What Doesn't Kill you Makes you Weaker: Prenatal Pollution Exposure and Educational Outcomes." Mimeo.

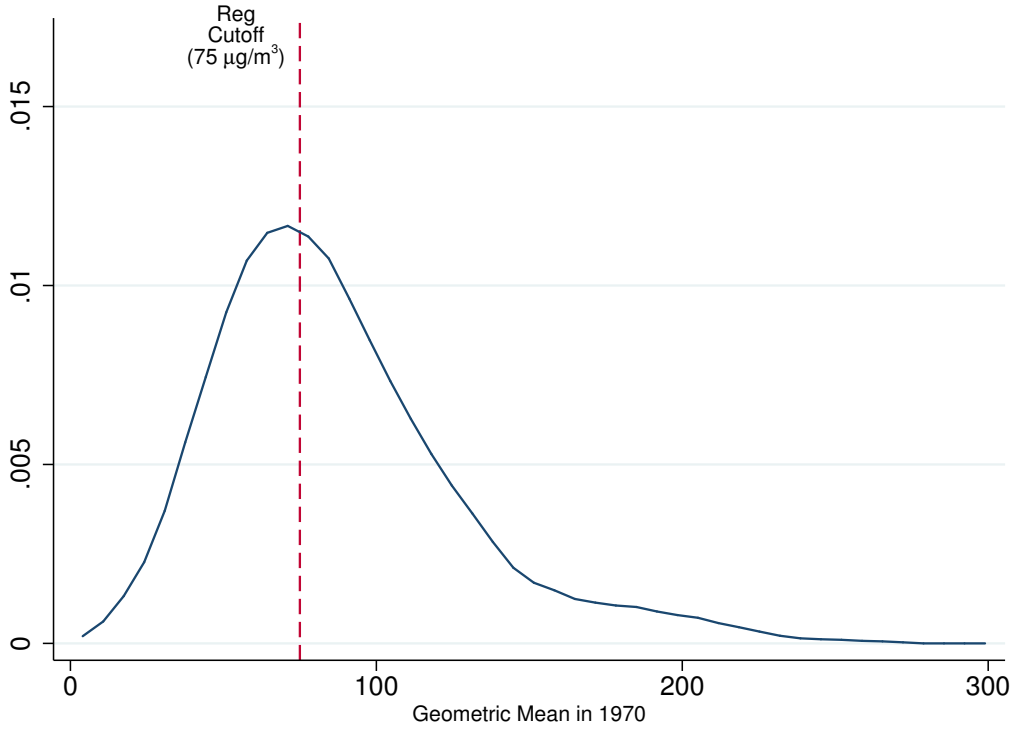
Trivers, Robert L. and Dan E. Willard. 1973. "Natural selection of parental ability to vary the sex ratio of offspring." *Science* 179 (4068):90.

Wang, Xiaobin, Hui Ding, Louise Ryan, and Xiping Xu. 1997. "Association between air pollution and low birth weight: a community-based study." *Environmental Health Perspectives* 105 (5):514.

World Health Organization. 1979. "Sulfur oxides and suspended particulate matter."

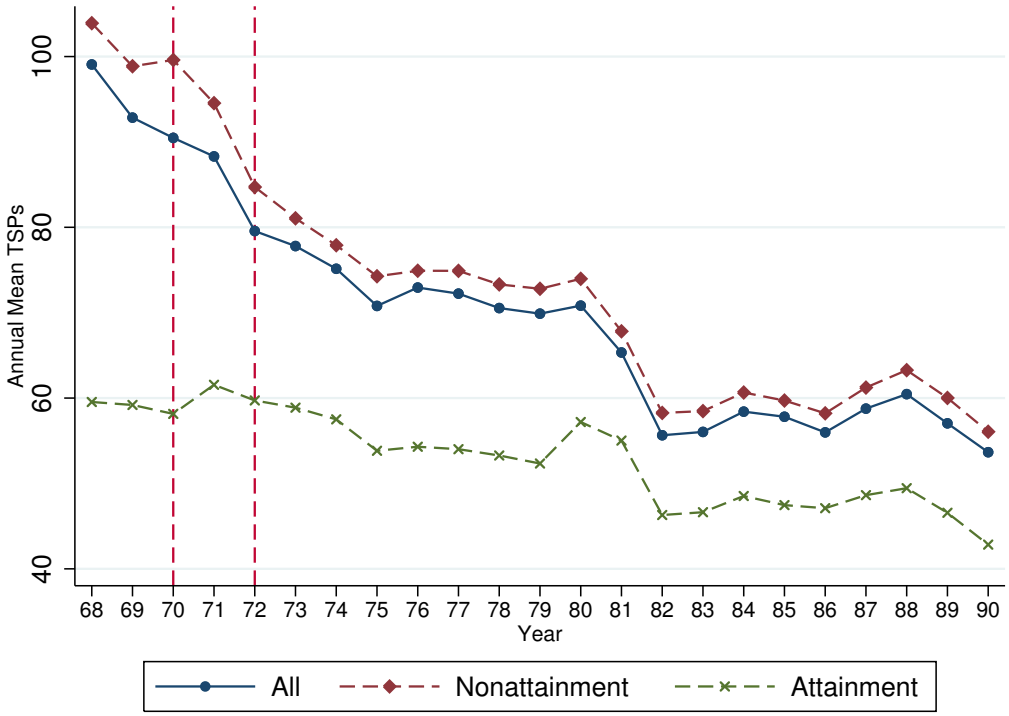
10 FIGURES

Figure 1
Density of 1970 TSP Levels



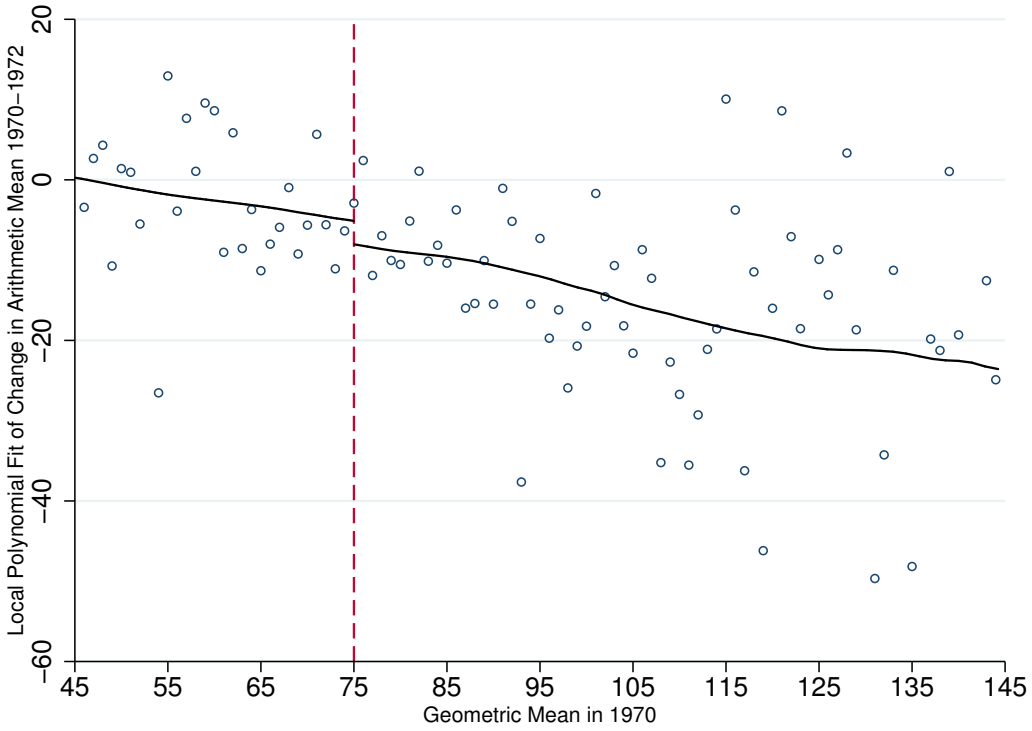
Notes: Kernel density is calculated with a bandwidth of 10 μg/m^3. County level geometric mean levels are calculated as discussed in Section 4.

Figure 2
Trends in TSPs by Estimated 1970's Attainment Status



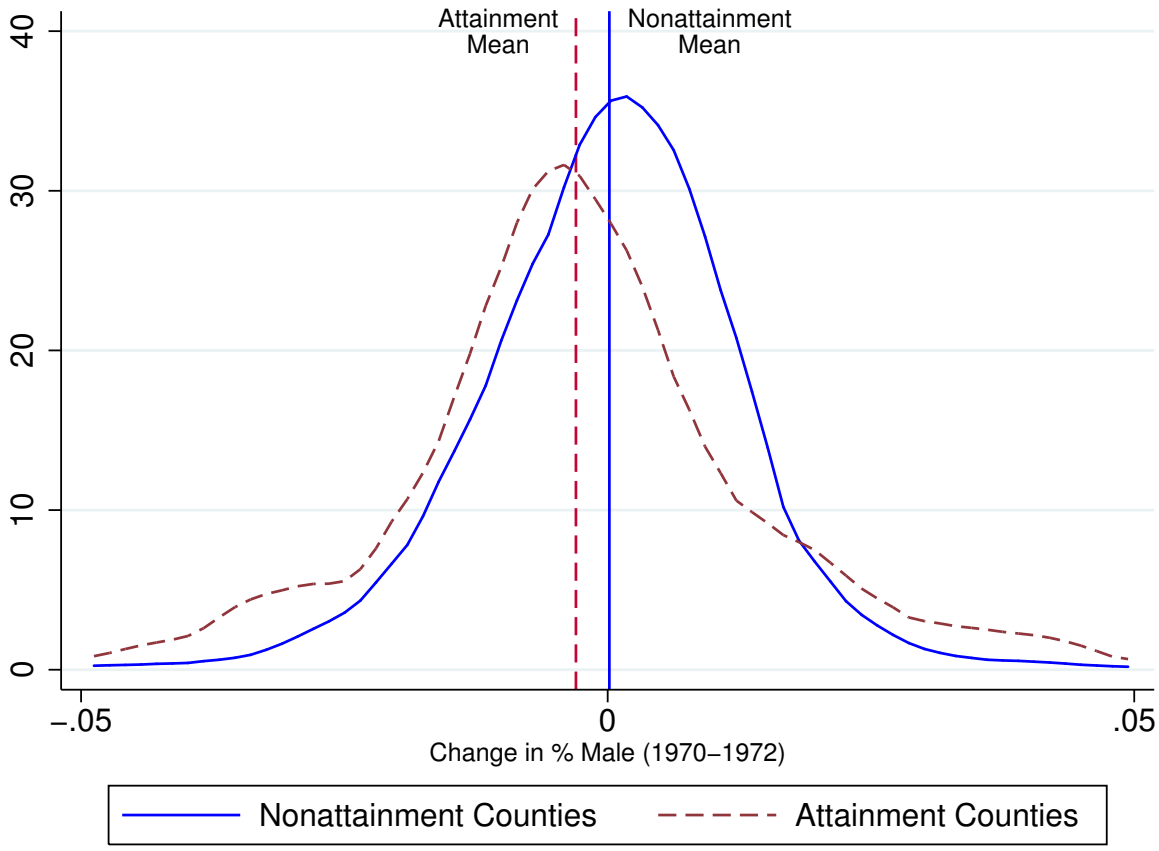
Notes: Yearly values are calculated using pollution data for the 457 counties in the primary analysis. Determination of attainment status is discussed in Section 4.

Figure 3
Local Polynomial Estimation of Changes in Arithmetic Mean by 1970 TSP Level



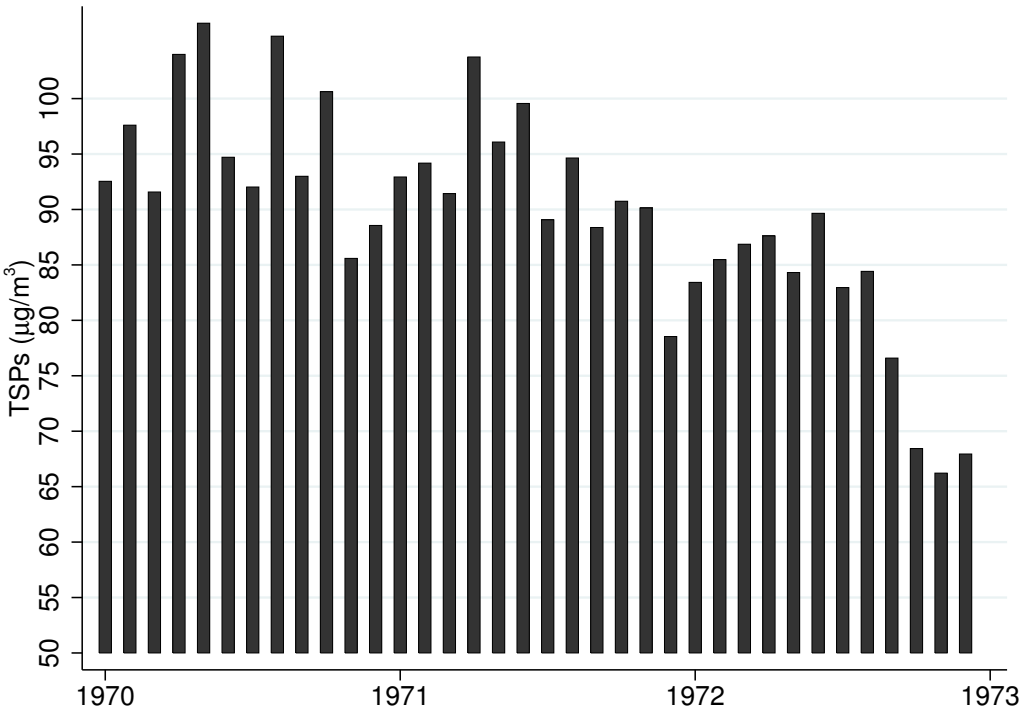
Notes: County level geometric mean levels are calculated as discussed in Section 4. Blountsville County (FIPS number 35301 with a ΔTSP of -68.9) is omitted from the scatter plot to retain scale but included in the local linear estimations.

Figure 4
Changes in Probability of a Live Birth Being Male Between 1970 and 1972



Notes: Kernel density is calculated with a bandwidth of 0.5 percentage points.

Figure 5
TSP Levels by Month 1970-1972



11 TABLES

Table 1
Comparing Change in Covariates from 1970-1972 by Attainment Status

	Means				p-value for Difference in Changes (95%)	
	Attain		Nonattain			
	1970	1972	1970	1972	All	Restricted
Variables of Interest						
Number of Births	509,028	440,947	1,750,584	1,480,411	0.000	0.000
Infant Mortality (one-year)	1,524	1,392	1,756	1,598	0.030	0.638
TSPs	57	55	94	78	0.000	0.001
% Low Birth Weight	0.078	0.075	0.082	0.079	0.065	0.587
% Very Low Birth Weight	0.012	0.011	0.012	0.013	0.008	0.148
% Male	0.513	0.510	0.513	0.513	0.084	0.216
Natality Controls						
Second Child	0.282	0.302	0.275	0.295	0.795	0.663
Third Child or Higher	0.325	0.287	0.331	0.295	0.488	0.686
White	0.834	0.820	0.808	0.790	0.249	0.713
Black	0.126	0.134	0.178	0.192	0.096	0.329
Born out of Hospital	0.003	0.005	0.003	0.005	0.920	0.331
Physician Present	0.998	0.997	0.998	0.997	0.614	0.438
Mothers Age	24.794	24.677	24.690	24.573	0.429	0.334
Economic Controls						
Employment Rate	0.428	0.433	0.496	0.494	0.644	0.018
Manufacturing Rate	0.077	0.073	0.114	0.107	0.056	0.543
Per Capita Income	23,196	24,546	24,567	26,036	0.323	0.062
Per Capita Net Earnings	17,867	18,717	19,144	20,129	0.421	0.062
Population	538,546	525,972	1,525,895	1,499,970	0.878	0.287
Per Capita Unemployment Insurance	124	164	126	158	0.346	0.182
Per Capita Total Income Transfers	2,113	2,484	2,048	2,444	0.558	0.338
Per Capita Public Medical Assistance	192	252	169	229	0.836	0.307
Per Capita Medical Transfers	408	488	377	459	0.883	0.270
Per Capita Income Maintenance Payments	305	354	289	374	0.022	0.815
Per Capita Food Stamp Payments	28	47	28	50	0.330	0.563
Per Capita Family Assistance Payments	157	186	156	213	0.010	0.244

Notes: Observations are at the county level and weighted by the number of births in the county-year. TSP measurements are from the EPA Air Quality Database. Natality and mortality data are from the Vital Statistics of the United States. Economic data are from the Regional Economic Information System. Dollar values are in 2009 terms. Differences in the final column are restricted to a sample of counties with 1970 geometric mean TSP levels between 60 and 90 μg/m^3. Standard errors for tests of differences are clustered at the state level.

Table 2
The Impact of CAAA Nonattainment Status on Ambient TSPs and the Probability of Live Births Being Male

	(1)	(2)	(3)	(4)
Panel A: First Stage — Change in Mean TSPs from 1970-1972				
Classified Nonattainment	-15.286***	-14.597***	-14.620***	-16.594***
	(3.227)	(3.333)	(2.392)	(2.123)
Panel B: Reduced Form — Impact of Nonattainment on Probability of a Live Birth Being Male				
Classified Nonattainment	0.317*	0.321*	0.471**	0.547**
	(0.180)	(0.188)	(0.194)	(0.212)
Natality Controls	N	Y	Y	Y
REIS Controls	N	N	Y	Y
State Fixed Effects	N	N	N	Y
Counties	457	457	457	457

Data are described in Section 5. Regressions are done at the county-year level and are weighted by the number of live births. Estimated standard errors, clustered by state, are shown in parentheses. Coefficients in Panel B indicate percentage point changes.
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 3
Probability of a Live Birth Being Male

	(1)	(2)	(3)	(4)
Panel A: OLS				
Mean TSPs	-0.005	-0.005	-0.007*	-0.007
	(0.004)	(0.004)	(0.004)	(0.005)
Impact of 1 st dev	-0.15	-0.13	-0.20	-0.20
Panel B: IV				
Mean TSPs	-0.021*	-0.022	-0.032***	-0.033***
	(0.012)	(0.013)	(0.011)	(0.010)
First Stage F	22.43	19.18	37.68	61.11
Impact of 1 st dev	-0.56	-0.60	-0.87	-0.89
Natality Controls	N	Y	Y	Y
REIS Controls	N	N	Y	Y
State Fixed Effects	N	N	N	Y
Counties	457	457	457	457

Notes: Data are described in Section 5. Outcome variable is the probability of a live birth being male. Coefficients indicate percentage point changes. Regressions are done at the county-year level and are weighted by number of births. Instrumental variables estimates of the effect of TSP on total births use the first stage estimates shown in Table 2.
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 4
IV Results for Probability of a Live Birth Being Male by Subgroups

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Mother Education		Child Race		Mother Age		
	$\leq$ HS	$>$ HS	White	Black	$\leq$ 19	$>$ 19 & $<$ 35	$\geq$ 35
Mean TSPs	-0.059***	-0.002	-0.032***	-0.092*	-0.066*	-0.021*	-0.074*
	(0.018)	(0.034)	(0.011)	(0.051)	(0.035)	(0.011)	(0.042)
First Stage F	52.78	33.96	89.81	14.52	55.83	61.93	107.74
Counties	436	436	457	398	457	457	456
Baseline	48,663	48,849	48,705	48,818	48,395	48,746	48,678
Total Births	2,005,362	642,265	3,364,999	718,240	732,232	3,197,191	246,921

Notes: Data are described in Section 5. Outcome variable is the probability of a live birth being male. Coefficients indicate percentage point changes. Regressions are done at the county-year-subgroup level and are weighted by number of births. Instrumental variables estimates of the effect of TSP on total births use the first stage estimates shown in Table 2. Baseline values refer to the value in attainment counties in our sample in 1970.
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 5
Timing of Effects by Quarter of Birth and Trimester

	(1)	(2)	(3)	(4)
Panel A: Effect by Quarter of Birth				
Quarter	1	2	3	4
Mean TSPs	-0.02	-0.028	-0.056***	-0.014
	(0.028)	(0.028)	(0.021)	(0.018)
Counties	422	437	457	457
Panel B: Effect by Trimester of Exposure				
Trimester	1	2	3	1,2,and 3 (jointly)
Mean TSPs	-0.055**	-0.045**	-0.037***	-0.043***
	(0.027)	(0.019)	(0.012)	(0.016)
Counties	457	457	456	456

Notes: Data are described in Section 5. Outcome variable is the probability of a live birth being male. Coefficients indicate percentage point changes. Regressions are done at the county-year level and are weighted by number of births. See Section 6 for a discussion of how pollution exposure was calculated using daily pollution data. Instrumental variables estimates of the effect of TSP on total births use the first stage estimates shown in Table 2.
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 6
IV Estimates Using Limited Bandwidth and Running Variables

	(1)	(2)	(3)
Panel A: Restricted bandwidth			
	40-110	50-100	60-90
Mean TSPs	-0.035*	-0.016	-0.039
	(0.020)	(0.035)	(0.065)
First Stage F	40.09	11.16	5.17
Counties	318	256	165
Panel B: Inclusion of running variable (no interaction)			
	Linear	Quadratic	Cubic
Mean TSPs	-0.056**	-0.057*	-0.066
	(0.023)	(0.030)	(0.091)
First Stage F	13.37	11.73	4.59
Counties	457	457	457

Notes: Data are described in Section 5. Outcome variable is the probability of a live birth being male. Coefficients indicate percentage point changes. Regressions are done at the county-year level and are weighted by number of births. Instrumental variables estimates of the effect of TSP on total births use the first stage estimates shown in Table 2.
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 7
Repeated IV Results Assigning 70-72 Pollution to Various Year-Pairs

	(1)	(2)	(3)	(4)	(5)	(6)
	70-72	73-75	76-78	79-81	82-84	85-87
Mean TSPs	-0.032***	-0.003	-0.008	-0.005	-0.009	-0.008
	(0.010)	(0.010)	(0.010)	(0.011)	(0.007)	(0.006)
First Stage F	62.42	44.84	60.89	62.31	87.12	43.86
Counties	457	454	455	457	457	457

Notes: Data are described in Section 5. Outcome variable is the probability of a live birth being male. Coefficients indicate percentage point changes. Regressions are done at the county-year level and are weighted by number of births. Column 1 repeats the result from column 4 of Table 3, all other columns use 1970-1972 pollution changes but all other data from indicated two-year difference span (see Section 7). Instrumental variables estimates of the effect of TSP on total births use the first stage estimates shown in Table 2.
* significant at 10%; ** significant at 5%; *** significant at 1%

Table 8
Estimated Impact on Total Fetal Deaths - Conversion Metrics Using IV Estimates of the Impact of Pollution on Infant Death

(1) Male Deaths	(2) Female Deaths	(3) Relative Sensitivity	(4) Impact of CAAA
Panel A: Neonatal Mortality Rate			
6.645**	1.978	3.36	35,860
(3.029)	(2.664)		
Panel B: One-Year Mortality Rate			
8.855**	2.071	4.28	31,151
(3.633)	(3.038)		

Notes: Data are described in Section 5. The regression estimates (Columns 1 and 2 of Panels A and B) show the impact of a one-unit change in TSPs on the probability of a post-natal death. Regressions are done at the county-year level using 457 counties, are weighted by number of births, and control for natality and economic covariates as well as state fixed effects as in column 4 of Table 3. Responses to the CAAA use the estimates from Table 2. Calculations are detailed in Section 8.
* significant at 10%; ** significant at 5%; *** significant at 1%

A APPENDIX

Table A-1
OLS Using Various Cutoffs

	All Counties	$60 \leq TSP \leq 90$	$TSP \leq 90$	$60 \leq TSP$
Mean TSPs	-0.007	-0.017	-0.012	-0.006
	(0.005)	(0.018)	(0.012)	(0.006)
Counties	457	165	282	340

Notes: Data are described in Section 5. Outcome variable is the probability of a live birth being male. Coefficients indicate percentage point changes. Regressions are done at the county-year level and are weighted by number of births.
* significant at 10%; ** significant at 5%; *** significant at 1%