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Where have all the young men gone? Using sex ratios to measure fetal death rates☆

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ABSTRACT

Fetal health is an important consideration in policy formation. Unfortunately, a complete census of fetal deaths, an important measure of overall fetal health, is infeasible, and available data are selectively observed. We consider this issue in the context of the Clean Air Act Amendments of 1970 (CAAA), one of the largest and most influential environmental regulations in the history of the United States. We discuss a model of potential bias in measuring observed fetal deaths, and present the sex ratio of live births as an alternative fetal health endpoint, taking advantage of the finding that males are more vulnerable to side effects of maternal stress *in utero*. We find the CAAA caused substantial improvements in fetal health, in addition to previously identified reductions in post-natal mortality.

1. Introduction

Improvements in air quality have led to observed improvements in health outcomes such as birth weight and infant mortality. As noted by Chay and Greenstone (2003b) and Currie (2011), improvements in these observable birth characteristics are downward biased estimates of true health gains since they cannot account for fetal selection *in utero*. This selection effect is potentially quite large. A report from the Centers for Disease Control and Prevention noted observed fetal deaths accounted for approximately 60% of perinatal deaths in the United States in 1989. Greater knowledge of fetal health effects would clearly be useful in effective policy construction to better understand the true benefits of health improvements.

We explore how the Clean Air Act Amendments of 1970 (CAAA), the first large-scale air pollution regulation in the United States, improved fetal health and averted fetal losses. As part of our analysis, we build a simple model to explain why analysis using observed fetal deaths likely yields incorrect results, and discuss the use of the sex ratio in live births as a metric to assess changes in fetal and maternal health by taking advantage of differences in fetal sensitivity to health shocks across the sexes. Our premise is based on earlier findings that males *in utero* are on average more sensitive to negative health shocks than females, and improvements in fetal health should result in (relatively) more averted males fetal losses, thus shifting the ratio of live births that are male (Kraemer, 2000).[1] We then use observable sex differences in pollution-driven neonatal and infant mortality rates as an estimate of relative sex sensitivity, and convert the sex ratio changes into an estimate of total fetal losses averted by the CAAA.

The sex ratio in live births, unlike total fertility, is largely orthogonal to other choice-based fertility factors correlated with health

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[1] One cannot use total birth rates as an effective measure of fetal losses in policy analysis: fertility choices or other behavioral decisions could change in response to interventions.

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stressors, providing a less biased measure of fetal losses. This does not make it a panacea for the problem of fetal health estimation. Relationships between the sex of live births and socioeconomic status make cross sectional analysis of pollution and sex ratios difficult. Almond and Edlund (2007), Currie (2011), and Norberg (2004) showed a higher male/female sex ratio in two-parent households, suggesting a link between family structure and sex of live births—such effects, combined with how selection into pregnancy might change with factors correlated with pollution (Dehejia and Lleras-Muney, 2004) suggest potential bias in cross-sectional analysis of sex ratios in response to treatments.

We therefore demonstrate this metric with panel data, using the CAAA as an exogenous driver of ambient TSP levels. We first present results as a reduced form measure of the effects of the policy on fetal selection, and then expand to an instrumental variable analysis using CAAA attainment status as an instrument for annual pollution levels. Chay and Greenstone (2003a) and Chay et al. (2003) first used the CAAA to help identify the impact of TSPs on infant and adult health. The policy meant a dramatic decrease in pollutant levels in a modern setting, and an exogenous policy shock that cleanly defines the shift in pollution. In addition, the CAAA occurred at a time when ultrasound technology use in pregnancy was in early stages, which lessens the concern of sex-selective pregnancy termination. This is important given the findings of Ananat et al. (2007) that abortion led to a permanent decrease in fertility for women with childbearing years in the early 1970s, which further invalidates the use of total fertility as an outcome of interest. Finally, the reduced form estimate of the impact of the CAAA on fetal health is an important question, given the substantial costs associated with the program and future consideration of other environmental legislation.

We present two estimation strategies to identify the effects of the CAAA on fetal health. Similar to Chay and Greenstone (2003a) (hereafter CG) we use CAAA regulation as a driving force for changes in ambient pollution levels during the early years of the policy—counties with pollution levels above a regulatory cutoff faced greater pollution reduction requirements. We begin with a first difference design most similar to CG, using regression discontinuity to estimate the reduced form effect the CAAA. We next expand our analysis to include more years of data and move to fixed effects estimation, using county-level attainment status in the CAAA as an instrument for pollution changes across the beginning of the policy. In both models, we show statistically and economically significant links between the implementation of the CAAA, ambient TSP levels, and the fraction of live births that are male. Our preferred model shows the CAAA caused a 0.47 percentage point increase in the probability of a live birth being male, a change of approximately 0.9% of baseline levels. This fetal health impact is largest for particularly vulnerable maternal subgroups, such as non-high school graduates, single mothers, and very young mothers.

As an expansion, we use our fixed effects model to explore converting an observed change in the sex ratio to a measure of fetal losses the CAAA prevented. We first estimate post-natal sex differences in susceptibility to pollution exposure by examining infant mortality rates by sex. Using this conversion, we find the CAAA meant greater reductions in fetal losses than reductions in infant losses, indicating substantial health improvements due to reduced air pollution not currently quantified in the economics literature. By our estimates, the CAAA prevented 9900 fetal losses.[2]

Our result contributes to information on fetal survival differences by sex in response to external shocks. To our knowledge, Sanders and Stoecker (2011) was the first paper to use a quasi-experimental, panel data design to consider the differential sex impacts caused by a common air pollutant. Prior research focused largely on rare, one-time events (Lyster, 1974; Fukuda et al., 1998; Almond et al., 2007, 2007; Peterka et al., 2007; Catalano, 2003; Kemkes, 2006; Catalano et al., 2010). Other work examined more frequently experienced shocks including temperature (Lerchl, 1999; Catalano et al., 2008; Helle et al., 2008), alcohol consumption (Nilsson, 2008), and job loss (Catalano et al., 2010). Shifts in sex ratios can have important implications not just as indicators of maternal health, but also in consideration of future social movements such as marriage markets (Angrist, 2002), returns to human capital investments, and even crime rates (Edlund et al., 2007).

We organize the remainder of this paper as follows. Section 2 provides background on the regulatory policy of the CAAA. Section 3 describes the data used in the analysis. Section 4 builds a model of observed fetal deaths and discusses why the use of fetal death data likely yields biased results when analyzing fetal health policies. Section 5 outlines our identification strategies. Section 6 describes our main results. Section 7 places our findings in context with prior work. Section 8 concludes. Our Appendix includes an extensive discussion of scientific evidence of sex differences in susceptibility to external stress, as well as differences in baseline sex ratios across various subgroups.

2. The Clean Air Act Amendments of 1970 and ambient pollution

On December 31, 1970 President Richard Nixon signed the first round of Clean Air Act Amendments which required states to prepare and submit plans for regulating ambient pollution by January, 1972. To reduce ambient pollution, the federal government classified regions as being in "attainment" or "nonattainment" based on regulatory caps on various pollutants. Regions 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 Goodman, 2002). Following CG, we assign attainment status at the county level, assuming states placed regulatory attention on individual counties within a regulatory region when deciding how to best lower ambient air measures.

In a given year, areas were in nonattainment for TSPs if they violated either of two conditions: (1) the annual geometric mean was greater than 75 $\mu g/m^3$, or (2) the second highest reading for the year was greater than 260 $\mu g/m^3$. We use nonattainment status as an instrument for pollution changes within counties, where counties that received the "treatment" of being in nonattainment see greater decreases in pollution due to regulatory pressure. Data on actual attainment status in the early 1970s are unavailable, and to construct our instrument we estimate regions most likely to be in attainment or not based on the available pollution data. Following CG, we use TSP monitor data from 1970 to assign attainment status at the county level for 1972 (when we discuss counties as being in attainment or nonattainment in 1972, we refer to status calculated using data from 1970 levels). This assumes that, in order to write their implementation plan in time for the January 1972 deadline, states would have needed to use pollution information from 1970, as 1971 data were not yet available.

[2] 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 seen today. In other currently industrializing countries, particulate levels are currently as high as they were during our period of analysis, if not higher.

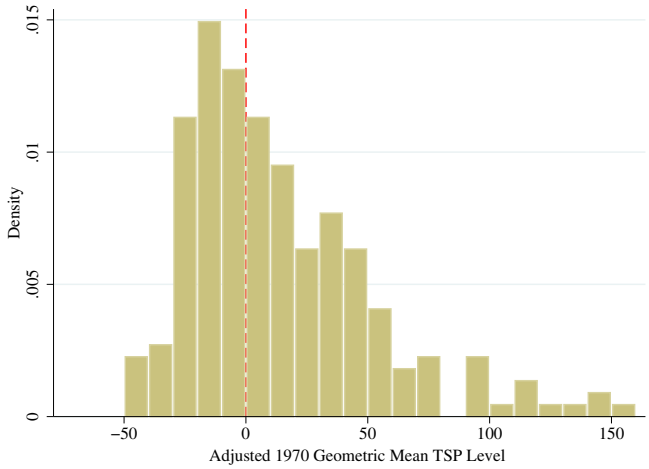

Fig. 1. Histogram of 1970 TSP levels. *Notes*: Section 5 describes the construction of county level geometric mean levels. 1970 pollution levels adjusted to equal zero at the regulatory cutoff of 75 micrograms per cubic meter.

Fig. 1 is a histogram of the distribution of 1970 pollution levels for counties in our sample using a bin width of 10 $\mu g/m^3$. We adjust 1970 pollution levels to reflect the distance from the regulatory cutoff point of 75 $\mu g/m^3$, indicated with a vertical dashed line. We examine the change in pollution levels between 1970 and 1973 to span a period prior to and just following the enactment of the CAAA, and focus on counties close to the regulatory cutoff of 75 $\mu g/m^3$. Specifically, we limit the following analysis to those within 60 $\mu g/m^3$ on either side to maximize similarity on unobservables across treated and untreated counties.

3. Data

Birth data come from the National Center for Health Statistics Vital Statistics Micro-data. County by gender records begin in 1968, and from 1968 to 1972 represent a 50% sample of all birth certificates in the United States (weighted up to represent the full population of births). We use county of maternal residence and year of birth to match birth cohorts to their relevant ambient pollution levels. 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, mother's age, and mother's education. Given that a number of observations are missing maternal education, we also include and indicator for missing for this variable. We conduct most estimation at the county-by-year of birth level and weight all regressions by baseline population in 1970—we are interested in changes in surviving birth rates, which in turn could change population after the policy, and thus use weights from before the beginning of the CAAA.[3] Given fetal survival determines observed characteristics of mothers, the infant and maternal observables may change with the policy. To avoid confounding these composition effects with health effects we control for baseline 1970 maternal characteristics interacted with linear time trends. Results using time varying observed mother and infant covariates are quantitatively similar.

Incidental to calculating total loss effects, we expand on the infant mortality analysis in CG and examine post-natal mortality by sex. Infant death data 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, sex, and age at death.[4] 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*. We construct the one-year mortality rate similarly. After 1982, limited micro-level fetal death data by sex are available in the Vital Statistics Fetal Death Detail Record. In Section 4, we discuss complications in using these data as potential measure of the fetal sensitivity differences across sexes. While nationally available fetal death data exist prior to 1982, they are aggregated to the race/county level and do not allow for sex comparisons and, as discussed below, suffer from a variety of possible sources of bias.

Pollution data are from the EPA Air Quality Database. We use the reported 24-hour average TSP level and collapse this day-by-station measurement to the year-by-county level using the number of observations as weights.[5] To closely approximate the regulations in the CAAA we estimate each county's attainment status using the 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 5). Since many monitors take readings only once per year during the winter, we limit analysis to monitors with at least 26 readings per year—the number of readings per year required to obtain a balanced distribution of samples across all 12 months.[6]

To control for economic confounders correlated with both air quality and fetal death rates, we use county-level data from the Regional Economic Information System (REIS), provided by the Bureau of Economic Analysis. 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) and employment in manufacturing (as the CAAA likely had differential impacts based on the size of the manufacturing sector). As with natality covariates, the policy could potentially shift economic covariates, and unless otherwise noted we control for 1970 baseline levels interacted with a linear time trend. We adjust all dollar values to 2009 dollars to control for inflation.

Weather is a potential confounder, as atmospheric conditions are linked to both pollution and fetal health outcomes (see, for example, Goodman et al. (2004) and Deschênes et al. (2009)). To control for such effects, we include information on temperature and precipitation at the county-year level. Specifically, we control for the annual average of daily maximum temperature and a quadratic in annual precipitation. Weather data are from Schlenker and Roberts (2009).

After combining all data sets and balancing on pollution data and economic covariates, there are 221 counties spanning 35 states, which represent almost 50% of all live births during this period.[7] We further restrict data to counties within 60 $\mu g/m^3$ on

[3] In prior versions of this paper, we weighted by the number of live births. Results are similar and available in Sanders and Stoecker (2011).

[4] We exclude deaths due to external causes (e.g., fractures, injuries, or adverse effects of medical agents) from our analysis.

[5] We have also explored using the geometric mean as the health-relevant pollution shock. Results are qualitatively similar.

[6] Results using all available monitors were noisier but quantitatively similar.

[7] Pollution was not regularly monitored in most counties. We drop states that do not have at least two counties with reporting pollution data to allow for the use of state-by-year effects. We drop two additional counties that were classified as nonattainment due not to exceeding the 75 $\mu g/m^3$ regulatory cutoff, but to the exceeding the second highest maximum allowable value (see Section 5), as these

Table 1
Comparing change in covariates from pre-treatment (1970–1971) and post-treatment (1972–1973) by attainment status.

	(1) Pre-regulation	(2)	(3)	(4) Post-regulation	(5)	(6)
	Attain	Nonattain	*p*-value	Attain	Nonattain	*p*-value
Variables of interest						
Number of births	394,721	1,074,614		338,909	901,171	
Infant mortality (one-year)	1422	1659	0.04	602	722	0.04
TSPs	74	99	0.00	70	83	0.08
% Low birth weight	0.074	0.080	0.14	0.072	0.079	0.10
% Very low birth weight	0.011	0.012	0.08	0.010	0.013	0.10
% Male	0.514	0.512	0.02	0.512	0.513	0.08
Natality controls						
Second child	0.292	0.285	0.08	0.318	0.306	0.03
Third child or higher	0.324	0.319	0.51	0.282	0.282	0.95
Single mother	0.084	0.101	0.39	0.092	0.114	0.36
Mother did not graduate high school	0.241	0.250	0.70	0.256	0.255	0.98
White	0.869	0.807	0.06	0.858	0.792	0.07
Black	0.114	0.174	0.08	0.121	0.185	0.09
Born out of hospital	0.004	0.003	0.06	0.004	0.004	0.29
Physician present	0.998	0.999	0.10	0.998	0.999	0.11
Mother's age	25.10	24.80	0.10	25.10	24.80	0.05
Economic controls						
Employment rate	0.411	0.520	0.07	0.424	0.537	0.06
Manufacturing rate	0.082	0.111	0.01	0.082	0.111	0.00
Per capita income	24,789	26,167	0.22	26,493	28,075	0.20
Per capita net earnings	19,107	20,012	0.29	20,281	21,359	0.28
Population	826,619	1,876,929	0.20	840,740	1,874,127	0.20
Per capita unemployment insurance	169	179	0.69	161	156	0.78
Per capita total income transfers	2252	2364	0.58	2610	2750	0.55
Per capita public medical assistance	228	235	0.91	310	310	1.00
Per capita medical transfers	453	472	0.79	566	583	0.86
Per capita income maintenance payments	333	375	0.60	357	428	0.42
Per capita food stamp payments	29	32	0.61	37	45	0.28
Per capita family assistance payments	190	210	0.68	204	247	0.43
Number of counties				98	100	

Notes: Pre-regulation includes the years 1970 and 1971. Post-regulation includes the years 1972 and 1973. Observations are at the county level and weighted by population in 1970. 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. Infant deaths are expressed per 100,000 live births. Dollar values are in 2009 terms. We cluster standard errors at the commute-zone level. Sample restricted to counties within 60 $\mu g/m^3$ of the regulatory cutoff, which occurs at 75 $\mu g/m^3$.

either side of the regulatory cutoff (15–135 $\mu g/m^3$ total), leaving 198 counties. Results are robust to the inclusion of all counties with data in all years from 1970 to 1973, though inclusion of counties with pollution levels further from the regulatory cutoff makes it less likely control counties are similar in unobservables to treatment counties. Table 1 shows summary statistics by attainment status across our period of analysis. Columns 1 and 2 show means for attainment and nonattainment counties in the period covering 1970–1971, before the policy had regulatory "bite". Column 3 reports the *p*-value from a test of differences between attainment and nonattainment counties in this period.[8] The government did not randomly assign nonattainment status, and high pollution counties may fundamentally differ from low pollution counties.[9] Nonattainment counties are more populous, have higher infant mortality rates, and have a higher share of low birth weight infants. However, treatment and control counties are similar on many observable (and potentially confounding) metrics. The only economic covariates that are statistically different from each other at standard levels are percentage employment and share of population employment in manufacturing.

Columns 4 and 5 report means for attainment and nonattainment counties, respectively, during the two years after the policy regulation (1972–1973). Column 5 shows *p*-values of tests for differences across the two groups after the CAAA. Economic covariates remain statistically similar, and many previously statistically significant differences are no longer significant after the CAAA. Specifically, treated and non-treated counties have converged on both pollution levels and percentage of births that are male—gaps in employment and manufacturing remained constant.

Table 1 addresses the validity of our regression discontinuity design, in that treatment and control groups have similar mean covariate levels. In Table 2, we explore the difference in pre-trends across treatment and control groups, which addresses the validity of our difference-in-difference design. Columns 1 and 2 show the inverse hyperbolic sine (IHS) of covariates in attainment counties in 1970 and 1971, respectively. Column 3 is the difference between these values (the IHS has properties similar to the natural log, in that one can interpret changes as percentage changes in the variable of interest, but can handle 0 values). Columns 4, 5, and 6 follow the same pattern, but for nonattainment counties. Finally, Column 7 shows the *p*-value from a two-tailed test of difference between Column 3 and Column 6, weighted by 1970 population and clustered by commute zone as we do in our primary regressions.

Ideally, treatment and control groups have similar pre-trends, and we would find no statistically significant differences between

groups fail our attainment status RD definition. Results were robust to inclusion of both groups.

[8] As with our regressions, we weight these tests using 1970 population levels and cluster standard errors on commuting zones.

[9] Given prior findings on government assistance programs and live birth sex ratios (Almond and Hoynes, 2011), we control for such factors in our regressions.

Table 2
Difference in trends before and after CAAA by TSP attainment status.

	(1) Attainment	(2)	(3)	(4) Nonattainment	(5)	(6)	(7)
	1970	1971	Change	1970	1971	Change	*p*-value
Variables of interest							
Infant mortality (one-year)	7.88	7.82	−0.06	8.09	8.01	−0.08	0.66
TSPs	4.88	5.01	0.13	5.25	5.27	0.03	0.32
% Low birth weight	0.73	0.73	0.00	0.74	0.73	0.00	0.35
% Very low birth weight	0.70	0.70	0.00	0.70	0.70	0.00	0.60
% Male	4.64	4.64	0.00	4.64	4.64	0.00	0.31
Natality controls							
Second child	0.85	1.01	0.16	0.84	0.99	0.15	0.01
Third child or higher	0.87	1.05	0.18	0.87	1.05	0.18	0.49
Single mother	0.74	0.78	0.05	0.75	0.80	0.05	0.37
Mother did not graduate high school	0.83	0.96	0.14	0.83	0.97	0.14	0.89
White	1.16	1.56	0.40	1.13	1.51	0.38	0.05
Black	0.75	0.81	0.06	0.78	0.87	0.09	0.09
Born out of hospital	0.70	0.70	0.00	0.69	0.70	0.00	0.17
Physician present	1.23	1.66	0.43	1.23	1.66	0.43	0.15
Mother's age	3.94	3.94	0.00	3.93	3.93	0.00	0.39
Economic controls							
Employment rate	0.91	1.14	0.22	0.97	1.24	0.26	0.04
Manufacturing rate	0.74	0.78	0.05	0.75	0.81	0.06	0.01
Per capita income	10.79	11.48	0.69	10.84	11.54	0.69	0.15
Per capita net earnings	10.53	11.23	0.69	10.58	11.28	0.69	0.14
Population	13.85	13.86	0.02	14.48	14.49	0.01	0.03
Per capita unemployment insurance	5.34	6.03	0.69	5.50	6.19	0.69	0.27
Per capita total income transfers	8.29	8.99	0.69	8.36	9.06	0.69	0.33
Per capita public medical assistance	5.42	6.11	0.68	5.71	6.40	0.69	0.33
Per capita medical transfers	6.58	7.27	0.69	6.67	7.37	0.69	0.30
Per capita income maintenance payments	6.15	6.84	0.69	6.32	7.01	0.69	0.24
Per capita food stamp payments	3.14	3.69	0.55	3.36	3.94	0.57	0.61
Per capita family assistance payments	5.49	6.18	0.69	5.67	6.36	0.69	0.30

Notes: Each variable is expressed as an inverse hyperbolic sine. *p*-values for the difference in the changes from 1970 to 1971 between attainment and nonattainment counties are given in the last column. Observations are at the county level and weighted by population in 1970. 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. Infant deaths are expressed per 100,000 live births. Dollar values are in 2009 terms. We cluster standard errors at the commute-zone level. Sample restricted to counties within 60 $\mu g/m^3$ of the regulatory cutoff, which occurs at 75 $\mu g/m^3$.

Columns 3 and 6. Among natality values, probability of being a second child and probability of a birth being white both show statistically different trends, though the economic differences are small (1 percent and 2 percent, respectively). In economic covariates, the changes in employment rate, manufacturing rate, and population are statistically different. Again, differences are economically small (1 percent for population and manufacturing employment, and 4 percent for overall employment), and all three are trending in the same direction in both treatment and control counties.

4. Potential bias in observed fetal deaths

While fetal conditions are an important consideration in health policy formation, lack of microdata on fetal deaths prior to 1988 makes it difficult to analyze how earlier policies changed fetal health. Better fetal death data exist today, but even microdata are an unreliable measure of true fetal health, as fetal deaths are selectively observed. While infant mortality will often result in a death certificate, and thus a record of occurrence, many fetal losses will not. Certain groups, those with health insurance, for example, are more likely to visit a hospital in the event of a fetal complication, while others might not seek medical attention, resulting in no record of the event. This means the probability of a fetal death being observed will correlate with a wide number of complicating factors, some of which are observable (health insurance, education, marital status, race, proximity to medical care) and some of which are not (motivation, baseline health, risk aversion).

In addition, United States reporting requirements classify a fetal death as death at 20 or more weeks of gestation. Medical definitions classify losses prior to 20 weeks as miscarriages instead of recorded fetal losses. Even if all occurrences classified as fetal deaths were (correctly) observed, such a number would still be an underestimate of changes in fetal health, as it would fail to account for losses before 20 weeks. Given the majority of fetal losses/miscarriages occur within the first trimester (Miller et al., 1980), this would be ignoring a large portion of any effect. If we stratify our sample by estimated trimester of exposure, we find suggestive evidence effects are largest in magnitude for first trimester, further complicating direct observation using fetal death microdata (see Sanders and Stoecker, 2011).

The situation is more problematic in the case where a shock moves fetal losses from one side of the 20 week classification mark to the other. A negative health shock might give the impression of a decrease in fetal deaths, which would be mistakenly interpreted as an increase in fetal health overall. This is an extension of the complication when considering infant mortality as a health endpoint if a negative shock causes some pregnancies that would have died in the first year to instead never reach full term.

The reporting bias inherent in a threshold can work in the either direction, making bias present in fetal death data impossible to sign. Any positive health shock, for example, could simultaneously move unavoidable losses into the recorded fetal death period (increasing reported fetal deaths) and prevent fetal deaths from occurring (decreasing reported fetal deaths).[10] Thus, the use of the sex ratio as a proxy for measuring fetal health is important not just because

[10] A positive maternal health shock (S) could increase gestation length (T) long

fetal death data are often unavailable (as in the history of the United States as well as the current state of many developing countries), but also because, even when available, and even when on the micro level, analysis using such data fall subject to measurement error and selection bias that is not only of uncertain size, but also of uncertain sign.[11]

5. Estimation strategy

We utilize two quasi-experimental strategies that exploit variation in pollution levels across counties and over time: a regression discontinuity design and a fixed effects difference-in-difference. For county c in year t, the relationship between a county level outcome of interest y and ambient pollution is

$$y_{c,t} = \alpha + \beta TSP_{c,t} + \delta X_c * t + \iota W_{c,t} + \lambda_c + \gamma_{s,t} + \epsilon_{c,t} \quad (1)$$

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 from 1970, which we interact with linear time trends, $W_{c,t}$ is a vector of county-year level weather 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 $\Delta y_c = y_{c,1972} - y_{c,1971}$ (the years just before and just following official CAAA regulation), with similar notation for TSP, X, W, and ϵ. Then,

$$\Delta y_c = \beta \Delta TSP_c + \delta \Delta X_c + \iota \Delta W_c + \gamma_s + \Delta \epsilon_c. \quad (2)$$

This difference eliminates time-invariant factors such as λ_c, and state-by-year fixed effects become state fixed effects in a specification that has one observation per period change. In both cases, the error term may still have period-specific, county-level unobserved factors, which will contribute to bias if such unobserved factors correlate with the estimate of interest even after controlling for covariates (as well as fixed effects), i.e.,

$$E[\Delta TSP_c, \Delta \epsilon | \Delta X, \Delta W, \gamma] \neq 0. \quad (3)$$

OLS results for TSPs 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 consider prenatal effects, where the exact exposure timeframe is unknown.[12] Any fixed-effects type model will accentuate existing measurement error, as such models remove some true variation while doing nothing to eliminate random noise, increasing the noise-to-signal ratio. To avoid such bias, we focus on the period around the CAAA and use the large shift in ambient TSPs as our primary source of variation with a focus on the reduced form impact of the policy.

In our first model, we perform a formal RD using the CAAA in a first difference framework, similar to the work done by CG

$$\Delta y_c = \kappa + \zeta \mathbf{1}(\cdot) + \pi \mathbf{1}(\cdot) TSP_{1970} + \tau(1 - \mathbf{1}(\cdot)) TSP_{1970} + \phi \Delta X_c + \kappa \Delta W_c + \gamma_s + \Delta \eta_c \quad (4)$$

where η is the error term and TSP_{1970} is normalized to zero at the regulatory threshold. $\mathbf{1}(\cdot)$ is an indicator function equal to one if the calculated county geometric mean surpassed the nonattainment threshold during the first years of the CAAA, namely:

$$\mathbf{1}(\cdot) = \begin{cases} 1 & \text{if (geometric } mean_{c,70} > 75) \\ 0 & \text{otherwise} \end{cases} \quad (5)$$

We use a standard first difference across 1971–1972, and the use of differences in variables means this model has one observation per county. To test for secular trends in pollution reduction and fetal health across pollution levels before the CAAA, we include a direct control for 1970 TSP levels. This matches a standard RD—the inclusion of the variable that determines treatment, or the "running variable" verifies the presence of a discontinuity rather than general pollution trend. Our main specification lets the slope vary on either side of the cutoff to allow the relationship between initial pollution level and subsequent change to be different for counties with initially high or low pollution levels.[13]

In our second model, we expand on (1) using a fixed effects IV, where we instrument for ambient TSPs using a combination of county-level attainment status and the beginning of the CAAA, similar to CG.

Attainment status provides a source of exogenous variation in ambient pollution shifts, but is fixed across the time period: we obtain variation by interacting attainment with an indicator for the beginning of official regulation (1972)

$$\begin{aligned} y_{c,t} &= \alpha + \beta \widehat{TSP_{c,t}} + \delta X_{c,1970} * t + \iota W_{c,t} + \lambda_c + \gamma_{s,t} + \epsilon_{c,t} \\ TSP_{c,t} &= \kappa + \mathbf{1}(\cdot) Xpost + \phi \Delta X_{c,1970} * t + \kappa W_{c,t} + \lambda_c + \gamma_{s,t} + \eta_c \end{aligned} \quad (6)$$

The underlying assumption in model (6) is that attainment status is orthogonal to other confounders, which we argue is valid around counties close to regulatory cutoff—counties with similar early pollution levels may face very different attainment status, and thus see different movements in pollution beginning in 1972.[14]

The test for differences in means in Table 1 shows no statistically significant differences in economic observables between treatment and control counties before the policy (beyond employment and manufacturing).[15] There is balance in the natality controls as well. There are statistically significant differences in infant mortality, and the share of births that are male, with a difference in very-low birth weight that is statistically significant at 8%. These differences

enough that a previously reported fetal death survives until birth (T_b) resulting in a decrease of the number of reported fetal deaths:

$$\frac{dPr[T > T_b]}{dS} > 0.$$

However, the same positive shock could increase gestation long enough that a previously unreported fetal death survives until the reporting threshold (T_r) increasing the number of reported fetal deaths:

$$\frac{dPr[T > T_r]}{dS} > 0.$$

Without the ability to measure the magnitude of these two effects, the sign of the bias is unknown.

[11] When using available macro fetal death data as our outcome in analysis of the CAAA, we obtain noisy and counter-intuitive results, in that we find the substantial decreases in pollution increased fetal deaths. These results are omitted here, but available upon request.

[12] 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 Sanders and Stoecker (2011) and find results consistent with our main specification.

[13] Sanders and Stoecker (2011) provides variations on this specification, including higher order controls and different bandwidth sample selections.

[14] Given we estimate attainment status based on 1970 pollution levels (see Section 3), there is the potential for measurement error in the instrument. Ignoring covariates, the use of a binary instrument is equivalent to the Wald estimator, thus error can either bias results upward or downward, depending on error in treatment status. If, for example, we incorrectly assign untreated (treated) regions with large pollution changes to the treated (untreated) group, this will incorrectly raise (lower) the denominator of the Wald estimator and bias our results downward (upward).

[15] These tests use observed values, not trend-adjusted baseline values as used in our regressions.

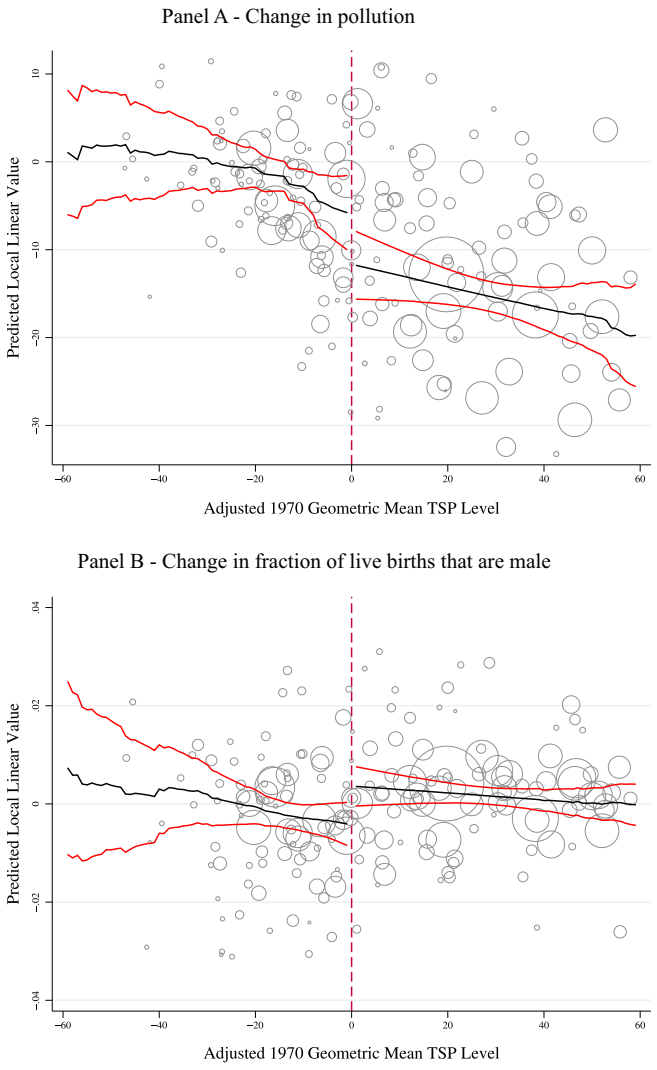

Fig. 2. Local linear estimation of changes between pre-treatment (1970–1971) and post-treatment (1972–1973) arithmetic mean and probability of a live birth being male by 1970 TSP level. *Notes*: Graphs use pollution data for the 198 counties in the primary analysis (within 60 $\mu g/m^3$ of the regulatory cutoff). Local linear estimation smoothed with a bandwidth of 60 $\mu g/m^3$ controls for state fixed effects as well as an indicator for treatment and the running variable of 1970 pollution levels interacted with treatment. We calculate pollution and male birth changes by separating data into the 1970 and 1971 period ("pre-treatment") and the 1972 and 1973 period ("post-treatment"), and comparing collapsed mean values across periods. Section 5 describes how we calculate county level geometric mean levels. Scatter plot does not show counties with outcomes above the 95th percentile and below the 5th percentile for ease of reading, though we include them in all local linear regressions.

align with our prior that more polluted counties (eventually in non-attainment) have lower baseline fetal health outcomes.

As a test of how attainment shifted outcomes, Panel A of Fig. 2 plots by-county pollution change from our "pre" period (1970 and 1971) and our "post" period (1972 and 1973) against 1970 pollution level. We scale scatter points (one for each county) by 1970 population, and overlay a (population-weighted) local-linear regression with a bandwith of 60 $\mu g/m^3$ to illustrate shifts around the regulatory cutoff. Panel B repeats the process, but using by-county changes in the fraction of births that are male between the same period. The graphs jointly illustrate a detectably greater decrease (increase) in pollution (male births) across the beginning of the CAAA that correlates with attainment status.

6. Results

We focus our results discussion on pollution, sex ratios at birth, and fetal losses, but note we base our identification design on the use of the CAAA as an instrument, which in turn requires assumptions about attainment status and correlation with unobservables. Use of the CAAA is not the main contribution of this paper, as prior economics literature has used it for research on infant health: see Sanders and Stoecker (2011) for detailed exploration of the CAAA as a valid instrument. We limit discussion here to the basic relationship between CAAA nonattainment and pollution reductions. We also note that while the EPA only measured TSPs at the time, the CAAA led to a substantial decrease in a number of other pollutants strongly correlated with TSPs as well, such as carbon monoxide. The benefit of the reduced form is that we need not worry about the impact of individual pollutants, but rather the policy as a whole. As we focus on TSPs in the IV, we interpret our results within the context of substantial decreases in correlated pollutants as well.

The probability that a live birth is male, our measure of changes in the sex ratio, provides an indirect measure of avoided fetal losses. Differences in fetal sensitivity suggest gains in fetal health should improve male outcomes relatively more than female outcomes, thus increasing the share of live births that are male. This presents an alternative measure of fetal health that has the advantage of being orthogonal to these parental conception decisions.[16] This also avoids the problem of selectively observed fetal death data as modeled in Section 4. While helpful, it is not a precise measure of changes in the true fetal loss rate. As a relative measure, it provides a lower bound of true health improvements, as the CAAA likely caused avoided female fetal losses as well. Section 7 considers a possible metric for expanding our findings and discusses methods to estimate the total fetal loss effect.

Panel A of Fig. 3 illustrates raw pollution levels separately by estimated attainment status across time. There is a general trend of declining pollution levels over the period of interest in both attainment and nonattainment counties, but in the two years following the passage of the CAAA air pollution in attainment counties increased slightly, while declines in nonattainment counties were dramatic. Panel B, which shows pollution relative to 1960 levels, more clearly illustrates the effect. Changes in both county groups move together before and after the 1970–1973 period, but differ drastically within the timeframe of our analysis. After the CAAA, pollution levels as a percentage of 1960 levels are even lower in nonattainment counties than attainment counties (though remain higher in raw values).

Fig. 4 illustrates a raw comparison of the change in the distribution of live births that are male before and after the beginning of the CAAA. The change in the fraction of births that are male between our pre- and post-treatment periods is more positive for nonattainment counties than attainment counties—changes for attainment counties are mildly negative (see Appendix for discussion of overall sex ratio changes across time in the United States). This is consistent with reductions in pollution stemming from the CAAA leading to increases in fetal health, though the figure does not control for any covariates.

Table 3 shows results from our RD first difference model. We weight regressions by baseline 1970 population and cluster standard errors on commute zone to account for common economic

[16] Individuals may choose to engage in behavior that they believe impacts the sex 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 the findings for this particular application.

Table 3
First differences: OLS estimate of the effect of TSPs and Clean Air Act attainment status on the probability of a live birth being male.

	(1)	(2)	(3)	(4)	(5)
Panel A: OLS estimate of Δ ambient TSPs on prob. of live birth being male					
Δ ambient TSPs	−0.009	−0.008	−0.008	−0.012*	−0.012
	(0.006)	(0.006)	(0.006)	(0.007)	(0.007)
Panel B: RD and nonattainment on prob. of live birth being male					
Classified	0.749***	0.993*	0.946	0.738	0.855*
nonattainment	(0.264)	(0.571)	(0.637)	(0.447)	(0.492)
Weather controls	×	×	×	×	×
State effects	×	×	×	×	×
Running variable—Constant slope		×	×	×	×
Running variable—Varied slope			×	×	×
Natality controls				×	×
REIS controls					×
Counties	198	198	198	198	198

Notes: Section 3 describes the data. Regressions are at the county-year level and weighted by population in 1970. We cluster standard errors at the commute-zone level. Sample restricted to counties within 60 μg/m^3 of the regulatory cutoff, which occurs at 75 μg/m^3.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

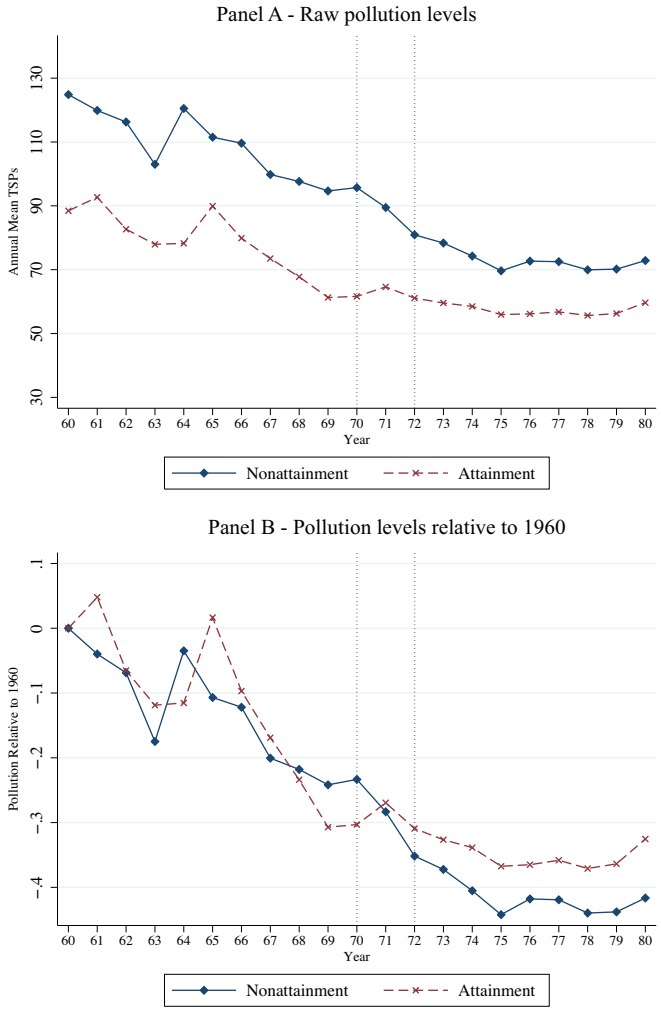

Fig. 3. Trends in TSPs by estimated 1970's attainment status. *Notes*: Yearly values use pollution data for the 198 counties in the primary analysis (within 60 μg/m^3 of the regulatory cutoff). Section 5 discusses determination of attainment status. Dotted lines indicate passage of the CAAA (1970) and commencement of official regulation (1972).

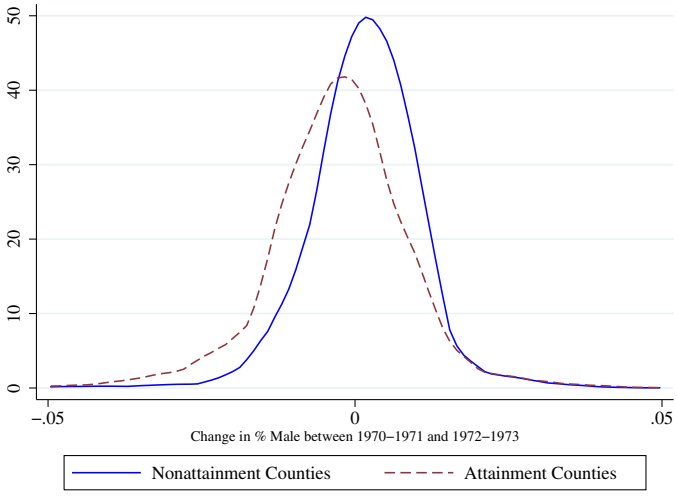

Fig. 4. Changes in probability of a live birth being male between pre- and post-CAAA regulation. *Notes*: We calculate pollution and male birth changes by separating our data into the 1970 and 1971 period ("pre-treatment") and the 1972 and 1973 period ("post-treatment"), and comparing mean values across periods. We calculate the kernel density with a bandwidth of 0.5 percentage points.

and health shocks.[17] Panel A of Table 3 shows the OLS first difference for 1971–1972. Column 1 controls for weather effects and state fixed effects (equivalent to state-by-year effects in the fixed effects model). Column 2 adds a linear control for the running variable. Column 3 allows the slope of the running variable to vary on either side of the regulatory cutoff.[18] Column 4 adds time-adjusted natality controls: baseline natality characteristics from 1970, which we interact with linear time trends. Column 5 adds similarly time-adjusted economic covariates. In all cases, OLS results are negative, suggesting higher TSP levels damage fetal health. Effects on the probability of a male birth range from −0.08 to −0.12 percentage points per unit of TSPs, though are sufficiently noisy to be statistically significant in only one case, and then at only 10%.

[17] Results are robust to clustering at the state level (Sanders and Stoecker, 2011).
[18] We view this as preferable to higher-order, common slope models, though Sanders and Stoecker (2011) details results for higher-order, more flexible versions of the running variable.

Table 4
Fixed effects: OLS estimate of the effect of TSPs and Clean Air Act attainment status on the probability of a live birth being male.

	(1)	(2)	(3)	(4)
Panel A: Estimate of ambient TSPs on prob. of birth being male				
Ambient TSPs	−0.001	−0.004	−0.004	−0.004
	(0.003)	(0.004)	(0.004)	(0.004)
Panel B: Diff-in-diff and nonattainment on prob. of birth being male				
Classified	0.388**	0.328*	0.424**	0.469
nonattainment	(0.158)	(0.173)	(0.178)	(0.204)
Weather controls	×	×	×	×
County effects	×	×	×	×
Year effects	×			
State-by-year effects		×	×	×
Natality controls			×	×
REIS controls				×
Counties	198	198	198	198
Observations	792	792	792	792

Notes: Section 3 describes the data. Regressions are at the county-year level and weighted by population in 1970. We cluster standard errors at the commute-zone level. Sample restricted to counties within 60 $\mu g/m^3$ of the regulatory cutoff, which occurs at 75 $\mu g/m^3$. "Post" indicates years 1972 and onward. Natality and economic controls use 1970 levels interacted with linear time trends (see Section 3).
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Panel B shows the reduced form relationship between regulatory status and the probability of a live birth being male, following the same column layout. The reduced form measures the overall effect of the policy, and has the benefit of avoiding the problem of measurement error in county-level pollution. As the policy caused a decrease in ambient TSPs, the consistently positive relationship further supports the OLS finding that ambient TSPs reduce fetal health. Our most saturated model shows an increase in the probability of a male birth of 0.86 percentage points after the CAAA, which is significant at 10%.

Both first difference measures suggest a link between decreased ambient TSPs and increases in the number of live births that are male, which in turn suggests an increase in overall fetal health. We next expand our analysis to include additional years of data, spanning from 1970 to 1973, by moving to our fixed effects model. Panel A of Table 4 shows OLS estimates of the effect of ambient TSPs, where marginal effects are again changes in the probability of a live birth being male. Column 1 controls for only county fixed effects, year fixed effects, and weather. Column 2 adds state-by-year fixed effects to flexibly account for differential state-level fixed effects across time. Column 3 adds time-adjusted natality controls. Column 4 adds time-adjusted economic covariates. As with the first difference model, in each case we find a negative but statistically insignificant impact of TSPs on male births, ranging from −0.04 to −0.01 percentage points.

Panel B shows the reduced form impact of the CAAA in the fixed effects framework. We find a statistically significant and positive relationship between nonattainment status, the beginning of the regulatory period, and the probability of a live birth being male. Effects range from 0.33 to 0.47 percentage points. This represents an increase of 0.6–0.9% in the overall share of live births that are male.[19] For measures of both the marginal impact of TSPs and the reduced form of the CAAA as a whole, our fixed effects results are smaller than the first difference. Given the potential sensitivity of year-to-year fluctuations in sex ratios and the limited number of counties, we favor the fixed effects model that covers a larger timeframe and takes advantage of more birth data.

Comparing Column 5 in Panel B of Table 3 to Column 4 in Panel B of Table 4, our RD estimates are approximately 1.8 times the size of estimates in our difference-in-difference model. We hypothesize a number of possibilities for the different values. First, the RD model is noisy, and our difference-in-difference results fall within one standard error if the RD estimate. Second, the RD model uses only two years of data, 1971 and 1972, making it more sensitive to potentially outlier years. Third, the RD design estimates the local average treatment effect, drawing the majority of information from counties just around the regulatory cutoff, while the difference-in-difference model estimates the average treatment effect across all counties equally.

We next move to an IV estimate of the marginal effect of an additional unit of ambient TSPs on the probability a live birth is male, using our fixed effects model. Table 5 reports both the first and second stage of our IV regressions. Panel A shows the effect of nonattainment on pollution at the county level after regulation in the CAAA. In all specifications, we find a statistically and economically significant negative effect of nonattainment status, with relative decreases in ambient TSPs between 12 and 16 $\mu g/m^3$ starting in 1972. Column 4, which accounts for weather, county and state-by-year fixed effects, and time-adjusted natality and economic controls, suggests a 12 $\mu g/m^3$ decrease.

Panel B shows IV estimates of the relationship between ambient TSPs and the percentage of male births, along with *F*-statistics for the strength of the first stage. Results are consistently negative and range from 0.029 to 0.043 percentage points change in the probability of a live birth being male per unit of TSPs. Marginal effects are statistically significant at a minimum of 10% in all specifications. Our first stage *F*-statistic is above 10 for all combinations of controls.

Like the reduced form, IV estimates help avoid the problem of classical measurement error, which may explain the discrepancy between OLS and IV marginal effects. Similarly, we note the local average treatment effect estimated by the IV specifications may be much larger than the average treatment effect. Omitted variables bias could influence OLS results if there is an omitted factor that is positively correlated with changes in both pollution levels and the fraction of live births that are male. For example, counties could experience economic downturns, 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

[19] We calculate the percentage effect using the change of 0.47 percentage points divided by the 1970 percentage of live births that are male in attainment counties (51.42%).

Table 5
Fixed effects: IV estimate of the effect of TSPs on the probability of a live birth being male.

	(1)	(2)	(3)	(4)
Panel A: Diff-in-diff estimate of nonattainment on ambient TSPs				
Classified nonattainment	−13.387*** (2.801)	−12.712*** (2.367)	−12.763*** (2.486)	−11.567*** (2.773)
Panel B: IV estimate of ambient TSPs on prob. of live birth being male				
Ambient TSPs	−0.029*** (0.011)	−0.026** (0.011)	−0.033*** (0.012)	−0.041** (0.017)
First stage *F*-stat	22.85	28.85	26.35	17.4
Weather controls	×	×	×	×
County effects	×	×	×	×
Year effects	×			
State-by-year effects		×	×	
Natality controls			×	×
REIS controls				×
Counties	198	198	198	198
Observations	792	792	792	792

Notes: We cluster standard errors at the commute-zone level. Sample restricted to counties within 60 $\mu g/m^3$ of the regulatory cutoff, which occurs at 75 $\mu g/m^3$. "Post" indicates years 1972 and onward. Natality and economic controls use 1970 levels interacted with linear time trends (see Section 3).
* Significant at 10%; ** significant at 5%; *** significant at 1%.

avoid this bias, provided that attainment status is independent of such confounding trends.[20]

In Appendix Table B1, we consider IV results by subgroup, including mother's education, child's race, mother's age, and mother marital status. If pollution exposure impacts the probability of a birth being male through the fetal loss mechanism, we expect larger impacts on more sensitive subgroups, due to 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.[21] Results in Table B1 support the theory that impacts are larger for groups with statistically lower fetal health capital and/or health resources, including blacks, mothers with lower education, very young mothers, and single mothers.[22] In all subgroup regressions, we weight by number of observed births by subgroup in 1970 rather than population to better account for the size of the subgroup in each county. We balance the county panels separately for each subgroup measure, resulting in varied sample sizes. As such, comparisons between estimates, both across groups and to our main findings, are only suggestive.

7. Discussion on potential confounders

Goodman (2002) shows the CAAA had substantial economic consequences for the manufacturing sector. Since male fetuses are more susceptible than female fetuses to loss from other stressors in addition to pollution, this could bias per-unit impacts of TSPs on fetal health. If, for example, the CAAA led to job loss in nonattainment counties, this may decrease mother health, either though income loss or additional non-pollution stress (see Walker, 2011). This suggests a byproduct of non-pollution effects of the CAAA would be a decrease in the number of male births. If job loss as a result of the CAAA leads to lower levels of maternal nutrition, findings by Almond and Hoynes (2011) indicate the fraction of male births should decrease as well. We find the lower pollution caused by the CAAA is associated with an *increase* in the number of male births: both effects exert pressure in the opposite direction of our main effect and bias results toward zero, if at all. Importantly, we appropriately incorporate such other effects in our estimate of the total impact of the CAAA when using the reduced form.[23]

In a cross-sectional comparison, Almond and Edlund (2007) find significant differences between sex ratios among socioeconomic groups. Specifically, single mothers with less than a high school education are less likely to have males than married mothers with some college education. If in response to the CAAA policies, lower education mothers move out of nonattainment counties (or are more likely to avoid pregnancy), a change in mother composition not captured by either state-by-year effects or natality controls would impose bias.[24] Empirical data on mother characteristics cannot answer this question, as composition of mothers might change due to fetal losses as well (the characteristics of mothers that never give birth are just as unobservable as births that never occur). We can bound our result using the reduced form estimate in Table 4, which shows the CAAA led to a change in the probability of a male birth of approximately 0.9% of baseline. Pure substitution effects require almost 100% substitution of high education mothers for low education mothers in attainment counties.[25]

[20] Sanders and Stoecker (2011) shows that our attainment status instrument does not correlate with background trends in pollution.

[21] It may also be a "culling" effect has already taken place, eliminating the more sensitive fetuses for more susceptible groups, which would cause more sensitive groups to have smaller marginal estimates.

[22] In Sanders and Stoecker (2011), we explore how results vary by quarter of birth and trimester of exposure, though such analysis is complicated as economic and weather data are only at the calendar-year level. We find suggestive evidence impacts are largest in the first trimester of exposure and for mothers that give birth in the fourth quarter when using our first-difference model.

[23] Three states in our sample (California, New York, and Washington) legalized abortion during the early CAAA period. State-by-year fixed effects capture general, state-level changes in sex-selective abortion use that do not correlate directly with county-level attainment status. While ultrasound technology was rare at the time, sex-selective abortion (in favor of male births) in these states would bias results if county-level attainment status correlates with availability and use of ultrasound technology and abortion behavior by county. These three states represent approximately 25% of our main sample, so their omission decreases statistical precision in the first stage. Regardless, omitting them from regressions decreases our point estimates in our fixed effects model by less than a standard error. Availability of medical technology may also vary by level of urban development. More urbanized counties could be more industrial (and thus polluted) and have greater access to abortion and ultrasound technology. Using data from the Department of Commerce Bureau of the Census, we also stratify results by urban status. Results are similar across groups, though slightly larger for less urban counties—this may reflect our subgroup findings, suggesting less urban areas have limited medical abatement capital.

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

[25] Effects need not necessarily be limited to pregnancies that suffered from fetal loss after successful insemination. Our results may also include avoided initial pregnancies. Research in the medical field has proposed that observed changes in the sex

It is informative to place our effects in light of other studies finding fetal sex differences in response to other external stresses. Using the data from Appendix Table A1, the magnitude of the impact of the CAAA (0.47 percentage points) is approximately equal to the difference in the probability of a male birth between mothers that got early vs. late prenatal care (0.42 percentage points) and approximately 2/3rd the size of the difference between black and white mothers (0.67 percentages points). These are prevailing differences across time, not results from policy adjustments, but remain informative in considering magnitudes.

Evidence from the one-time shock of fallout from Chernobyl in Sweden (Almond et al., 2009) finds exposure resulted in a decrease in the probability of having a male of 1.6%, just under twice the size of our estimated 0.9% of baseline effect. Peterka et al. (2004) find the radiation-induced change in the number of male births was almost 4% for the Czech Republic, around 4 times the size of our estimates. These are one-time extreme shocks, while the CAAA was a large permanent improvement in ambient air quality. As an example of a more permanent program, Almond and Hoynes (2011) find the Food Stamp Program (FSP) rollout increased the fraction of births that are male among whites by 0.09% and blacks by 0.32%, while Norberg (2004) finds parents living together at time of conception were 14% more likely to conceive males. We are unaware of any conversion of health impacts from subclinical levels of radiation, nutritional deficiencies, or family makeup to the reduction in air pollution levels we examine, but we note that findings for other *in utero* health and stressor shocks are qualitatively similar to those we find for air pollution and the CAAA.

7.1. Expansion into estimating total fetal losses

Under proper conditions, one can use changes in the observed live sex ratio as a proxy for changes in fetal health capital. We next consider a metric to convert a change in this ratio into a raw number of avoided fetal losses. We combine our findings with several estimates of relative *in utero* sensitivities of males and females to estimate total avoided fetal losses. We highlight here that what we estimate is fetal *losses*—our estimates include any loss of a pregnancy, including those after 20 weeks gestation (classified as fetal deaths) and those occurring prior to 20 weeks (classified as miscarriages).

We build a range of estimates of the total *in utero* mortality effect of pollution levels using the causal impacts of pollution on neonatal deaths and deaths within one year to establish a relative sensitivity to pollution across sexes. Using post-natal mortality ratios as a proxy for unobservable fetal ratios assumes similar susceptibility differences across sexes to external stressors, which may not be the case, particularly given the potential for fetal selection which suggests surviving infants are more resilient that those that perished.[26]

Panels A and B of Table 6 present the causal impact of avoided CAAA deaths on neonatal mortality (death within 28 days of birth) and one year mortality for overall deaths (Column 1), and by gender (Column 2). We use our difference-in-difference reduced form fixed effects model, where each cell is a coefficient from a regression similar to Column 4 of Panel B in Table 3 and includes controls for weather, natality covariates, economic covariates, county fixed effects, and state-by-year fixed effects (our sample of counties differs slightly due to using only counties with observed mortality data for both sexes in all four years). The sign on overall losses, as well as losses split by gender, shows the CAAA reduced infant mortality as CG found. Consistent with the hypothesis of differential fetal loss rates *in utero*, males have greater post-natal mortality reductions than females in response to the CAAA. Column 1 of Panel A shows the CAAA reduced overall neonatal mortality by approximately 88 infant deaths per 100,000 live births. Column 2 shows the avoided losses were largely male, with 29 avoided deaths per 100,000 female births and 147 avoided deaths per 100,000 male births. Column 1 of Panel B shows 108 avoided infant losses per 100,000 total births. Column 2 shows 23 per 100,000 live female births and 193 per 100,000 live male births.

Column 4 of Table 6 shows estimated total fetal impacts using these relative sensitivity estimates. Using the ratio from neonatal mortality rates, the estimated reduced form impact in Table 4 translates to a combined impact of 9900 prevented fetal losses as a result of the CAAA. Using the ratio from one-year mortality rates, we estimate the total effect is estimated at around 8400 prevented fetal losses. We obtain these estimates noting that

$$\beta_{CAAA} = \frac{M}{M+F} - \frac{(M - male\,\text{deaths})}{(M - \text{male deaths}) + \left(F - \frac{1}{\Omega} male\,\text{deaths}\right)} \quad (7)$$

where M and F are the 352,488 male and 333,207 female births in the sample nonattainment counties in 1972 and Ω is the sensitivity of males relative to females, provided above.

We can also derive a total number of fetal losses by assuming female fetuses were entirely unaffected by the CAAA. This lower bound estimates the CAAA prevented 6600, total deaths (all male) or 0.01% of the total live birth population in sample nonattainment counties.

These calculations mean a one-unit TSP decrease, using the estimate from Column 4 in Panel B in Table 5, results in 100–110 fewer fetal losses per 100,000 live births (assuming a linear impact of TSPs). These effects are larger than those found in the literature on pollution and infant deaths. It is likely live births are more robust to stresses than a developing fetus (Chay and Greenstone, 2003b hypothesize much of the infant mortality effect they find is driven by fetal damages), and individuals can enact abatement actions in the presence of health complications more easily 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 offset negative effects. One cannot directly observe such effects with an injured fetus, and treating a fetus is more difficult that providing medical treatment to an infant.

Placing our results in the context of prior work on fetal losses, medical research suggests 31% of conceptions results in fetal loss (Wilcox et al., 1989). Using the observed births in our sample nonattainment counties, this suggests approximately 650,000 fetal losses per year. Thus, our estimates indicate the CAAA averted approximately 1% of annual fetal losses.[27]

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 sex 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 sexes, 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 losses. Similarly, if TSP or a correlated pollutant serves as an endocrine disruptor, some of our results may be due to sex switching *in utero*, though we are unaware of any human studies suggesting such effects.

[26] How this might bias calculations is dependent on such differences. If the sensitivity ratio for males vs. females is lower for fetuses than infants (e.g., fetal defenses help offset damages from pollution), this will artificially inflate our estimates. If fetal deaths effectively serve as "culling" and the surviving males are (relative to females) stronger, our estimates will be biased downward.

[27] There remains the challenge of quantifying the exact financial value of the change in sex ratios at birth and associated fetal losses. In the case of infant mortality,

Table 6
Total fetal losses—conversion metrics using diff-in-diff estimates of the effect of the CAAA on Infant death.

	(1) Total deaths	(2) Deaths by sex	(3) Relative sensitivity	(4) Impact of CAAA
Panel A: Neonatal mortality rate				
Classified	−87.793**	−29.232	5.07	9900
nonattainment × Post	(35.682)	(39.161)		
Classified		−117.489***		
nonattainment × Post × Male		(29.639)		
Panel B: One year mortality rate				
Classified	−107.794***	−23.274	8.39	8400
nonattainment × Post	(39.432)	(45.190)		
Classified		−169.570***		
nonattainment × Post × Male		(33.462)		
Counties	186	186		
Observations	1488	1488		
Total births	4,275,157			
Males		2,191,873		
Females		2,083,284		

Notes: Section 3 describes the data. Regression estimates (Columns 1 and 2 of Panels A and B) show impact of CAAA nonattainment status on the number of post-natal deaths. Regressions at the county-year level weighted by population in 1970, and control for weather, natality and economic covariates as well as county and state-by-year fixed effects as in column 4 of Table 4. We detail calculations in Section 7. We cluster standard errors at the commute-zone level. Sample restricted to counties within 60 $\mu g/m^3$ of the regulatory cutoff, which occurs at 75 $\mu g/m^3$. "Post" indicates years 1972 and onward. Natality and economic controls use 1970 levels interacted with linear time trends (see Section 3).
* Significant at 10%; ** significant at 5%; *** significant at 1%.

8. Conclusion

Measuring changes in fetal health presents several challenges. Post-natal measures of fetal health are net of selection, as researchers only observe birth weight averages and mortality rates among infants that survive to term. Medical data rarely record fetal deaths, and any records face selective recording and measurement problems, such as a truncated measurement window. Further, microdata are rarely available for historical or developing country data. Our solution to these complications, using changes in the sex ratio of live births as a proxy for changes in fetal health, exploits the medical finding that male fetuses are more susceptible to death from external stresses. This measure has the advantage that sex determination is orthogonal to many traditional sources of fertility bias, and researchers can use it to estimate the effects of other policy measures intended to improve maternal health and infant outcomes.

Using this metric, we consider how the Clean Air Act Amendments of 1970, one of the largest federal pollution regulations in the United States, improved fetal health. We use variation in pollution changes caused by CAAA attainment status to estimate how pollution improvements led to changes in the sex ratio of live births. The CAAA resulted in a 0.47 percentage point change in the probability of a live birth being male in affected counties, a change of approximately 0.9%. This means substantial improvements in fetal health from the policy, much of which is missed when using standard birth outcomes such as premature birth and low birth weight. Conventional estimates of the social cost of pollution and the benefits of environmental regulation are thus lower bounds of the true health gains.

We present a method to convert sex ratio changes to a measure of avoided total fetal losses, combining the change with observed differential effects of the policy post-birth. This method requires only that the researcher have information regarding, (1) the sex ratio of live births and (2) the differential effects of a policy on a measurable post-birth outcome by sex, which they can use to extrapolate the total effect from the observed impact on male fetal losses relative to females. This method also accounts for fetal losses prior to 20 weeks, serving as a better overall measure of fetal health than the recorded fetal death rate.

Appendix A. Environmental stressors, fetal susceptibility, and effects by sex

The health consequences of early-life exposures to environmental externalities have received a good deal of attention in applied research as of late. Reyes (2007) and Nilsson (2009) link lead exposure to lowered IQ and increased aggression, while Clay et al. (2010) find lead exposure leads to increased infant mortality. Studies link carbon monoxide to increased infant mortality (Currie and Neidell, 2005), low birth weight and preterm birth (Ritz and Yu, 1999; Currie et al., 2009), and increased school absences in young children (Currie et al., 2009), ozone to higher asthma rates and cardiac difficulties (Neidell, 2004, 2009; Lleras-Muney, 2010; Moretti and Neidell, 2011), and particulate matter to increases in infant mortality rates (Chay and Greenstone, 2003b,a; Knittel et al., 2011), as well as the incidence of low birth weight (Wang et al., 1997). Studies on less pollutant-specific environmental factors such as proximity to traffic pollution (Currie and Walker, 2011), presence of toxic releases (Currie and Schmieder, 2009), proximity to Superfund cites (Currie et al., 2011), and presence of a steel mill (Parker et al., 2008) show various negative fetal health impacts including low birth weight, premature birth, infant mortality, and congenital anomalies. A more comprehensive review of the fetal environmental literature is available in Currie (2011).

We focus on total suspended particulates (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

one can use measures of the value of a statistical life to financially quantify the impacts. Fetal losses are more complicated. While miscarriages and fetal deaths carry with them both medical and substantial psychological costs, some may occur without the knowledge of the mother. Of the fetal deaths that occur in known pregnancies, the argument could be made that actual financial costs should be less than that of an infant death, as less time and fewer resources have been invested, but it remains difficult to quantify the costs associated with the potentially substantial psychological impacts. We leave this for as an area for further study.

micrometers in size.[28] Exposure to TSPs could harm fetal development by compromising the mother's health or impacting the fetus directly, both of which have been documented in the medical and environmental literature. TSP levels are also highly correlated with other damaging pollutants that, while un-monitored at the time of the CAAA, likely saw substantial decreases due to the regulations imposed by the CAAA. A number of pollutants have noted fetal impacts: elevated prenatal radiation exposure links to lower test scores (Almond et al., 2009), and particulate exposure to lower IQ scores at age 5 (Perera et al., 2009) and lower high school standardized test performance (Sanders, 2011). For further discussion of how negative fetal shocks (environmental and otherwise) can have lasting life effects, see Almond and Currie (2011).

We base our use of the sex ratio as a measure of fetal losses on the greater susceptibility of male fetuses to external stressors (see Kraemer, 2000).[29] In our context, exposure to pollutants compromises the health of the mother, and the Trivers–Willard mechanism suggests this leads to a lower probability of a live male birth. In order for this hypothesis to apply, we do not assume an evolutionary response to TSP exposure *per se*. More simply, if an evolutionary response to poor health exists, and TSP exposure increases poor health, the application is a natural one. Observational evidence of lower male/female sex ratios among those exposed to higher levels of particulate matter in São Paulo, Brazil suggests such a link exists (Lichtenfels et al., 2007).

Such favoring occurs via male fetal loss, or shortly after conception via preventing implantation of male embryos. 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 sex 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 et al. (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, and the associated increase in consumption, decreased the percentage of male births among cohorts that had been conceived prior to the price decrease. In Sanders and Stoecker (2011) we examine pollution effects differently by trimester of gestation and find similar effects across all three trimesters. We use the term fetal loss to encompass both failed implantations and post-implantation fetal deaths. Unfortunately, the direct mechanism through which TSPs might influence either maternal or fetal health is unknown. We focus on the causal relationship between higher pollution rates and fetal losses but do not attempt to identify the direct mechanism through which this effect operates. We next consider suggestive data evidence of differentials in live male births across time and subgroups in the United States.

Table A1
Percentage of live births that are male by subgroup.

Mother category	Percentage male	Births (1000)
Early prenatal care	51.25	53,874
Late prenatal care	50.83	1288
Married	51.31	58,571
Single	51.03	11,795
Young	51.33	7273
Mid	51.27	59,116
Older	51.11	3978
Black	50.71	11,423
White	51.38	56,659
HS or less	51.24	35,965
HS and above	51.36	16,048
QOB1	51.23	16,962
QOB2	51.37	17,077
QOB3	51.27	18,703
QOB4	51.20	17,625

Notes: Each line presents the mean percentage of live births that are male and total live births by group from 1968 to 1988.

A.1. Birth trends in the United States

Given the hypothesis that male fetuses are "weaker", we expect to see a greater number of females born to subgroups who are more likely to be exposed to stressors (e.g. pollution, nutrient depravation, smoking, alcohol consumption, etc.) and/or less likely to have fetal "damage abatement" capital (e.g., prenatal care). In Table A1, we show the mean percentage of live births that are male by year as well as differences by subgroup for 1968–1988, the years for which natality data by county and sex are available.

As documented by Almond and Edlund (2007), there are differences in the sex ratio among subgroups, and all differences move in the direction supporting the vulnerable male fetus hypothesis. A male birth is less likely for mothers with lower education, single mothers, older (over 35) mothers, black mothers, mothers that delayed prenatal care, and mothers that gave birth in quarters 1 and 4 (see Buckles and Hungerman, 2008). In Appendix B, we explore differences in the impact of pollution across subgroups (when sufficient data are available) and find effects are largest for those with the greatest susceptibility to fetal loss (as indicated by the baseline sex ratio).

Sex ratios of live births not only vary across groups, but across time. The live birth sex ratio in the United States as of 2006 was approximately 1.05 males per female, a probability of any one live birth being male of approximately 51.21 percent. The probability during our study period was similar at 51.29 percent. The sex ratio at birth in the United States has slowly fluctuated over the years, with a largely constant downward trend beginning in the 1970s, as Column 6 of Table A2. No known explanation exists for the gradual decrease in the share of live male births, though there exist a number of theories. Mathews and Hamilton (2005) specifically notes changes in the age of the father, lower maternal weight, stress, and environmental toxins as potential factors previously studied in the literature.

Most relevant to our period of interest is the shift in trend in 1970, which was accompanied by a brief downward swing in the share of overall live births that are male. Between 1970 and 1972, there was a nationwide drop in the share of male births, with a reversal of almost equal size occurring in 1973 and 1974 (see Mathews and Hamilton, 2005). As with the general trend in sex ratios, no current theory can explain this particular dip. What

[28] 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.

[29] A proposed evolutionary advantage for this susceptibility is that women in poor health are more likely to produce female children than male children. This hypothesis, first proposed by Trivers and Willard (1973), states carrying a fetus to term is costly and it is beneficial to ensure the ensuing child will produce grandchildren. Since males can simultaneously father children with multiple females, males in good health could secure several mates, while males in poor health might secure none. For females the relationship between health and mating is less pronounced, as females in poor health can still secure mating opportunities with males in good health. If maternal health is an indicator for potential future infant health conditions, the Trivers–Willard hypothesis predicts that fetal survival rates are more likely to biologically favor females when mothers are in poor health themselves, as this will maximize the mating opportunities for their children, and thus also maximize their chances of having grandchildren.

Table A2
Differences in percentage of live births that are male by subgroup and year.

Year	Yearly differences by subgroups					Annual avg.
	Prenatal care	Marital status	Mother age	Child race	Mother education	
1969	0.69	0.34	0.28	0.88	0.05	51.29
1970	1.10	0.44	0.55	0.68	0.17	51.33
1971	0.47	0.46	0.31	0.67	0.13	51.26
1972	0.29	0.50	0.36	0.79	0.17	51.25
1973	0.31	0.42	0.36	0.69	0.20	51.27
1974	0.77	0.45	0.10	0.71	0.04	51.33
1975	0.21	0.35	0.30	0.68	0.05	51.31
1976	0.29	0.21	−0.17	0.73	0.08	51.28
1977	0.54	0.35	0.22	0.78	0.06	51.28
1978	0.66	0.31	−0.02	0.71	0.40	51.28
1979	0.13	0.24	−0.08	0.67	0.01	51.26
1980	0.10	0.19	0.20	0.69	0.14	51.29
1981	0.28	0.15	0.24	0.70	0.12	51.26
1982	0.56	0.30	0.03	0.50	0.14	51.23
1983	0.39	0.30	0.24	0.70	0.25	51.27
1984	0.17	0.28	0.07	0.55	0.06	51.22
1985	0.41	0.17	0.10	0.63	0.28	51.27
1986	0.40	0.20	0.14	0.47	0.13	51.24
1987	0.36	0.22	0.05	0.61	−0.03	51.22
1988	0.14	0.17	−0.05	0.49	0.17	51.22

Notes: Each subgroup line presents the difference between live male birth rates by groups described in column headers: first prenatal care (1–3 months – 7–9 months), marital status (married – single), mother age (over 35 – 19–35), child race (white – black) and maternal education (above high school – below high school). Percent male over time is for all sample counties.

is clear is that such general movements can make OLS identification problematic, which drives our use of the CAAA as a source of identification and a potential solution to the estimation complications of general sex ratio and pollution trends. The fundamental assumption is that the nonattainment counties serve as good counterfactuals for attainment counties.

Table A2 shows that, despite changes in the average sex ratio at live birth over time, there remain differences between the more susceptible vs. less susceptible groups discussed in Table A1. Columns 1 through 5 show differences over time by: first prenatal care use (7–9 months vs. 1–3 months), marital status (married vs. single), mother age (over 35 vs. 19–35), child race (white vs. black), and maternal education (above high school vs. high school and below). In each case, the percentage of live births that are male in the disadvantaged group is subtracted from the same percentage from the advantaged group. Over all periods between 1969 and 1988, there remain differences between said groups. In nearly all group and year cells, the difference is in the direction of the advantaged group having more males as a percentage of total live births than the disadvantaged group, showing strong correlation between fetal health and sex ratios.

Appendix B. Effects by subgroup

Tables A1 and A2 suggest certain groups are more susceptible to fetal shocks, given lower health capital and availability of health resources. On the other hand, lower baseline male-birth ratios might indicate "culling", suggesting lower health capital groups might see less of an impact. To better investigate this issue, we consider how impacts vary by different demographic groups in Table B1. Here we weight by number of baseline 1970 births in each subgroup rather than by population, given our specific focus on subgroups. We also limit counties to those with observed data for each subgroup across all four years. While we weight by births in 1970 (before the policy and thus unaffected by treatment), it still makes comparison to population weight regressions more difficult. However, information on subpopulation counts for groups like single mothers and mothers of different age groups at the county-by-year level are not available. As such, we view these results as suggestive and less directly comparable in magnitudes to our primary results.

Columns 1 and 2 show results for mothers with high school education or lower and greater than a high school education,

Table B1
First difference IV results for probability of a live birth being male by maternal subgroups.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Mother education		Child race		Mother age			Marital status	
	<HS	≥HS	White	Black	≤19	>19 & ≤35	>35	Single	Married
Mean TSPs	−0.084**	0.044	−0.043**	−0.06	−0.115*	−0.02	0	−0.118*	−0.036**
	(0.040)	(0.037)	(0.017)	(0.123)	(0.064)	(0.018)	(0.097)	(0.068)	(0.017)
First stage *F*	11.79	22.97	19.54	4.77	18.5	14.61	25.04	23.28	26.68
Counties	740	740	528	528	668	668	668	704	704
Baseline (1970) births	2,143,200	713,992	2,730,856	534,736	495,344	2,581,600	219,544	441,192	3,380,104

Notes: Section 3 describes the data. 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 subgroup baseline births in 1970. We cluster standard errors at the commute-zone level. Sample restricted to counties within 60 $\mu g/m^3$ of the regulatory cutoff, which occurs at 75 $\mu g/m^3$. Natality and economic controls use 1970 levels interacted with linear time trends (see Section 3).
* Significant at 10%; significant at 5%; *** significant at 1%.

respectively. Results show mothers with lower education levels (comprising approximately $\frac{3}{4}$ of the sample), a factor correlated with availability of fetal damage abatement capital, see a larger impact on sex ratios when exposed to pollution. The result remains statistically significant at 5%. Higher education mothers show no statistically significant effects. Column 3 shows the effect for whites. While still large and significant, it is approximately $\frac{1}{2}$ the estimated effect for blacks (though the standard error on the black estimate is large). 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.[30] These noted differences in use of prenatal care across races further support our prior finding—blacks have a lower use of prenatal damage abatement capital, and thus see a larger effect on fetal loss. Columns 5, 6, and 7 show results for mothers younger than 20, 20–34, and 35 years old and up, respectively. Effects decrease with age, though no group result has results significant at conventional levels. Finally, results for single and married mothers are shown in columns 8 and 9, respectively. Results for married mothers are approximately the same as the full sample result, while results for single mothers are much larger though statistically insignificant at conventional levels.

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