Air Quality and Early-Life Mortality
Evidence from Indonesia's Wildfires

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ABSTRACT

Smoke from massive wildfires blanketed Indonesia in late 1997. This paper examines the impact that this air pollution (particulate matter) had on fetal, infant, and child mortality. Exploiting the sharp timing and spatial patterns of the pollution and inferring deaths from "missing children" in the 2000 Indonesian Census, I find that the pollution led to 15,600 missing children in Indonesia (1.2 percent of the affected birth cohorts). Prenatal exposure to pollution drives the result. The effect size is much larger in poorer areas, suggesting that differential effects of pollution contribute to the socioeconomic gradient in health.

I. Introduction

Between August and November 1997, forest fires engulfed large parts of Indonesia, destroying over 12 million acres. Most of the fires were started intentionally by logging companies and palm oil producers clearing land to plant new crop.[1] Because of the dry, windy conditions caused by El Niño, the fires burned out of control and spread rapidly. In November, rains finally doused the fires.

1. The Indonesian Minister of Forestry estimated that ''[commercial] plantations caused some 80 percent of the forest fires,'' and that small farmers caused the remainder (*Straits Times*, September 3, 1997). Rabindran (2001), using satellite data on land use, finds that the 1997 incidence of fires on plantations was higher than the ''natural'' level (based on a benchmark from conservation areas), but the incidence of fires on small farms was at its natural level.

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While the fires were burning, much of Indonesia, especially Sumatra and Kalimantan where the fires were concentrated, was blanketed in smoke. This paper examines fetal, infant, and child mortality caused by the episode of poor air quality (specifically, high levels of particulate matter). Daily satellite measurements of airborne smoke at locations across Indonesia provide information on the spatial and temporal patterns of the pollution. The outcome is fetal, infant, and child mortality before age three—hereafter called early-life mortality—and is inferred from ''missing children'' in the 2000 Census, overcoming the lack of mortality records for Indonesia and the small samples in surveys with mortality data.

The paper finds that higher levels of pollution caused a substantial decline in the size of the surviving cohort, and that exposure to pollution during the last trimester in utero is the most damaging. The fire-induced increase in air pollution is associated with a 1.2 percent decrease in cohort size. This estimate is the average across Indonesia for the five birth-month cohorts with high third-trimester exposure to pollution; it implies that 15,600 child, infant and fetal deaths are attributable to the pollution. Indonesia's under-three mortality rate during this period was about 6 percent; if the estimate is driven mainly by child and infant deaths (rather than fetal deaths), this represents a 20 percent increase in under-three mortality.

The paper also finds a striking difference in the mortality effects of pollution between richer and poorer places. Pollution has twice the effect in districts with below-median consumption compared to districts with above-median consumption. Individuals in poorer areas could be more susceptible to pollution because of lower baseline health, more limited options for avoiding the pollution, or less access to medical care. Another possibility is that people exposed to indoor air pollution on a daily basis suffered more acute health effects from the wildfires because they received a double dose of pollution. Consistent with this view, the effects are larger in areas where more people cook with wood-burning stoves. Surprisingly, mother's education does not seem to play a role. While these correlations do not pin down causal relationships, they provide suggestive evidence on why the poor are especially vulnerable to the health effects of pollution and add to our understanding of the socioeconomic (SES) gradient in health (Marmot *et al.* 1991; Smith 1999). An open question in the literature is how much of the health gradient is due to low-SES individuals being more likely to suffer adverse health shocks and how much is due to a given health shock having worse consequences for the poor (Case, Lubotsky, and Paxson 2002; Currie and. Hyson 1999). This paper provides some evidence for the second mechanism: People faced a common environmental-cum-health shock, and the consequences were much worse for the poor.

This study examines an extreme episode of increased pollution, which has advantages for empirical identification. The abruptness and magnitude of the pollution are useful for identifying when in early life exposure to pollution is most harmful. In utero exposure is found to be especially important, suggesting that targeting pregnant women should be a priority of public health efforts concerning air pollution. The extreme level of pollution, however, also means that one must be cautious about extrapolating the effect size to more typical levels of pollution.

That being said, rampant wildfires are not uncommon in Indonesia and other countries, mainly in Southeast Asia and Latin America, where fire is used to clear land. Most cost estimates of the 1997 Indonesian fires have focused on destroyed timber,

reduced worker productivity, and lost tourism and are in the range of $2 to 3 billion (Tacconi 2003). This study shows that the health costs of the fires are much larger: Assuming a value of a statistical life of $1 million, the early-life mortality costs alone were over $15 billion.[2] The costs of the fires very likely overwhelm the benefits to firms from setting them; the annual revenue from Indonesia's timber and palm oil industries at this time was less than $7 billion.

The remainder of the paper is organized as follows. Section II provides background on the link between pollution and health and on the Indonesian fires. Section III describes the data and empirical strategy. Section IV presents the results, and Section V concludes.

II. Background

A. Link between air pollution and early-life mortality

1. Related Literature

Previous related work includes that by Chay and Greenstone (2003b), who use variation across the United States in how much the 1980–81 recession lowered pollution and find that better air quality reduced infant mortality. Chay and Greenstone (2003a) also find that pollution abatement after the Clean Air Act of 1970 led to a decline in infant deaths.[3] Currie and Neidell (2005), in their study of California in the 1990s, find that exposure to carbon monoxide and other air pollutants during the month of birth is associated with infant mortality.[4]

In addition, there have been studies on the adult health effects of Indonesia's 1997 fires. Emmanuel (2000) finds no increase in mortality but an increase in respiratory-related hospitalizations in nearby Singapore. Sastry (2002) finds increased mortality for older adults on the day after a high-pollution day in Malaysia. Frankenberg, Thomas, and McKee (2004) compare adult health outcomes in 1993 and 1997 for areas in Indonesia with high versus low exposure to the 1997 smoke. They find that pollution reduced people's ability to perform strenuous tasks and other measures of health. Their data set covers 321 of the 4,000 subdistricts in Indonesia, and only one of Kalimantan's four provinces is in their sample. Thus, one advantage of this paper is its broader geographic coverage, which allows one to explore heterogeneous effects and nonlinearities in the health impact of pollution, for example.

2. This value of a statistical life (VLS) is calculated using $5 million in 1996 dollars as the value for the United States from Viscusi and Aldy (2003), Murphy and Topel (2006), and the U.S. Environmental Protection Agency (2000); an income elasticity of a VSL of 0.6 from Viscusi and Aldy (2003); and the fact that Indonesia's per capita gross domestic product was one tenth of U.S. GDP. Note that the total health costs from early-life exposure to the pollution would also include costs among those who survived. Recent work suggests that there could be long-term consequences among surviving fetuses (Barker 1990; Almond 2006).

3. Other natural experiments used to measure health effects of air pollution include the temporary closure of a steel mill in Utah during a labor dispute; the reduction in traffic during the 1996 Olympics in Atlanta; and involuntary relocation of military families (Pope, Schwartz, and Ransom 1992; Friedman *et al.* 2001; Lleras-Muney 2006).

4. For research on pollution and infant mortality outside the United States, see, for example, Bobak and Leon (1992) on the Czech Republic, Loomis *et al.* (1999) on Mexico, and Her Majesty's Public Health Service (1954) on the 1952 London "killer fog" episode.

Another important advance over previous work is the use of both the sharp timing and extensive regional variation of the pollution; one can then identify causal effects while allowing for considerable unobserved heterogeneity across time and place.

2. *Physiological Effects of Pollution*

Smoke from burning wood and vegetation, or biomass smoke, consists of very fine particles (organic compounds and elemental carbon) suspended in gas. Fine particles less than 10 microns (μm) and especially less than 2.5 μm in diameter are considered the most harmful to health because they are small enough to be inhaled and transported deep into the lungs. For biomass smoke, the modal size of particles is between 0.2 and 0.4 μm, and 80 to 95 percent of particles are smaller than 2.5 μm (Hueglin *et al.* 1997).

Prenatal and postnatal exposure to air pollution could affect fetal or infant health through several pathways. Postnatal exposure can contribute to acute respiratory infection, a leading cause of infant death. Prenatal exposure can affect fetal development, first, because pollution inhaled by the mother interferes with her health, which in turn disrupts fetal nutrition and oxygen flow, and, second, because toxicants cross the placenta. Several studies find a link between air pollution and fetal growth retardation or shorter gestation period, both of which are associated with low birthweight (Dejmek *et al.* 1999; Wang *et al.* 1997; Berkowitz *et al.* 2003). The biological mechanisms behind these pregnancy outcomes are related to the main toxicant in particulate matter, polycyclic aromatic hydrocarbons (PAHs). In utero exposure to particulate matter has been associated with a greater prevalence of PAH-DNA adducts on the placenta, and PAH-DNA adducts, in turn, are correlated with low birth weight, small head circumference, preterm delivery, and fetal deaths (Perera *et al.* 1998; Hatch, Warburton, and Santella 1990). Laboratory experiments on rats have confirmed most of these effects (Rigdon and Rennels 1964; MacKenzie and Angevine 1981). PAHs disrupt central nervous system activity of the fetus, and during critical growth periods such as the third trimester, the disruption has a pronounced effect on fetal growth. PAHs are also hypothesized to reduce nutrient flow to the fetus by suppressing estrogenic and endocrine activity and by binding to placental growth factor receptors (Perera *et al.* 1999).

B. Description of the Indonesian Fires

The 1997 dry season in Indonesia was particularly dry. Figure 1 shows the monthly rainfall recorded at a meteorological station in South Sumatra in 1997 compared to previous years. The 1997 dry season was both severe and prolonged: Rainfall amounts in June to September were lower than usual, and the rainy season was delayed until November. The rest of Indonesia experienced rainfall patterns similar to Sumatra's.

Fires are commonly used in Indonesia to clear land for cultivation, and the dry season is considered an opportune time to set fires because the vegetation burns quickly. Industrial farmers burn forest land in order to replant it with palm or timber trees, and small farmers use ''slash–and–burn'' techniques in which land is cleared with fire to ready it for cultivation. In addition, logging companies are thought to have set some

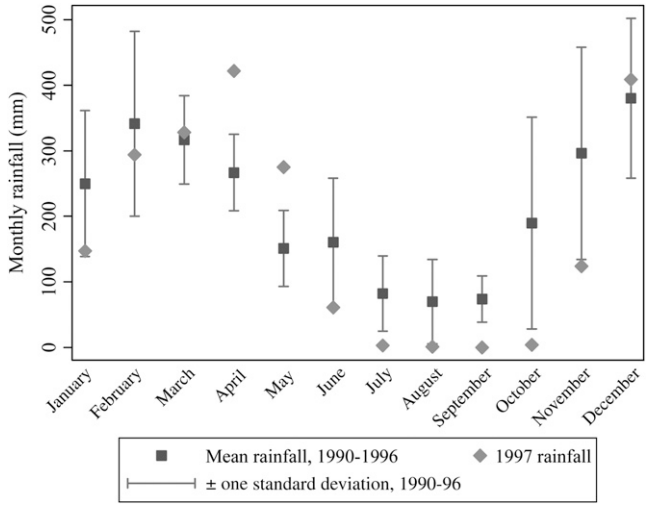

Figure 1
Rainfall at Palembang Airport meteorological station, South Sumatra, 1990-97

virgin forests on fire since the government would then designate the degraded land as available for logging.

With expansion of the timber and palm oil industries in Indonesia, many tracts of forest land have become commercially developed, and logged-over land is more prone to fires than pristine forest.[5] Roads running through forests act as conduits for fire to spread, and with the canopy gone, the ground cover becomes drier and more combustible and wind speeds are higher. Also, because logging firms were taxed on the volume of wood products that left the forest, they often left behind waste wood, even though it had economic value as fertilizer or wood chips. The left-behind debris wood made the forest more susceptible to fast-spreading fires (Barber and Schweithhelm 2000).

In September 1997, because of the dry conditions, the fires spread out of control. The Indonesian government made some attempt to fight the fires, but the efforts were ineffective. The fires continued until the rains arrived in November. The fires were concentrated in Sumatra and Kalimantan. About 12 million acres were burned, 8 million acres in Kalimantan (12 percent of its land area) and four million in Sumatra (4 percent of its area). The practice of clearing land with fire is used throughout Indonesia, and El Niño affected all of Indonesia. What set Sumatra and Kalimantan apart is that Indonesia's forests are mainly in these areas. The majority of crop plantations are located in Sumatra, and plantations are a fast-growing use of land in Kalimantan. Timber operations are also primarily in these regions.

5. In 1996 forest products accounted for 10 percent of Indonesia's gross domestic product, and Indonesia supplied about 30 percent of the world palm oil market (Ross 2001).

The location of the smoke generally tracked the location of the fires, though because of wind patterns, not entirely. Fires were concentrated on the southern parts of Sumatra and Kalimantan, and these two areas experienced the most pollution. On the other hand, the northern half of Sumatra was strongly affected by smoke while Java was relatively unaffected, yet neither of these areas experienced fires, for example.

A common measure of particulate matter is PM_{10}, the concentration of particles less than ten μm in diameter. The U.S. Environmental Protection Agency has set a PM_{10} standard of 150 micrograms per cubic meter ($\mu g/m^3$) as the 24-hour average that should not be exceeded in a location more than once a year. During the 1997 fires, the pollution in the hardest hit areas surpassed 1000 $\mu g/m^3$ on several days and exceeded 150 $\mu g/m^3$ for long periods (Ostermann and Brauer 2001; Heil and Goldammer 2001). The pollution levels caused by the wildfires are comparable to levels caused by indoor use of wood-burning stoves. The daily average PM_{10} level from wood-burning stoves, which varies depending on the dwelling and duration of use, ranges from 200 to 5000 $\mu g/m^3$ (Ezzati and Kammen 2002).

III. Empirical Strategy and Data

A. Empirical Model and Outcome Variable

The goal of the empirical analysis is to measure the effect that air pollution from the wildfires had an on early-life mortality. Ideally, there would be data on all pregnancies indicating which ended in fetal, infant, or child death, and the following equation would be estimated:

$$(1) \quad Survive_{jt} = \beta_1 Smoke_{jt} + \delta_t + \alpha_j + \varepsilon_{jt}.$$

The variable $Survive_{jt}$ is the probability that fetuses whose due date is month t and whose mothers reside at the time of the fires in subdistrict j survive to a certain point, such as live birth, one year, etc. The prediction is that β_1 is negative, or that exposure to smoke around the time of birth reduces the probability of survival.

In practice, mortality records are unavailable for Indonesia, and survey samples are too small to examine the effects of month-to-month fluctuations in pollution. For example, the 2002 Demographic and Health Survey has on average one birth and 0.05 recorded child deaths per district-month for the affected cohorts.[6] Therefore, the approach I take is to infer early-life mortality by measuring "missing children." The outcome measure is the cohort size for a subdistrict-month calculated from the complete 2000 Census of Population for Indonesia. The estimating equation is

$$\ln(CohortSize)_{jt} = \beta_1 Smoke_{jt} + \beta_2 PrenatalSmoke_{jt} + \beta_3 PostnatalSmoke_{jt} + \delta_t + \alpha_j + \varepsilon_{jt} \quad (2)$$

The dependent variable, $\ln(CohortSize)_{jt}$, is the natural logarithm of the number of people born in month t who are alive and residing in subdistrict j at the time of the 2000

6. Appendix 1 verifies that population counts from the Census move one-for-one with births and deaths in the Demographic and Health Survey sample.

Census. $Smoke_{jt}$ is the pollution level in the month of birth, and $PrenatalSmoke_{jt}$ and $PostnatalSmoke_{jt}$ are the pollution level before and after the month of birth. Each observation is weighted by the subdistrict's population (the number of people enumerated in the Census who were born in the two years prior to the sample period).

An advantage of the Census compared to a survey is that the data are for the entire population. Also, the outcome variable measures fetal deaths in addition to infant and child deaths, albeit without distinguishing between the different outcomes; most surveys do not collect data on fetal deaths. Finally, population counts are often better measured than infant and child mortality because of underreporting of deaths and recall error on dates of deaths.

There are several potential concerns about inferring mortality from survivors, however. Since the data come from a cross-section of survivors in June 2000, the outcome represents a different length of survival for individuals born at different times, and the mean level of survival will differ by cohort, independent of the fires. For a cohort born in December 1997 around the time of the fires, the outcome is survival until age two and a half, while for an older cohort born in December 1996, the outcome is survival until age three and a half, for example.[7] The inclusion of birthyear-birthmonth (hereafter, month) fixed effects in the regression will control for any average differences in survival by cohort.

In using ln(*CohortSize*) as a proxy for the early-life mortality rate, an assumption is that, conditional on subdistrict and month fixed effects, pollution is not correlated with *ln*(*Births*). This seems like a reasonable assumption. First, by using a short panel, subdistrict fixed effects absorb most variation in the number of women of childbearing age and other determinants of fertility. Month effects control for fertility trends and seasonality. Second, it seems unlikely that there were large fluctuations in fertility that coincided with the air pollution both spatially and temporally. Even area-specific trends could not explain the patterns since the sample includes control periods both before and after the fires; any omitted fertility shift causing bias would have to be a short-term downward or upward spike in particular regions. In addition, Section IVB provides empirical evidence that fertility is unlikely to be a confounding factor.

Another concern is that if pollution affects the duration of pregnancies, then missing children might result from the shifting of births from certain months to other months. If exposure to smoke induces preterm labor, then one would expect to see an excess of births followed by a deficit of births. In Section IVB, I examine and am able to reject that the results are an artifact of changes in gestation period.

There are also potential empirical concerns not unique to using ln(*CohortSize*) as the dependent variable. First, pollution might affect not only mortality but also fertility. This would influence the population counts for the later "control" cohorts and could lead to sample selection problems even if mortality were directly measured. I therefore restrict the sample to births occurring no more than eight months after the outbreak of the fires, or cohorts who were conceived before the fires. Second, the empirical model assumes that exposure to pollution just before or after birth affects mortality, an assumption motivated by previous findings. However, exposure

7. One advantage of observing survival more than two years after the due date is that for deaths that occur around birth, the estimates are less likely to reflect simply short-term "harvesting."

to pollution earlier in a pregnancy or later after birth also could affect health. If the control cohorts are in fact also treated, though less intensely, then the results would underestimate the true effects.

A third important concern arises from the fact that individuals are identified by their subdistrict of residence in 2000 rather than the subdistrict where their mother resided during the end of her pregnancy or just after giving birth. If families living in high-smoke areas with children born around the time of the fires were more likely to leave the area (either during or after the fires), then the cohort size would be smaller in areas more affected by pollution. Fortunately, one can directly examine this concern since the Census collects the district of birth and the district of residence in 1995. As discussed in Section IVB, the results are identical using birthplace, current location, or mother's location in 1995.

Table 1 presents the descriptive statistics. The sample comprises monthly observations between December 1996 and May 1998 (18 months) for 3,751 subdistricts (*kecamatan*). Of this starting sample size of 67,518 observations, 64 observations are dropped because the cohort size for the subdistrict-month is 0.[8] There are on average 96 surviving children per observation. The larger administrative units in Indonesia are districts (*kabupaten*), of which there are 324 in the sample, and provinces, of which there are 29.

B. Pollution Variable

The measure of air pollution is the aerosol index from the Earth Probe Total Ozone Mapping Spectrometer (TOMS), a satellite-based monitoring instrument. The aerosol index tracks the amount of airborne smoke and dust and is calculated from the optical depth, or the amount of light that microscopic airborne particles absorb or reflect. The TOMS index has been found to quite closely track particulate levels measured by ground-based pollution monitors (Hsu *et al.* 1999). Ground monitor data are not available for Indonesia for this period. The aerosol index runs from -2 to 7, with a higher index indicating more smoke and dust.

The TOMS data contain daily aerosol measures (which are constructed from observations taken over three days) for points on a 1 degree latitude by 1.25 degree longitude grid. Adjacent grid points are approximately 175 kilometers (km) apart. The probe began collecting data in mid-1996, and the data I use begin in September 1996. For each subdistrict, I calculate an interpolated daily pollution measure that combines data from all TOMS grid points within a 100-km radius of the geographic center of the subdistrict, weighted by the inverse distance between the subdistrict and the grid point. The number of TOMS grid points that fall within the catchment area of a subdistrict ranges from one to six and is on average four. The mean distance between a subdistrict's center and the nearest grid point is 50 km.

8. The Census covers 3,962 subdistricts that make up 336 districts. For subdistricts dropped from the sample, either the latitude and longitude could not be determined or there were no enumerated children for more than 15 percent of the monthly observations due to missing data or very small subdistrict size. In addition, I drop four districts that make up Madura since this area received a large influx of return migrants in 1999 in response to ethnic violence against them in Kalimantan, and also Aceh province where separatist violence is thought to have affected the quality of the Census enumeration. The results are robust to dropping Irian Jaya, another area where unrest could have affected data quality.

Table 1
Descriptive Statistics

	Mean	Std. Dev.
Cohort size variables		
Cohort size (for subdistrict-month)	95.6	89.7
Ln(cohort size)	4.8	0.8
Pollution variables		
Smoke (median daily value for month)	0.087	0.424
Prenatal smoke ($Smoke_{t\text{-}1,2,3}$)	0.095	0.330
Postnatal smoke ($Smoke_{t+1,2,3}$)	0.074	0.342
Proportion of days with high smoke (aerosol index > 0.75)	0.047	0.154
Average smoke (daily values averaged for the month)	0.120	0.445
Mean of smoke for Aug-Oct 1996	0.048	0.069
Mean of smoke for Aug-Oct 1997	0.578	0.791
Mean of prenatal smoke for Sept 1996–Jan 1997	0.038	0.052
Mean of prenatal smoke for Sept 1997–Jan 1998	0.341	0.522
Other variables		
Fires (any fires)	0.157	0.364
Intense fires (number x duration of fires $\geq$ 10 fire-days)	0.026	0.157
Rainfall for June to November 1997 relative to 1990–95	0.480	0.241
Ln(median 1996 household food consumption)	10.52	0.26
75th percentile	10.71	
50th percentile	10.49	
25th percentile	10.33	
Median HH food consumption in 1996 / Median HH food consumption in 1998	0.742	0.070
National consumer price index (for food)	1.131	0.202
Urbanization	0.57	0.39
Wood as primary cooking fuel	0.636	0.413
Doctors per 1,000 people	0.161	0.241
Maternity clinics per 1,000 people	0.031	0.050
Educated mothers (completed junior high)	0.386	0.215

Note: The sample consists of 67,454 subdistrict-birthmonths from December 1996 to May 1998. Sample averages are weighted by population (the number of people enumerated in the Census born in the year before the sample period), except for cohort size for which the unweighted mean is shown. Cohort size is the number of people enumerated in the 2000 Census who were born in a subdistrict in a given month. Smoke is the monthly median of the daily TOMS aerosol index which is interpolated from TOMS grid points within 100 km of the subdistrict's geographic center and weighted by the inverse distance between the grid point and subdistrict center. Prenatal and Postnatal Smoke are averages of Smoke for the three months before and after the month of birth. Fire-days is calculated from European Space Agency hot spots within 50 km of the subdistrict's center. Rainfall is measured at the nearest grid point on a 0.5 degree latitude/longitude grid and is the mean of 1997 rain relative to 1990–95 for June to November. Urbanization is the subdistrict's percent of births in urban areas based on those born 1994–96 and uses an indicator in the Census of whether the respondent's locality is rural or urban. Educated mothers is the percent of infants whose mother has completed junior high and is based on matching infants to mothers in the Census. Median food consumption is a per capita measure for each district that uses data from the 1996 and 1999 SUSENAS household survey. Consumer price index is from the Indonesian central bank. Healthcare variables are calculated for each subdistrict using the 1996 PODES (survey of village facilities). PODES and SUSENAS data are available for 63,158 observations.

The monthly measure is calculated as the median of the daily values, and I also use the mean of the daily values and the number of days that exceed a threshold value of 0.75.

The data include more than 3,700 subdistricts, but only 226 unique pollution grid points used. Interpolation adds spatial variation at a finer grain, but uncorrected standard errors would nevertheless overestimate how much independent variation there is in the pollution measure. Moreover, the actual pollution level is spatially correlated. Therefore I allow for clustering of errors among observations within an island group by month. The sample has ten island groups (Sumatra, Java, Sulawesi, Kalimantan, Bali, West Nusa Tenggara, East Nusa Tenggara, Irian Jaya, Maluku, and North Maluku).[9]

The estimating Equation 2 includes pollution in the month of birth ($Smoke_{jt}$) as well as lags of $Smoke_{jt}$ which measure exposure to pollution in utero, and leads that measure exposure after birth. Note that $Smoke_{jt}$ measures both prenatal and postnatal exposure, with the balance depending on when in the calendar month an individual is born (the Census did not collect the specific date of birth, only the month). It becomes difficult to separately identify each lag and lead with precision, so the main specification uses an average of the pollution level for the three months before the birth month ($PrenatalSmoke_{jt}$) and after the birth month ($PostnatalSmoke_{jt}$). The population-weighted mean values of *Smoke*, *PrenatalSmoke*, and *PostnatalSmoke* are 0.09, 0.10, and 0.07, as shown in Table 1. On average, the pollution index exceeds 0.75 on 5 percent of days.

During the months of the fires, August to October 1997, the mean aerosol index for Indonesia was 0.58. For the same months in 1996, the mean was 0.05. Similarly, the mean of *PrenatalSmoke* was 0.34 for the most affected cohorts (births in September 1997 to January 1998) while during the same months a year earlier, the mean was 0.04. These gaps are helpful when interpreting the regression coefficients and quantifying the impact of the fires.

The intensity of smoke varied across Indonesia. Figure 2a shows the coefficients on island-specific month fixed effects for Kalimantan and Sumatra, the hardest hit regions. The pollution was most severe during August to October 1997, both for Indonesia as a whole and in relative terms for Kalimantan and Sumatra, as shown. Kalimantan experienced another episode of smoke in early 1998 after the rainy season ended.

Figure 2b plots the corresponding island-specific month effects using log cohort size as the outcome variable. Intuitively, the identification strategy is to test whether in the areas most affected by the pollution, the size of cohorts that were born around the time of the pollution is abnormally low. The regression analysis uses not only this between-island variation but also within-island variation. Even using just this coarse variation, one can see in the point estimates the decline in cohort size for the affected island-months.

C. Other Variables

Several other variables are used either as controls or to examine differential effects of pollution (that is, as interaction terms). First, I construct a measure of the financial

9. The standard errors are almost identical if they are clustered on island, suggesting that serial correlation is not a major concern. However, using only ten clusters is not advisable since ''the cluster approach may be quite unreliable except in the case where there are many groups'' (Donald and Lang 2007).

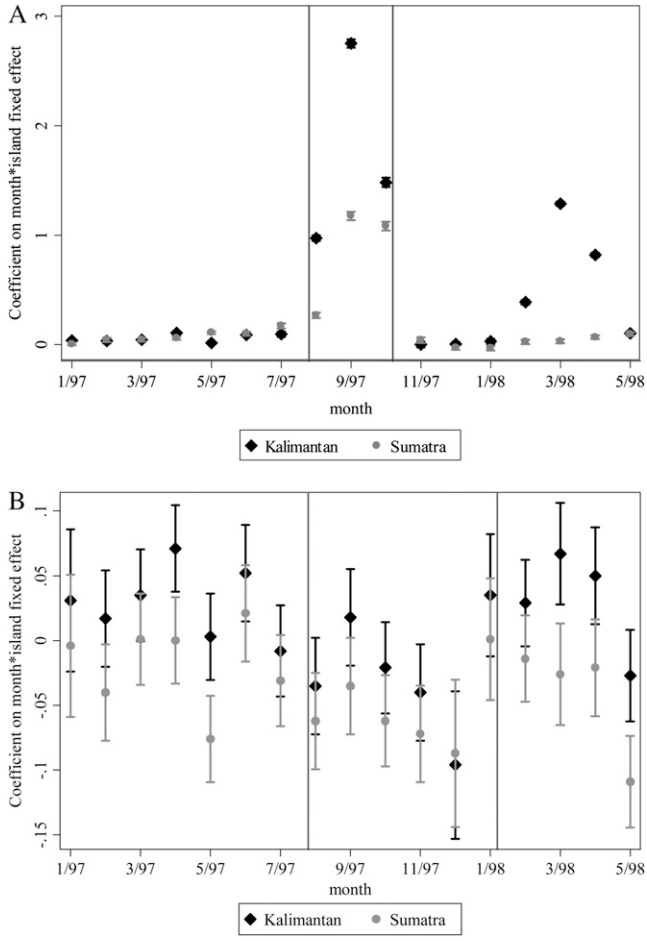

Figure 2

Pollution and Log Cohort Size by Month and Location

Note: *This figure plots coefficients and standard errors from a regression of A. pollution and B. log cohort size on subdistrict and month fixed effects and island-specific month fixed effects for Kalimantan and Sumatra. The month fixed effects for Kalimantan and Sumatra are plotted. The vertical lines delineate the three months of highest pollution in A and the six birth cohorts that experienced high pollution in the month of birth or third trimester in utero in B.*

crisis that hit Indonesia in late 1997. Cross-sectional variation in the crisis is measured as the 1996 to 1999 ratio of the median log food consumption per capita in a district; the value is higher in areas hit harder by the crisis. The consumption data are from the National Socioeconomic Survey (SUSENAS), a household survey conducted annually by the national statistics bureau. The survey is representative at the district rather than subdistrict level, so data are aggregated to the district. The data

appendix (Appendix 2) describes in more detail how the consumption measure is constructed. The national consumer price index for food is from the central bank and is used as a measure of temporal variation in the crisis. The interaction of these two variables is the crisis measure.

The cross-sectional consumption measure for 1996 is interacted with the pollution variables to examine how the effects of pollution differ for rich and poor areas. Healthcare measures such as doctors and maternity clinics per capita, as well as the type of fuel people cook with are from the 1996 Village Potential Statistics (PODES), a census of community characteristics. The PODES has an observation for each of over 66,000 localities, which I aggregate to the subdistrict level. In the analyses that use data from the PODES or SUSENAS, the sample size is 63,158 since some Census subdistricts could not be matched to the surveys.

To measure the extent of fires (as opposed to pollution) in an area, daily data on the location of ''hot spots'' are used. The data are from the European Space Agency, which analyzed satellite measurements of thermal infrared radiation to locate fires. To control for rainfall, I use monthly rainfall totals from the Terrestrial Air Temperature and Precipitation data set and match each subdistrict to the nearest node on the rainfall data set's 0.5 degree latitude by 0.5 degree longitude grid. Finally, I use additional variables from the Census including mother's education and whether a locality is rural or urban.

IV. Results

A. Relationship Between Exposure to Smoke and Mortality

Table 2, Column 1, presents the relationship between cohort size and exposure to smoke. The independent variables are *Smoke*, which is pollution in the month of birth, *PrenatalSmoke* which is pollution in the three months before birth, and *PostnatalSmoke* which is pollution in the three months after birth. Prenatal exposure to pollution decreases the survival rate of fetuses, infants, and children: *PrenatalSmoke* has a coefficient of -0.035 that is statistically significant at the 1 percent level. The coefficient for *Smoke* is very close to zero, while the coefficient for *PostnatalSmoke* is -0.014 though statistically insignificant. Standard errors are clustered within an island-month.[10] In Column 2, when *PrenatalSmoke* is the only variable in the regression (besides fixed effects), the coefficient is similar to that in Column 1.[11]

The regressions are weighted by population, but as a specification check, the unweighted regression is shown in Column 3. The results are similar, with coefficients and standard errors that are larger in magnitude. The larger standard errors suggest that, given heteroskedasticity, weighting by population improves efficiency. The other rationale for weighting is that the dependent variable measures proportional rather than absolute changes in cohort size. Columns 4 and 5 consider alternative

10. The standard error for *PrenatalSmoke* is 0.014 if one clusters on island instead of island-month.

11. See Appendix Table A2 for an instrumental variable estimate of the effect of *PrenatalSmoke* on cohort size that uses only coarse variation in pollution. The instrument for *PrenatalSmoke* is a dummy for Kalimantan or Sumatra interacted with a dummy for September 1997 to January 1998 (first stage F-statistic of 45.6). The IV coefficient is -0.037.

Table 2
Relationship Between Air Pollution and Cohort Size

	Statistic Used for Smoke Measures							
Dependent variable: Log cohort size	Median (1)	Median (2)	Median (3)	Mean (4)	% high-smoke days (5)	Median (6)	Mean (7)	% high-smoke days (8)
Smoke	−0.0005		−0.005	−0.001	−0.010	0.001	0.018	0.035
	(0.006)		(0.008)	(0.007)	(0.020)	(0.009)	(0.014)	(0.036)
Prenatal smoke	−0.035***	−0.032***	−0.048***	−0.032**	−0.085**			
($Smoke_{t\text{-}1,2,3}$)	(0.012)	(0.011)	(0.015)	(0.013)	(0.033)			
Postnatal smoke	−0.014		−0.017	−0.016*	−0.042*			
($Smoke_{t+1,2,3}$)	(0.009)		(0.013)	(0.010)	(0.025)			
$Smoke_{t\text{-}1}$						−0.010	−0.028*	−0.069*
						(0.009)	(0.016)	(0.040)
$Smoke_{t\text{-}2}$						−0.023***	−0.006	−0.035
						(0.008)	(0.013)	(0.038)
$Smoke_{t\text{-}3}$						−0.003	−0.005	0.005
						(0.013)	(0.015)	(0.030)
$Smoke_{t+1}$						−0.010	−0.019	−0.030
						(0.009)	(0.014)	(0.031)

$Smoke_{t+2}$						−0.005	−0.003	−0.034
						(0.008)	(0.014)	(0.034)
$Smoke_{t+3}$						0.001	−0.001	0.010
						(0.009)	(0.012)	(0.031)
Observations	67,454	67,454	67,454	67,454	67,454	67,454	67,454	67,454
Weighted?	Y	Y	N	Y	Y	Y	Y	Y
Subdistrict and month FEs?	Y	Y	Y	Y	Y	Y	Y	Y

Note: Each observation is a subdistrict-month. Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$, * indicates $p < 0.10$. Observations are weighted by the number of individuals enumerated in the Census who reside in the subdistrict and were born in the year before the sample period, except in Column 3 which is unweighted.

monthly pollution measures, first, the mean rather than median of the daily pollution values and, second, the proportion of days with high pollution (aerosol index above 0.75). Mean pollution gives nearly identical results as the median value, with postnatal exposure now having a negative impact on cohort size that is marginally significant. For the proportion of days with high pollution, the point estimate implies that when there are three additional high-smoke days in a month (an increase of ten percentage points), cohort size decreases by 0.85 percent.

Exposure to pollution in utero is associated with a decrease in fetal, infant, and child survival. To assess the aggregate magnitude of the effect, note that *PrenatalSmoke* was higher by 0.30 during September 1997 to January 1998 compared to the same calendar months a year earlier; this five-month period are the cohorts for whom *PrenatalSmoke* includes a month during the fires. Multiplying that gap by the coefficient of -0.035 implies that the fires led to a 1.1 percent decrease in cohort size. A more precise way to estimate the total effect is to use the coefficient for *PrenatalSmoke* and calculate what the population would have been for each subdistrict if during the period during and immediately after the fires, *PrenatalSmoke* had taken on its value from 12 months earlier. Aggregated over the five months for the 3,751 subdistricts, this calculation similarly implies a population decline of 1.2 percent, or 15,600 missing children.[12] Indonesia's baseline under-3 mortality rate was roughly 60 per 1,000 live births at this time.[13] If the effect of pollution was due exclusively to infant and child deaths, the estimates would represent a 20 percent effect; if half of the effect was due to fetal deaths, the coefficient would imply a 10 percent effect on under-three mortality.[14]

Figure 3 shows the nonparametric relationship between third-trimester exposure and cohort size. The effect of pollution is linear for the most part. There appears to be a somewhat steeper relationship at high levels of pollution, but the nonlinearities are statistically insignificant when estimated parametrically with a spline or quadratic term.

The next regressions use the pollution level in each of the three months preceding and following birth, rather than aggregated for a quarter. Table 2, Column 6, reports the results using the median pollution level. For prenatal exposure (lags of *Smoke*), the effect is strongest two months before the month of birth. For postnatal exposure (leads of *Smoke*), the effect is strongest immediately after birth, though the estimates are imprecise. The next two columns repeat the exercise using the month's mean pollution and the proportion of days that have high pollution. The general pattern of the coefficients for postnatal pollution remains the same, but the pattern for prenatal exposure changes. For the mean pollution level or number of high-smoke days (Columns 7 and 8), exposure in the month immediately before the month of birth now has the strongest negative

12. The estimates using high-smoke days imply a 1.0 percent aggregate effect. (The mean of the prenatal high-smoke variable is 0.131 during the 1997-98 episode and 0.006 for the same calendar months a year earlier, and the coefficient in Table 2, Column 4, is -0.085.)

13. The government estimates of under-one and under-five mortality rates at this time are 5 percent and 7 percent, respectively. I assume that half of deaths between age one and five occur before age three.

14. The welfare implications of pollution-induced mortality depend on how long individuals otherwise would have lived. One can calculate using the child mortality rate that in the extreme "havesting" scenario, all deaths between ages three and six or seven would have to have been pushed forward to the time of the fires. Moreover, by most standards, the shortening of children's lives by even a few years is a significant welfare loss.

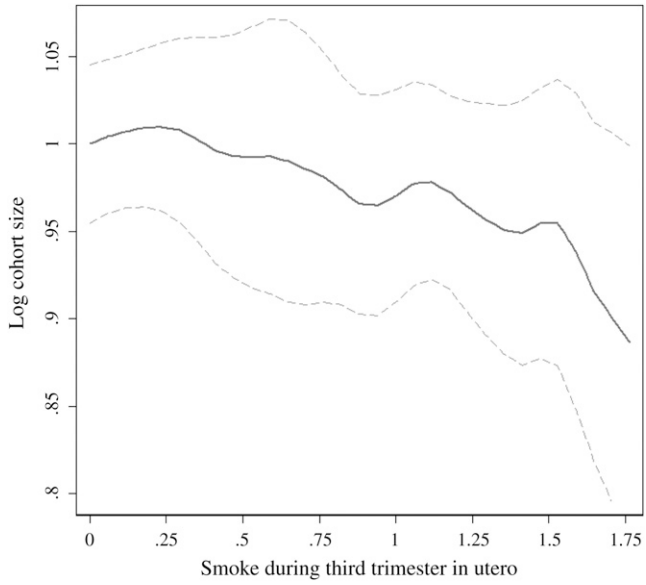

Figure 3

Kernel Regression of Log Cohort Size on Prenatal Exposure to Pollution

Note: The solid line is the estimated relationship between log cohort size and pollution (*PrenatalSmoke*). The dashed lines are the bootstrapped 95 percent confidence interval, with errors clustered within an island-month. The model estimated is a locally weighted nonparametric regression of log cohort size on pollution conditional on year and district fixed effects, following Robinson (1988). Log cohort size has been offset by a constant so that its value is one at an aerosol index of zero.

relationship with cohort size. One interpretation is that at different points during gestation, fetuses are more vulnerable to sustained exposure to pollution versus extreme levels of pollution. A more likely interpretation is that there is not enough precision to determine at this level of detail how the timing of exposure affects survival.[15] Thus, for the rest of the analysis, I focus on the three-month measures of prenatal and postnatal exposure. The results are similar using two-month measures.

B. Effect of Smoke on Mortality versus Alternative Hypotheses

The results in Table 2 suggest that exposure to smoke in utero causes early-life mortality. This section considers other possible explanations for the results.

1. Migration

The Census identifies respondents by their subdistrict of current residence, but exposure to pollution depends on where one resided during the fires. Migration could be a

15. The month-by-month patterns, unlike the results with the three-month measures, are also somewhat sensitive to using a different sample period or a different threshold for high-smoke days.

reason that cohorts with the highest prenatal exposure to pollution are smaller if women in the third trimester of pregnancy were especially likely to migrate away from high-pollution areas, either while pregnant or after giving birth. Fortunately, the Census collects data on the district (though not subdistrict) where an individual was born and where he or she lived five years earlier, which enable one to probe this concern.[16]

To examine the extent of pollution-induced migration that occurs *after* birth, I repeat the main analysis by district of birth. Cohort size is aggregated to the district level, and the pollution measure for the district is a population-weighted average of the subdistrict measure. The regression is weighted by the district population in the two years prior to the sample period. For comparison, Column 1 of Table 3 presents results by district of residence, and Column 2 presents results by district of birth. The results are nearly identical to each other, as well as to the subdistrict-level analysis, in terms of both point estimates and precision. Between-district migration after the birth of the infant is not the likely explanation for the relationship between pollution and cohort size.

Pollution-induced migration also may take place *before* the infant is born. If some women spent most of their third trimester of pregnancy in the hardest-hit areas but migrated away before giving birth, then neither place of residence in 2000 nor place of birth would accurately reflect the fetus's location during the fires. While the Census did not ask respondents where they resided in August to October 1997, it did ask where they lived in 1995. As long as people do not migrate across districts repeatedly, this measure should be a good proxy for where pollution-induced migrants lived at the time of the fires. To test for migration that occurs before birth, I match infants to their mothers as described in the data appendix and repeat the estimate by the district where the mother resided in 1995. The results, shown in Column 3, are unchanged from the earlier estimates. In sum, migration, either before or after birth, does not seem to account for the negative relationship between exposure to pollution and cohort size.[17]

2. *Fertility*

The empirical approach interprets decreases in *ln*(*CohortSize*) as increases in early-life deaths, but there would also be fewer survivors if the number of births decreased. It seems unlikely that conceptions declined nine months before the fires with a spatial pattern matching the pollution, but this concern also can be tested more directly by constructing a measure of predicted births. First, I measure the percentage of women of each age who give birth, using a time period not in the sample (namely, the youngest cohorts in the Census, those born in 1999 and 2000). I then apply these birth rates to the demographic composition of each district-month in the sample. This

16. For 9 percent of the sample, district of residence differs from district of birth, for 7 percent it differs from mother's residence five years earlier, and for 12 percent it differs from one or the other.

17. Within-district migration is unlikely to be driving the results since there is very little within-district variation in pollution, and most of it derives from interpolation so is noisy. In a model with district-month fixed effects, the coefficient for *PrenatalSmoke* is −0.013, smaller than in the main specification (Table 2, Column 1), and imprecise, suggesting that between-district variation is dominant in the main estimates.

Table 3
Distinguishing Between Mortality and Migration

	District of Residence Versus Birthplace Versus Mother's 1995 Residence		
Dependent variable: Log cohort size	Residence (1)	Birthplace (2)	Mother's 1995 residence (3)
Smoke	−0.002	0.002	0.002
	(0.006)	(0.006)	(0.006)
Prenatal smoke	−0.035***	−0.037***	−0.038***
	(0.012)	(0.012)	(0.012)
Postnatal smoke	−0.013	−0.015	−0.016
	(0.010)	(0.010)	(0.010)
Observations	5,829	5,829	5,829
Fixed effects	month, district	month, district	month, district

Note: Each observation is a district-month. Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$, * indicates $p < 0.10$. Observations are weighted by the number of individuals enumerated in the Census who reside in the district and were born in the year before the sample period.

gives a predicted number of births based on demographic shifts. (See the data appendix for further details.) Table 4, Column 1, shows the results when *ln*(*PredictedBirths*) is included as a control variable. The coefficient of survivors on births is predicted to be slightly less than 1. Because the measure is noisy especially after conditioning on subdistrict and month indicators, the estimate is likely to suffer from attenuation bias. The estimated coefficient on predicted births is less than but statistically indistinguishable from 1. More importantly, the coefficients on the pollution variables are essentially unchanged with this control variable included. Fluctuations in fertility caused by demographic shifts do not appear to be a confounding factor in the analysis.[18]

3. Preterm Births

Another concern is that the missing children are not deaths but instead are an artifact of changes in gestation length. Exposure to pollution may have induced preterm delivery which is often associated with traumatic pregnancies. The reason this mechanism could conceivably generate the results is that it is *prenatal* exposure that has a strong negative relationship with cohort size. Consider August 1997, the month the fires started. Pollution levels were high in August, and the value of *PrenatalSmoke* for August is low since there was no significant smoke in May, June, or July. In

18. Appendix Table A3 addresses another potential concern about fertility, namely that the seasonality of births or deaths could differ for areas more affected by the pollution, generating a spurious result. The results are robust to restricting the sample to the months with high *PrenatalSmoke* plus the same calendar months one year earlier.

Table 4
Alternative Hypotheses

Dependent variable: Log cohort size	Control for Predicted Fertility (1)	Excluding August 1997 (2)	SUSENAS and PODES subsample (3)	Control for Financial Crisis (4)	Excluding Areas with Fires (5)	Control for Fires (6)	Control for Rainfall (7)
Smoke	0.001	0.001	0.002	0.002	0.003	0.004	0.001
	(0.006)	(0.006)	(0.006)	(0.006)	(0.011)	(0.006)	(0.006)
Prenatal Smoke	−0.035***	−0.036***	−0.032***	−0.032***	−0.035**	−0.032**	−0.032**
	(0.012)	(0.012)	(0.011)	(0.011)	(0.018)	(0.014)	0.013
Postnatal smoke	−0.014	−0.009	−0.012	−0.012	0.016	−0.005	−0.014
	(0.009)	(0.010)	(0.009)	(0.009)	(0.014)	(0.011)	(0.009)
Ln(predicted births)	0.875						
	(0.696)						
Financial crisis				−0.049			
				(0.038)			
Any fires						−0.004	
						(0.010)	
Prenatal any fires						0.007	
						(0.017)	
Postnatal any fires						−0.004	
						(0.014)	
Intense fires						−0.028*	
						(0.016)	
Prenatal intense fires						−0.017	
						(0.025)	

Postnatal intense fires						−0.021	
						(0.029)	
Rainfall							−0.023
							(0.033)
Observations	67,454	63,703	63,158	63,158	52,646	67,454	67,454
Subdistrict and month FEs?	Y	Y	Y	Y	Y	Y	Y

Note: Each observation is a subdistrict-month. Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$, * indicates $p < 0.10$. Observations are weighted by the number of individuals enumerated in the Census who reside in the subdistrict and were born in the year before the sample period. Predicted Births is constructed using the fertility rate by age and the number of women of different child-bearing ages within a district, as described in the data appendix. The financial crisis variable is standardized to have a mean of 0 and standard deviation of 1 for the sample. Areas without fires are those with fewer than 20 fire-days over the entire period. Any fires is an indicator of any fires and intense fires is an indicator of at least 10 fire-days in the month. The rainfall variable is subdistrict-level rainfall for the 1997 drought months of June to November divided by the 1990–95 average for those calendar months, interacted with a dummy for being in a birth cohort between September 1997 and January 1998.

September, in contrast, *PrenatalSmoke* is high since it incorporates the pollution in August. If infants due in September were instead born in August, then births would have shifted from a high-*PrenatalSmoke* month to a low-*PrenatalSmoke* month, generating a negative relationship between *PrenatalSmoke* and cohort size that is unrelated to mortality. To test the preterm-birth hypothesis, I repeat the analysis dropping August 1997 from the sample. If the above hypothesis were correct, the coefficient on *PrenatalSmoke* would become less negative compared to the baseline results. As shown in Table 4, Column 2, this does not occur. The coefficients are nearly identical between the full sample and the subsample, contrary to what one would expect if the pollution had induced preterm births but had not affected infant survival.[19]

The effect of pollution on cohort size is not due to preterm births *instead of* being due to fetal and infant deaths. Note, however, that pollution may have caused infant deaths precisely by inducing premature births (which put infants at greater risk of death); that is, preterm delivery is potentially an important channel through which exposure to pollution led to mortality.

4. Financial Crisis

The Indonesian financial crisis began shortly after the fires, as shown in Figure 4. To verify that the analysis is not attributing to air pollution deaths that were caused by the crisis, a measure of the financial crisis is added to the model. No monthly subdistrict-specific data on the crisis were collected, to my knowledge, so I construct a measure of the crisis by interacting a cross-sectional measure, the inverse ratio of median income (consumption) at the height of the crisis in 1999 to median income before the crisis in 1996, and a time-series measure, the consumer price index for food. The regression results can be anticipated by noting that the cross-sectional correlation between the crisis measure and pollution in September 1997 (peak of the fires) is 0.03; the spatial patterns of the crisis are not similar to the spatial patterns of pollution. For regressions that use variables from the SUSENAS or PODES surveys, a slightly smaller sample of subdistricts is used due to data availability. Table 4, Column 3, shows the regression results for the baseline model and confirms that the subsample is similar to the full sample, with a coefficient on *PrenatalSmoke* of -0.032. Column 4 then shows the results when the crisis measure for the month of birth is included as a control variable. The estimated effect of *PrenatalSmoke* remains -0.032. The crisis measure has been normalized to have a mean of zero and standard deviation of one for the sample, so the coefficient implies that a one standard deviation increase in the crisis is associated with a 4.9 percent smaller cohort, though the coefficient is statistically insignificant (and moreover could be due to migration rather than mortality).[20,21]

19. Appendix Table 3 restricts or expands the sample to other time periods, and the results are robust to this change. One noteworthy finding is that the estimated effect of *PrenatalSmoke* is smaller when the window extends more than eight months after the fires, suggesting that the fires may have reduced fertility.

20. Since the crisis accelerated a few months after the fires, I also estimated models that control for the crisis measure for the three months following the month of birth. This generates more variation in the crisis measure during the period of interest. The estimated effect of *PrenatalSmoke* is unchanged.

21. Rukumnuaykit (2003) finds a 3 percent increase in infant mortality in 1997-98 in the Indonesia Family Life Survey, which is interpreted as due to the financial crisis, drought and smoke.

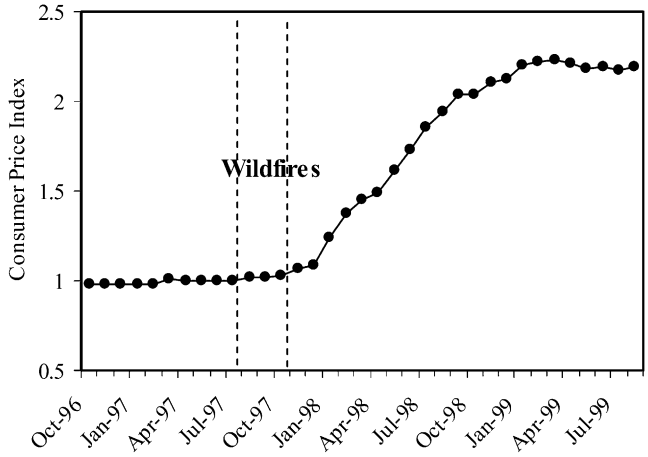

Figure 4
Timing of the Fires and the Financial Crisis

5. Effect of Pollution versus Effect of Fires or Drought

Another interpretation of the results is that they represent reduced-form effects of the fires rather than effects of specifically air pollution. The regressor is the pollution level, but the smoke affected places nearby the sites of fires, and the fires could have caused mortality through income effects, degraded food supply, and other channels. To separate the effect of pollution from other effects of the fires, I use data on where the fires occurred. I calculate the number of fire-days occurring in or near a subdistrict based on satellite data on ''hot spot'' locations and durations. *Firedays* is the duration of each fire summed over all fires within 50 km of the subdistrict center. First, I examine the effects of pollution in areas that did not experience extensive fires. In essence, the identification comes from fires in neighboring areas and the direction that the winds blew the pollution. In Table 4, Column 5, the sample is restricted to subdistricts where fewer than 20 fire-days occurred over the sample period, which eliminates 22 percent of subdistricts, predominantly in Kalimantan and Sumatra.[22] The coefficient on *PrenatalSmoke* on log cohort size remains −0.035 for these areas that experienced only the pollution from the fires. Next, I include measures of fire prevalence as regressors. The fire-days variable is highly skewed, so I use two indicator variables, one for whether there were any fire-days in the subdistrict-month (sample mean of 0.16) and a second for whether there were intense fires, defined as at least ten fire-days during the month (sample mean of 0.03). In Column 6, the fires variable and intense fires

22. Because of measurement error in the hotspot data, eliminating any subdistrict with at least one fire-day would eliminate more than two-thirds of subdistricts, which is inconsistent with the actual geographic extent of the fires. Fewer than 22 percent of subdistricts were probably affected, so the results shown are conservative. They are similar if other thresholds are chosen.

variable in the month of birth, averaged over the three months before birth (prenatal exposure), and averaged over the three months after birth (postnatal exposure) are included as regressors. The effect of *PrenatalSmoke* is −0.032, nearly identical to earlier estimates, which supports the interpretation that air pollution is the cause of the increase in early-life mortality. There is also some evidence that intense fires in the month of birth are associated with a decrease in cohort size, suggesting that fires may have an additional effect on survival (or migration) through channels besides pollution, but the effect size is relatively small. The coefficient of −0.028 implies that intense fires are associated with a 0.25 percent decrease in cohort size.

Another hypothesis is that the effects are due to the drought. There was below-normal rainfall throughout Indonesia in 1997, not just in areas affected by pollution; given that month fixed effects are included, drought seems unlikely to be driving the results. Nevertheless, I test directly for effects of the drought and in a way that stacks the cards in favor of finding that the drought is driving the results. Monthly rainfall for the subdistrict is measured relative to the 1990–95 average for that calendar month, and I construct the subdistrict-level mean for the months of June to November 1997, when the drought occurred. The mean of this variable is 0.48. I then control for this measure of rainfall interacted with an indicator for being in a birth cohort between September 1997 and January 1998. In other words, the drought is assumed to affect specifically the cohorts that I find are harmed by the pollution. As shown in Table 4, Column 7, the coefficients for the pollution variables are essentially unaffected when rainfall is included as a control. The coefficient for rainfall is statistically insignificant and small. The results are similar controlling for contemporaneous rainfall in the month or rainfall with a one to nine-month lag. One might worry that drought reduces fertility, but the drought began in June, and the affected cohorts were conceived by April. Moreover, when rainfall nine months prior to birth is added as a control variable, again, rainfall has a small and insignificant effect on cohort size, and the effect of prenatal pollution is unchanged. The changes in cohort size do not seem to be due to rainfall shortages.

C. Effects by Gender and Income

1. Effects by Gender

This subsection examines how the mortality effects of pollution vary across groups. I first test whether there are differential effects for boys and girls. In Table 5, Column 1, the number of surviving boys and girls are totaled separately, each observation is a subdistrict-month-gender, and the three pollution variables are interacted with a dummy for male. The coefficients are imprecise but follow an interesting pattern. The male interaction terms are positive for contemporaneous and postnatal smoke, but negative for prenatal smoke. The more negative effect for boys in utero (30 percent larger effect) is consistent with findings in the literature that male fetuses are less physiologically robust than female fetuses and have more delayed lung development (Jakobovits 1991). The more negative effect for girls in the month of birth and after birth could reflect physiological differences, but it is also consistent with gender discrimination. If parents are more likely to take steps to minimize a newborn son's

exposure to pollution or to seek medical treatment for his respiratory infection, for example, then one would expect the effects of postnatal pollution to be stronger for girls. However, these interpretations should be treated with caution since the gender differences are not statistically significant.

2. Effects by Income

The next estimates test whether the effects of pollution are more pronounced in poorer places. This type of heterogeneity could arise if the poor effectively are exposed to more pollution, for example, because they spend more time outdoors doing strenuous work or are less likely to evacuate the area. It could also arise if the same amount of effective pollution leads to bigger health effects for the poor, for example, because they have lower baseline health, making them more sensitive to pollution, or have less access to healthcare to treat the health problems caused by the pollution.

Column 2 of Table 5 uses food consumption as a proxy for income to examine this hypothesis, interacting the pollution measures with a dummy variable for whether the district's median log consumption in 1996 is above the 50th percentile among all subdistricts. All three of *Smoke*, *PrenatalSmoke*, and *PostnatalSmoke* are associated with smaller cohorts for the bottom half of the consumption distribution, and the interaction terms for the top half of the distribution are large and positive. The weighted average of the coefficients for the bottom and top halves of the distribution would be more negative than the average effect found earlier, however. The reason is that month effects vary with income. As has been documented in the demography literature, seasonality in fertility tends to be stronger and qualitatively different in poorer areas (Lam and Miron 1991). Thus, Column 3 includes separate month fixed effects for the top and bottom halves of the consumption distribution. The results are qualitatively similar to those in Column 2. The effect of prenatal exposure is large and negative when consumption is below the median. In these areas, postnatal exposure is also statistically significant, with an effect size about 60 percent that of prenatal exposure. Each of the interaction coefficients for districts with above median consumption is positive, and in the case of *PrenatalSmoke*, significant at the 1 percent level. The effect of a one unit change in *PrenatalSmoke* is -0.06 for the top half of the distribution and -0.13, or over twice as large, for the bottom half. Average log consumption is 0.4 log points larger in the top half of the distribution compared to the bottom half, so another way to view the results is that when consumption increases by 50 percent ($e^{0.4}$), the effect size decreases by 50 percent.

The dependence of seasonal patterns on income suggests that including separate month effects for the two halves of the consumption distribution might be the preferred specification even for estimating the average effect. In addition, for the reasons explained by Deaton (1995), given the heterogeneous effects, the average effect should be calculated by separately estimating the effect by consumption level and then averaging. This amounts to averaging the coefficients in Column 3, weighted by the population in each half of the consumption distribution. As shown in Column 4, the average effect for prenatal smoke is then -0.090 and

Table 5
Effects by Gender and Income

	By gender	By income (log consumption) of the district						
Dependent variable: Log cohort size	(1)	(2)	(3)	(4)	Top quartile	3rd quartile	2nd quartile	Bottom quartile
						(5)		
Smoke	−0.008	−0.060***	−0.024	−0.013	−0.004	−0.011	−0.028	0.002
	(0.007)	(0.021)	(0.016)	(0.017)	(0.009)	(0.010)	(0.024)	(0.045)
Prenatal smoke	−0.030**	−0.158***	−0.129***	−0.090***	−0.058***	−0.076***	−0.094**	−0.121**
	(0.012)	(0.037)	(0.028)	(0.015)	(0.018)	(0.017)	(0.047)	(0.061)
Postnatal smoke	−0.019*	−0.158***	−0.047*	−0.035**	−0.025	−0.040***	−0.046	0.009
	(0.010)	(0.027)	(0.024)	(0.019)	(0.016)	(0.014)	(0.032)	(0.052)
Male	0.014***							
	(0.003)							
Smoke * male	0.016***							
	(0.005)							
Prenatal smoke * male	−0.009							
	(0.007)							
Postnatal smoke * male	0.010							
	(0.006)							
Smoke * high consumption		0.066***	0.017					
		(0.021)	(0.014)					
Prenatal smoke * high consumption		0.127***	0.072***					
		(0.038)	(0.027)					

Postnatal smoke * high consumption		0.161*** (0.026)	0.017 (0.014)		
Observations	134,734	63,158	63,158	63,158	63,158
Fixed effects included	subdistrict, month	subdistrict, month	subdistrict, month * high cons.	subdistrict, month * high cons.	subdistrict, month*quartile of log consumption

Note: Each observation is a subdistrict-month. Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$, * indicates $p < 0.10$. High consum. is an indicator that equals 1 if the district's median log food consumption is above the sample median. Observations are weighted by the number of individuals enumerated in the Census who reside in the subdistrict and were born in the year before the sample period. Column 4 reports the average of the coefficients in Column 3, weighted by the population in each half of the consumption distribution; the standard error and the joint significance of the linear combination of coefficients is shown.

the coefficient for postnatal smoke is −0.035, both considerably larger than seen earlier in Table 2.

Next, I further break down the income distribution into quartiles (and include month-quartile fixed effects). Column 5 shows the separate coefficients by quartile, estimated as one regression. The point estimate on *PrenatalSmoke* becomes more negative moving from higher to lower quartiles. The results are not very precise, though, and the *PrenatalSmoke* coefficients for different quartiles are not statistically distinguishable from one another. The coefficients for the other smoke variables are also imprecise, especially for the bottom two quartiles, and the point estimates do not monotonically decline with consumption. Above- versus below-median consumption, as opposed to a linear interaction term, is therefore used below to parsimoniously characterize the heterogeneous effects by income.

3. Effects by Urbanization, Wood-Stove Use, Healthcare, and Mother's Education

This subsection tests some hypotheses about why there is an income gradient in the health effects of pollution. The tests are merely suggestive because the measures used could be correlated with omitted variables and data are available to test only a limited number of hypotheses.

One possibility is that urban areas experience smaller effects from the fires than rural areas, generating the heterogeneity by income. Urbanization would only be a proximate cause, but one might think that in urban areas, housing stock is less permeable, healthcare is better, there is less outdoor work, or there are more effective public advisories urging people to stay indoors, for example. On the other hand, pollution from the fires may have been particularly noxious in cities where it mixed with industrial pollution from cars and factories. Column 1 of Table 6 interacts the pollution measures with the proportion of the subdistrict population that lives in urban localities (based on those born in the year before the sample period). Only the coefficients for *PrenatalSmoke* and its interaction terms are reported, but *Smoke*, *PostnatalSmoke,* and their interactions are also included in the regressions. The effects of pollution do not vary by urbanization level, suggesting that the effects described above may have offset each other. In unreported results, I also find that children whose mothers work in agriculture do not experience larger effects.

Next I test whether the effects depend on the prevalence of wood-burning stoves. If the health impact of pollution is convex in exposure, those who have daily exposure to indoor air pollution could suffer more acutely from the wildfires. For each village or town, data are available on whether the majority of people used wood or other biomass as their primary cooking fuel in 1996. I construct for each subdistrict the population-weighted average of this measure, which serves as a crude measure of the proportion of people in the subdistrict who use wood as their cooking fuel. The mean of the variable is 0.64. As shown in Column 2 of Table 6, wood fuel use is strongly associated with more negative effects from any given level of exposure to the wildfire smoke. A 20 percentage point decrease in wood fuel use reduces the net effect of prenatal pollution by 0.03. By comparison, moving from the bottom half to the top half of the consumption distribution (50 percent

Table 6
Effects By Urbanization, Wood Fuel Use, and Healthcare Sector

Dependent variable: log cohort size	(1)	(2)	(3)	(4)	(5)
Prenatal smoke	−0.121***	0.015	−0.115***	−0.113***	−0.007
	(0.028)	(0.032)	(0.027)	(0.028)	(0.025)
Prenatal smoke * urbanization	−0.013				
	(0.013)				
Prenatal smoke * wood fuel use		−0.155***			−0.120***
		(0.036)			(0.026)
Prenatal smoke * maternity clinic			0.030***		0.011**
			(0.009)		(0.005)
Prenatal smoke * doctors				0.048***	0.016
				(0.015)	(0.013)
Prenatal smoke * high consumption	0.071***	0.048*	0.058**	0.052**	0.044*
	(0.027)	(0.025)	(0.025)	(0.025)	(0.025)
Observations	63,158	63,158	63,158	63,158	63,158
Subdistrict and month FEs?	Y	Y	Y	Y	Y

Note: Each observation is a subdistrict-month. Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$, * indicates $p < 0.10$. All regressions also include Smoke and Postnatal Smoke and their interactions with the relevant variables for each column. Urbanization is the proportion of the population in urban localities and is based on 1994 to 1996 birth cohorts. Wood fuel use is an approximate measure of the proportion of people in the subdistrict who cook with wood fuel rather than kerosene and gas. Health variables are normalized to be mean zero, standard deviation one for the sample. High consumption is an indicator that equals one if the district's median log food consumption is above the sample median. Observations are weighted by the number of individuals enumerated in the Census who reside in the subdistrict and were born in the year before the sample period.

increase in consumption) reduces the effect of prenatal pollution by 0.07. The obvious caveat is that use of wood-burning stoves could be proxying for an omitted variable.

I next examine whether the effects vary with the availability of healthcare. A good healthcare system could lead to improved baseline health of the mother and child and to better medical treatment of morbidities caused by the pollution. Columns 3 and 4 of Table 6 present the results when interactions of the pollution measures with maternity clinics and doctors per capita are successively included. The per capita measures, which are for 1996, have been normalized to be mean 0, standard deviation 1. In areas with more maternity clinics or doctors, pollution has a significantly smaller effect on cohort size. The net coefficient for *PrenatalSmoke* is smaller by 0.03 in an area with one standard deviation above the average number of maternity clinics compared to the average area, and smaller by 0.05 in an area with one standard deviation above average doctors per capita.[23]

In Column 5, the interactions with wood fuel use, doctors, and maternity clinics are estimated in a single regression. The effects of in utero pollution continue to be considerably larger when wood fuel use is higher. In addition, the interaction terms for maternity clinics and doctors remain positive and, for maternity clinics, statistically significant, though smaller in magnitude than when estimated separately. As noteworthy, these interaction terms do not fully explain the differential effects by income.

Table 7 examines how the effects vary by mother's education. Mother's education is important per se as a factor that might affect children's health, and it is also the best available individual-level (that is, Census) measure of SES. I match each infant to his or her mother and create a dummy variable for whether the mother has completed junior high. The sample mean is 0.38. In Column 1, there are two observations for each subdistrict-month, the number of surviving children for high-education and for low-education mothers. The three pollution variables are interacted with an educated-mother dummy, as are the month and subdistrict dummies. The main effect for prenatal pollution remains negative and statistically significant. The interaction terms are small and insignificant. Surprisingly, the mortality effects of pollution do not seem to vary by mother's education. Next I examine whether differences in average maternal education across areas explain why the effects of pollution are smaller in richer areas. In Column 2, the pollution variables are interacted with the percentage of children in the subdistrict born to educated mothers. The regression is fully interacted; that is, month fixed effects are interacted with the subdistrict-level measure of maternal education. The interaction of the pollution variables with mother's education are positive but not large and generally not statistically significant, though there is some evidence that postnatal pollution has detrimental effects in areas with fewer educated mothers. In Column 3, I also include the pollution variables interacted with a dummy for above-median consumption and interact the month effects with this consumption measure. Maternal education does not explain the heterogeneity in effect size between more and less developed areas.

23. Frankenberg (1995) examines within-village changes over time in the health sector and finds that overall infant mortality decreases when a village acquires more maternity clinics or doctors.

Table 7
Effects by Mother's Education

	Measure of Mother's Education		
Dependent variable: Log cohort size	Individual-Specific	Subdistrict Average	
	(1)	(2)	(3)
Smoke	0.002	−0.004	−0.013
	0.007	0.012	(0.017)
Prenatal smoke	−0.041***	−0.054***	−0.113***
	(0.013)	(0.018)	(0.029)
Postnatal smoke	−0.010	−0.036**	−0.044
	(0.012)	(0.018)	(0.028)
Smoke * educated mother	−0.007		
	(0.005)		
Prenatal smoke * educated mother	0.007		
	(0.008)		
Postnatal smoke * educated mother	−0.010		
	(0.009)		
Smoke * percent educated mothers		0.003	−0.009
		(0.020)	(0.020)
Prenatal smoke * percent educated mothers		0.021	−0.034
		(0.029)	(0.028)
Postnatal smoke * percent educated mothers		0.057*	0.053*
		(0.034)	(0.034)
Smoke * high consumption			0.018
			(0.014)
Prenatal smoke * high consumption			0.099***
			(0.026)
Postnatal smoke * high consumption			0.011
			(0.034)
Observations	134,908	63,158	63,158
Fixed effects included	subdistrict * educated mother, month * educated mother	subdistrict, month * % educated mothers	subdistrict, month * high consum., month * % educated mothers

Note: Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$, * indicates $p < 0.10$. In Column 1, each observation is a subdistrict-month-education category. Educated mothers are defined as those who have completed junior high. In Columns 2 and 3, each cell is a subdistrict-month, and percent educated mothers is the subdistrict average over the sample period. When interacted with month fixed effects, percent educated is standardized to mean 0. High consumption is an indicator that equals 1 if the district's median log food consumption is above the sample median.

The factors examined are unable to fully explain why prenatal exposure to pollution has a smaller effect on mortality in higher income areas. Even after controlling for wood-stove pollution, access to healthcare, and maternal education, there remains a positive coefficient for the interaction of pollution and high consumption. With better measures of indoor pollution, healthcare, and parental education, one might be able to explain more of the heterogeneity by income. There are also several channels not tested here. Mothers and newborns in poor areas might be less healthy to begin with because of poor nutrition. Dwellings in poor areas might be more permeable to smoke, allowing pollution from the wildfires to contaminate indoor air quality. Behavioral responses might differ by income, with those in richer areas being more likely to stay indoors, avoid strenuous activity, or temporarily evacuate to less affected areas, for example.[24] But regardless of the underlying causes, the findings suggest that the mortality costs of pollution are disproportionately borne by the poor.[25]

V. Conclusion

Air pollution from land fires that engulfed Indonesia in late 1997 led to over 15,600 child, infant, and fetal deaths, or a 1.2 percentage point decrease in survival for the affected cohorts. This paper uses the abrupt timing of the pollution and the spatial variation across Indonesia to identify these effects. The paper presents evidence on the timing of exposure that is most harmful: in utero exposure to pollution has the largest effect on survival. At levels that are common both indoors and outdoors in many poor countries, particulate matter has a sizeable effect on early-life mortality.

Questions in developing countries sometimes go unstudied because data are not available. Research on infant mortality effects of air pollution in the United States makes use of linked natality-mortality records and ground-based pollution monitors. No such data exist for Indonesia. To overcome this obstacle, this paper uses an unconventional methodology. First, child, infant and fetal death are inferred from ''missing children'' in the 2000 Indonesian Census. While the indirect method could introduce potential problems, the paper is able to show that migration, changes in gestation length, and other potential concerns do not seem to be

24. In unreported results, the effect of prenatal smoke is smaller in areas where more houses are constructed with bricks and concrete (compared to wood, palm leaves, etc.). Worse road quality, which could be a proxy for higher costs of evacuating, is also associated with larger mortality effects of pollution. Another approach to measuring evacuation would be to use distance to the nearest low-smoke area, but in practice this variable is too highly correlated with the pollution level to be useful as an interaction term. Use of surgical masks might also vary with income. Kunii *et al.* (2002) surveyed 532 people during the fires and found that use of surgical masks was associated with fewer respiratory problems, but most medical experts believe that surgical masks are ineffective at blocking out fine particles.

25. The income gradient in health for survivors could go in the same direction or, if the weakest individuals were ''harvested,'' in the opposite direction. Also, note that if richer people have less severe health effects because they are spending more money on healthcare or smokeless cooking stoves, then the income gradient in the welfare effects of pollution might be smaller than the income gradient in the health effects.

driving the results. Second, smoke data from a satellite-based spectrometer are used in lieu of ground-monitor pollution data. Because of the satellite's global coverage, proxy measures of particulate matter are available for even remote, underdeveloped areas.

There are at least two broader lessons about environmental issues in developing countries worth highlighting. First, environmental damage—and the accompanying health effects—are yet another consequence of weak governance. Corruption, which is prevalent in Indonesia as in many low–income countries, was an important factor behind the catastrophic fires. The Suharto government turned a blind eye when large firms started fires in violation of the law. The Minister of Forestry did try to hold firms accountable. In September 1997 he named 176 firms suspected of illegally setting fires. However, he quickly lost his job. Suharto, in a move that was brazen even by his formidable standards of crony capitalism, installed his golfing partner Bob Hasan—a timber magnate—as the new Minister of Forestry in early 1998. Hasan was outspoken in blaming small farmers for the fires and exonerating large firms, including his own. In virtually no cases were firms punished for starting illegal blazes.

The findings of the paper highlight a second link between the environment and economic development: the health burden from pollution seems to fall disproportionately on the poor. The estimated effect size is much larger in poorer areas than in richer areas. There is suggestive evidence that the heterogeneity could be due to people in underdeveloped areas using wood-burning stoves and facing a compounded effect of indoor plus outdoor air pollution. Differences in access to healthcare also seem to play a part. An interesting direction for further research would be to compare the early-life mortality effects in richer neighboring countries that were affected by Indonesia's pollution such as Malaysia and Singapore. Why the health effects of pollution vary with income remains an open question—and an area to pursue to better understand how environmental degradation creates unique challenges in developing countries.

Appendix 1

Verification That Census Counts Track Infant Mortality

This section verifies that population counts from the Census track data on births and infant deaths from the 2002 Demographic and Health Survey (DHS). The log of the number of surviving children should increase one-for-one with the log of total births and decrease one-for-one with the infant mortality rate (as can be derived with a few steps of algebra). Thus, I estimate

$$(3)\quad \ln(CohortSize)_{JT} = \alpha + \gamma_1 \ln(Births)_{JT} + \gamma_2 IMR_{JT} + \varepsilon_{JT}$$

where J is a province and T is a quarter, *Births* is the number of children born in the province-quarter, and *IMR* (infant mortality rate) is the fraction of those children who died by June 2000 when the Census was taken. Very few births per subdistrict-month are sampled in the DHS, so the validation exercise aggregates to provinces and

Table A1
Comparison of Log Cohort Size Variable to Survey Data on Infant Mortality

	Level of an Observation		
Dependent variable: Log cohort size	Quarter * Province (1)	Quarter * Province * Gender (2)	Month * Province (3)
Infant mortality rate	−1.34	−0.83	−0.54
	(1.12)	(0.65)	(0.41)
Log births	1.60	1.11	0.83
	(0.29)	(0.23)	(0.18)
Male		−0.01	
		(0.02)	
p-value of test that IMR coefficient = −1	0.78	0.79	0.27
p-value of test that Log Births coefficient = 1	0.05	0.62	0.36
Observations	1,248	2,496	3,742

Note: The dependent variable is the log of the cohort size enumerated in the 2000 Census. The independent variables are from the 2002 Demographic and Health Survey. Infant Mortality Rate is the number of children who have died by June 2000 divided by all children born. Log births is all children born. There are 26 provinces in the sample, and the period covers 48 quarters from 1988 to 1999. In Column 3, for two of the potential 3,744 observations, there are no births. Standard errors allow for clustering within a province.

quarters and uses a longer panel from 1988 to 1999 to gain power.[26] Note that *ln*(*Births*) varies not only with the number of births in the province-quarter but also with the DHS sampling rate for the province. The *IMR* variable should not be affected by this problem.

Appendix Table A1 presents the results. In Column 1, the coefficients on *IMR* and *ln*(*Births*) are −1.3 and 1.6, respectively, which are surprisingly close to the predictions of −1 and 1 given the crudeness of the exercise. In Column 2, each observation is a province-quarter-gender, and in Column 3, a province-month. The coefficients remain on the order of −1 and 1 but become smaller in magnitude, which is consistent with attenuation bias when smaller and hence noisier cell sizes are used. One cannot reject that the estimated coefficients are 1 and −1 at standard significance levels. In short, variation in population counts in the Census indeed tracks household survey data on the number of births and, importantly for this study, the infant mortality rate.

26. Ideally, the DHS would have recorded pregnancies that ended in fetal deaths. Some of the missing children in the Census are not among the live births measured by the DHS. Also, ideally, the validation exercise would use the same unit of observation and sample period as the main analysis, but the survey data are then too noisy to obtain meaningful results.

Table A2
Instrumental Variables Estimate

Dependent variable	First stage Prenatal Smoke (1)	IV Log Cohort Size (2)
(Sumatra or Kalimantan) * Sept 97 to Jan 98)	0.624*** (0.092)	
Prenatal smoke		−0.037** (0.017)
Observations	67,454	67,454
Fixed effects	month, subdistrict	month, subdistrict
F-statistic for instrument	45.6	n/a

Note: Each observation is a subdistrict-month. Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$. Observations are weighted by the number of individuals enumerated in the Census who reside in the subdistrict and were born in the year before the sample period.

Appendix 2

Data Sources and Description

A. Census

Indonesia conducted a Census of its population in June 2000. The dependent variable, the cohort size for a subdistrict-month, is the count of all enumerated people born in a given month who reside in the subdistrict. The specific date of birth is not available. The population weight for each subdistrict is the total number of people born in 1994 to 1996 enumerated in the Census.

I link mothers to children for the analyses by mother's 1995 residence and education and for constructing the measure of predicted births. Using a household identifier, I link each child to women who are 14 to 42 years older than the child. When there are multiple matches, I give preference to household heads or spouses of heads and to women closer to the peak of the fertility age distribution. To construct predicted fertility, I perform this matching for children born in 1999 and 2000 (through May) and calculate the mother-child age gap. Then for each age in months of women between 14 and 42, I calculate the number of children these women give birth to divided by the total number of women of that age. This gives the fertility rate (net of infant mortality) for each age. I make two adjustments to the fertility rate. First, I smooth the distribution using values for the four ages in months before and after each data point. Second, after the age of 38, I replace the estimate with a linear extrapolation from the estimated value at age 38 to zero at age 42 to correct for the fact that the matching process mistakenly assigns grandmothers as mothers in some cases (giving an implausibly high fertility rate for older ages). The next step is to calculate the number of women by age for each district in the sample period, and multiply it by the age-specific fertility rate.

Table A3
Different Sample Periods

	Shorter Periods			Balanced Calendar Months		Longer Period
Dependent variable: log cohort size	3/97- 5/98 (1)	12/96- 2/98 (2)	3/97- 2/98 (3)	11/96- 2/97 & 10/97- 2/98 (4)	11/96-2/97 & 11/97- 2/98 (5)	12/96- 5/98 (6)
Smoke	−0.003	−0.004	−0.009*	−0.005	−0.021	−0.001
	(0.005)	(0.005)	(0.005)	(0.009)	(0.042)	(0.006)
Prenatal smoke	−0.043***	−0.036***	−0.049***	−0.026*	−0.030**	−0.026**
	(0.012)	(0.012)	(0.012)	(0.014)	(0.014)	(0.012)
Postnatal smoke	−0.023***	−0.012	−0.026***	0.025	0.038	−0.006
	(0.009)	(0.010)	(0.008)	(0.030)	(0.032)	(0.009)
Observations	56,220	56,201	44,967	33,684	29,933	78,703
Subdistrict & month FEs?	Y	Y	Y	Y	Y	Y

Note: Each observation is a subdistrict-month. Standard errors, in parentheses below the coefficients, allow for clustering at the island-month level. *** indicates $p < 0.01$; ** indicates $p < 0.05$, * indicates $p < 0.10$. Observations are weighted by the number of individuals enumerated in the Census who reside in the subdistrict and were born in the year before the baseline sample period.

Summing across all the ages of women of childbearing age gives the predicted number of births for each district-month in the sample.

B. TOMS Pollution

In addition to the information provided in the text, further details on the Total Ozone Mapping Spectrometer can be found at http://toms.gsfc.nasa.gov.

C. Fire Location and Rainfall

The European Space Agency compiles the ATSR World Fire Atlas, a list of "hot spots" (date, time, latitude, and longitude) identified by nighttime infrared measurements by the ATSR-2 instrument onboard the ERS-2 satellite. A hot spot corresponds to a reading of > 312 Kelvins at 3.7 micron wavelength. For each subdistrict-month, hot spot-days within 50 km of the subdistrict's center are used to calculate the number of fire-days, or the sum over discrete fires of the duration of the fire in days.

The rainfall data set, Terrestrial Air Temperature and Precipitation: Monthly and Annual Time Series, is from the Center for Climatic Research, University of Delaware. The rainfall measure for each node on a 0.5 degree latitude by 0.5 degree longitude grid is interpolated from 20 nearby weather stations using a spherical version of Shepard's distance-weighting method. The rainfall measure for a subdistrict uses the closest node.

D. SUSENAS

I use household level data from the 1996 and 1999 SUSENAS core modules, which aggregate item-by-item consumption data to two categories, food and nonfood. For each household, per capita consumption is calculated weighting children by 0.75 and infants by 0.6 compared to adults who are weighted by 1. The district log consumption measure is the median across all households of log food consumption per capita. The survey is representative at the district level, so I use district-level measures.

E. PODES

The PODES is a census of all villages and towns in Indonesia. I use population, fuel use, and health facilities questions for 1996. One question asks what cooking fuel the majority of the village uses, and I group the answers as wood fuel (wood plus other biomass) or other (kerosene and gas). The population-weighted average of this indicator variable across villages in a subdistrict is the fuel-use variable. Healthcare measures are unweighted per capita measures for the subdistrict, for the reason that people have access to facilities throughout the subdistrict.

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