The Persistent Effects of Early-Life Exposure to Air Pollution: Evidence from the Indonesian Forest Fires*

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Abstract

We analyze the effects of early life exposure to air pollution in a developing country on children's human capital outcomes across the life cycle. To this end, we exploit the geographical variation of Indonesia's forest fires during the El Niño weather phenomenon in 1997 and cohort variation in exposure as a natural experiment. Children affected by air pollution in utero and in their early years have worse health outcomes but do not suffer significant effects in cognitive function relative to children not exposed to this shock. While the negative effects on the children's health persist, we find no differential effects by socio-economic characteristics, thus suggesting that the adverse health effects of air pollution are not mitigated by socio-economic status.

1 Introduction

Through the release of carbon dioxide (CO_2) and other gases like methane (CH_4), air pollution is a key source of climate change. In 2012, outdoor air pollution was associated with

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2.6 million premature deaths in Southeast Asia and the Western Pacific Regions, while indoor air pollution was linked to around 3.3 million premature deaths worldwide (WHO[1]). About 30 million people are involved in slash and burn agriculture– a common source of forest fires Dixon et al. [2001]. Indoor air pollution constitutes another health hazard that is particularly salient in developing countries, where around 3 billion people rely on biomass fuels such as wood, dung, and crop waste for domestic energy. More recently, air pollution reached record levels in New Delhi, India[2] and Beijing, China[3] that prompted school closures to protect children.

Given the vulnerability of children's respiratory systems, exposure to air pollution especially concerns their health, since among those who do not die prematurely due to pollution, early-life exposure to air pollution have been shown to adversely affect children's outcomes, and these effects sometimes persist into adulthood (Chay and Greenstone, 2003; Kajekar, 2007; Currie et al., 2014; Currie and Vogl, 2013 for a review). Up to now, however, the majority of these studies have examined air pollution's effects on children in high income countries. This paper contributes to the growing body of literature by examining the persistent effects of early-life exposure to air pollution in a middle income country: Indonesia. The analysis of lower income countries in air pollution studies is particularly pertinent because, unlike households in high income countries, households in low and middle income countries generally have limited resources to mitigate the negative effects of health shocks. As a result, the effects of air pollution may be more severe for children in low income countries relative to children in high income countries [Arceo et al., 2016, Hanna and Oliva, 2016].

To study the causal effects of pollution, we need to take into account that exposure to pollution is not exogenous. In particular, exposure to air pollution may be endogenous since families may sort based on environmental quality. These families may differ in unobserv-

[1]http://www.who.int/mediacentre/news/releases/2014/air-pollution/en/

[2]New York Times http://www.nytimes.com/2016/11/23/world/asia/india-delhi-pollution.html?_r=0

[3]New York Times http://www.nytimes.com/2015/12/09/world/asia/beijing-smog-red-alert-schools.html

able ways from other families in how they value environmental quality. If these unobserved characteristics are correlated with children's human capital, they can confound the estimated effects of air pollution. Therefore, studies have relied on natural experiments such as radioactive emissions, wild fires, changes in government regulations, economic crises, and plant closings to estimate the causal effects of air pollution [Currie and Vogl, 2013, Currie et al., 2014]. We exploit exposure to the Indonesian fires in 1997, which burned more intensely and for a longer period of time due to the El Niño weather phenomenon that caused extremely dry conditions and delayed the monsoon rain in Indonesia. Therefore, we use the unprecedented intensity and sudden timing and duration of the fires as a source of variation in cohort exposure to air pollution as a natural experiment [Jayachandran, 2009, Frankenberg et al., 2005]. Although we rely on a well-known extreme pollution event for causal identification, forest fires are episodic events whose frequency may increase due to climate change [United Nations Framework Convention on Climate Change, 2007]. Depending on the environmental conditions, forest fires can create uncontrolled haze that is comparable to the chronic exposure to indoor air pollution generated by the use of biomass fuels [Hanna et al., 2016].

The Indonesian forest fires have been studied in previous literature to examine the short term effects of air pollution on infant mortality and adult health in Indonesia and neighboring countries [Emmanuel, 2000, Sastry, 2002, Banerjee, 2014]. This paper is most closely related to Jayachandran [2009] and Frankenberg et al. [2005]. Frankenberg et al. [2005] study the immediate consequences of exposure to the fires on adult health and find that the fires had a negative impact on self-reported physical health. Jayachandran [2009] finds that the 1997 fires led to a decline of 1.2 percent in cohort size, especially from exposure during the third trimester in utero. These findings are consistent with previous studies, mainly in high income countries, that have found that exposure to pollution in utero affects children's health at birth and increases the likelihood of neonatal and infant mortality (Chay and Greenstone, 2003; Arceo et al., 2016; Foster et al., 2009; Currie et al., 2014; Currie and Vogl, 2013 for a

review). Instead of analyzing the immediate effects of the Indonesian forest fires, we focus on estimating the longer-term effects of exposure to these fires on surviving children's human capital. This is motivated by the current literature that argues that prenatal and early childhood periods are sensitive stages of child development, and negative shocks such as pollution during these years may predispose children to poor outcomes later in life [Barker, 1995, Currie and Vogl, 2013]. For example, negative shocks during these early years can impair skill formation, thus resulting in long-term consequences since past human capital investments influence both future skills and investments [Cunha and Heckman, 2007]. We extend this literature by estimating the persistence of early life exposure to air pollution as a policy-relevant hazard in developing countries.

There may be competing effects between selective mortality and scarring effects associated with air pollution. On one hand, to the extent that surviving children are healthier, we may see limited effects on children's health. On the other hand, surviving children may have negative scarring effects. With scarring, these surviving children may have poor human capital outcomes in the longer term as a result of exposure to the fires. Other studies, mostly from high income countries, have found that exposure to pollution during the first year of life is associated with lasting negative effects on adult outcomes, including cognitive function, education, and labor market outcomes (Bharadwaj et al., 2014; Isen et al., 2014; Peet, 2015; Sanders, 2012; Almond et al., 2009; Black et al., 2013; Currie and Vogl, 2013 for a review). Indeed, we find that surviving children's health is adversely affected 3, 10, and 17 years post exposure in a middle income country.

These Indonesian forest fires that occurred between August and November 1997 primarily affected the islands of Sumatra and Kalimantan. We exploit the geographic and cohort variation to estimate the effects of early life exposure to air pollution. In our estimation, we combine data from NASA's Earth Probe Total Ozone Mapping Spectrometer (TOMS) and the Indonesian Family Life Survey (IFLS), which includes communities in affected and unaffected areas. The data were collected during the fires, followed by 3, 10, and 17 years after

the 1997 fires. More specifically, data from TOMS provide daily measures of air pollution, while the IFLS' rich longitudinal data set includes children's health status, anthropometric measures, lung capacity, and cognitive tests. We use children's month, year, and place of birth to identify children who were and were not exposed to the fires in utero, in their first year of life, and/or their second year of life.

Our results suggest that health effects persist as evidenced by exposed children's lower height. We find that children exposed to air pollution in utero have lower height-for-age z-scores. In addition, these same children are also on average 0.2 standard deviations shorter later in life at 10 and 17 years post-exposure. Furthermore, children who were exposed in utero or their first 2 years of life have 5 to 9 percent lower lung capacity 10 years post-exposure (note, however, that this effect is no longer significant 17 years post-exposure). However, there appears to be no significant effects on children's cognitive function. We find limited heterogeneous treatment effects by maternal education or urban residence, suggesting that the health effects of exposure to air pollution are not mitigated by socio-economic status. Regarding potential mechanisms underlying these effects, we find suggestive evidence that during and immediately after the shock expectant mothers experienced a deterioration in their lung capacity and infant children suffered breathing problems. We also show evidence that selective migration or fertility responses are not likely to confound our results. Examining the impact of early-life exposure to pollution in low and middle income countries is of particular interest since pollution is an important policy challenge in such countries. The lasting consequences of air pollution on children's human capital may not be apparent immediately, but hidden in future damages.

The remainder of the paper is organized as follows. Section 2 presents the background on the 1997 fires in Indonesia. Section 3 describes the data and estimation strategy. Section 4 presents the results, section 5 provides a discussion, and section 6 concludes.

2 Background

2.1 Fires in Indonesia

In many low and middle income families, farmers, especially small scale ones, have traditionally used controlled burning as a method of land clearing. Land clearing happens before planting and, in Indonesia, takes place during the dry season, typically between July and October. This method of clearing is cheaper than its alternative, which involves heavy machinery and requires higher upfront capital investments. Historically, land clearing was limited to domestic settings, so the scope and effect of the fires were small. However, over time the scale of the fires increased as timber and palm oil plantations developed. These industries created more flammable debris and required larger areas of land to be cleared. In addition, peat land in parts of Sumatra and Kalimantan makes it difficult to contain fires. Because peat contains a mixture of decaying organic matter, when exposed to drought conditions, it subsequently dries and combusts easily, thereby causing fires to spread both quickly and usually underground. These conditions make it difficult to extinguish these fires without the help of heavy rainfall. In 1995, Indonesia banned the practice of use of fire to clear land. Since then, however, farmers, have ignored this ban, and the magnitude of the fires has continued to increase [Sastry, 2002].

Between August 1997 and November 1997, Indonesia experienced the most intense and long lasting series of forest fires in its history. These fires resulted from both annual land clearing by fire and the El Niño-Southern Oscillation weather phenomenon. El Niño is associated with unusual weather patterns around the world and causes extremely dry conditions in Indonesia, resulting in droughts and delaying Indonesia's monsoonal weather pattern. Consequently, once the Indonesian farmers began land clearing via fire, the drought conditions caused by El Niño exacerbated the fires, causing them to rage out of control and forcing Indonesia's then president Suharto to declare a state of emergency. The islands of Sumatra and Kalimantan were most affected by these fires, which destroyed 2 to 3 percent

of Indonesia's land area and produced thick haze that spread to such neighboring Southeast Asian countries as Brunei, Malaysia, Singapore, Thailand, and Vietnam [Sastry, 2002, Fullerton et al., 2008]. The estimated cost of these fires ranged from $2 to 3 billion in lost productivity and tourism, which was about 1 to 1.4 percent of GDP in 1997 [Tacconi, 2003]. The El Niño weather phenomenon happened again in 2015, causing about 100,000 deaths in the region as the fires to burned intensely in Indonesia, producing thick haze that also spread to neighboring countries [Koplitz et al., 2016].

The 1997 Indonesian fires resulted in increased levels of pollution as measured by total suspended particulate matter (TSP) and PM_{10}. TSP denotes the number of airborne particles or aerosols that are less than 100 micrometers, while PM_{10} designates smaller particles with a diameter of 10 micrometers or less. The smaller PM particles are able to reach the lower regions of the respiratory tract, and, consequently, can be responsible for adverse health effects such as: inhibited breathing and respiratory system function, damage to lung tissue, cancer, and premature death. The elderly, children, and people with chronic lung disease, influenza, or asthma, are especially sensitive to the effects of particulate matter. The main gases produced by these fires are carbon dioxide (CO_2), carbon monoxide (CO), methane (CH_4), nitrogen oxides (NO and NO_2), and ammonia. The peat land causes a higher level of methane to be emitted compared to fires in areas without peat. The PM_{10} standard set by the US Environmental Protection Agency is an average of no more than 150 $\mu g/m^3$ within a 24-hour period in a location more than once per year. According to WHO's guidelines for air quality, the PM_{10} 24-hour average standard is 50 $\mu g/m^3$. During the 1997 Indonesia fires, the PM_{10} levels reached the hazardous range for the most affected provinces, surpassing 2000 $\mu g/m^3$ in September, while TSP levels ranged between 150 and 4000 $\mu g/m^3$ [Heil and Goldammer, 2001, Ostermann and Brauer, 2001]. The level of pollution associated with these fires is comparable to levels of pollution associated with the chronic exposure to indoor air pollution generated by the use of biomass fuels [Hanna et al., 2016].

These pollution measures have been shown to be correlated with the aerosol index

recorded by TOMS [Torres et al., 2002, Frankenberg et al., 2005]. The TOMS aerosol measurements were taken daily from July 1996 to December 2005 in one degree latitude and 1.25 degree longitude grids. The index ranges from to 0 to 6 in Indonesia, where values of less than 0.1 correspond to clear skies and a value of 4 or greater corresponds to low visibility that makes it difficult to see the mid-day sun. During the fires, the aerosol index recorded a value of close to 6 in the most affected areas, and PM_{10} levels in those areas were above 3000 $\mu g/m^3$ [Frankenberg et al., 2005].

This paper is most closely related to Frankenberg et al. [2005] and Jayachandran [2009]. Jayachandran [2009] studies fetal, infant, and child mortality using data from the 2000 Indonesian census as well as pollution measures from the TOMS aerosol index recorded. This study finds that higher levels of air pollution due to the 1997 fires led to a decline of 1.2 percent in cohort size, especially due to exposure during the third trimester in utero. Jayachandran [2009] also finds that the effects are twice as large in poorer districts. Additionally, Frankenberg et al. [2005] study the consequences of the fires on adult health using data from the IFLS and pollution data from TOMS. They find that the fires had a negative impact on self-reported physical health, as measured by difficulty carrying a heavy load, respiratory problems, and general health status. We complement this literature by examining the effects of early life exposure to air pollution on surviving children's human capital outcomes in the longer term using additional waves of the IFLS and pollution measure from TOMS.

2.2 Air pollution and child development

Children in-utero are especially vulnerable to negative health shocks because their fetal programming may be altered by these shocks, leaving them susceptible to diseases such as coronary heart disease [Barker, 1995]. In-utero exposure to air pollution has been shown to adversely affect morbidity and mortality since a child's lung development commences in-utero [Currie et al., 2009, Jayachandran, 2009]. Likewise, ex utero children also remain vulnerable to the effects of air pollution, since in utero lung development continues postnatally. For

example, the elevated risk of altering the development of lung function when children are exposed to viral infections during infancy suggests that early life exposure to air pollution can be equally damaging [Kajekar, 2007]. While studies have shown the effects of air pollution on children's lung function, as well as neonatal and infant mortality [Currie and Vogl, 2013], we focus on the effects of air pollution on in and ex utero children's lung function and other health outcomes, conditional on surviving the initial exposure to air pollution.

We also explore the effects of pollution on children's cognitive function. While the precise mechanism that links air pollution to cognitive achievement is currently unknown, the hypothesized channel is that carbon monoxide crosses the placental barrier, which subsequently negatively affects the cardiovascular and respiratory functions that influence cognitive outcomes [Greingor et al., 2001]. Exposure to carbon monoxide, especially carbon monoxide, in the first year of life is associated with poor infant and child health [Plopper and Fanucchi, 2000, Neidell, 2004, Mortimer et al., 2008, Currie et al., 2009], including lower lower IQ scores [Bharadwaj et al., 2014] and lower educational attainment [Sanders, 2012]. While the scope of this study and the data we utilize take into account cognitive function, it is possible that mediating interventions were done to mitigate the initial effects of negative health shocks– parents may adopt compensating behavior to overcome the effects of negative early life shocks [Almond and Mazumder, 2013]. For instance, positive shocks offer some protective effects when children were exposed to both health interventions and natural disasters [Gunnsteinsson et al., 2016]. It is therefore an empirical question whether children's cognitive function would be affected 10 and 17 years post exposure.

3 Data and Empirical Strategy

3.1 Data

This paper combines data from the Indonesian Family Life Survey (IFLS) and NASA's Earth Probe Total Ozone Mapping Spectrometer (TOMS). TOMS (version 8) provides the pollu-

tion variable, while the outcomes of interest come from the IFLS. The IFLS is a longitudinal household survey that is representative of approximately 83 percent of the Indonesian population in 1993. The first wave of the survey (IFLS1) was conducted in 1993, the second wave (IFLS2) in 1997, the third wave (IFLS3) in 2000, the fourth wave (IFLS4) in 2007, and the fifth wave (IFLS5) in 2014.

The IFLS over sampled urban areas and rural areas outside of the main island of Java. The survey includes both affected provinces in Sumatra and Kalimantan as well as other provinces that were not affected by the 1997 fires. IFLS1 included 7,224 households residing in 321 enumeration areas in 312 communities in 13 of Indonesia's 26 provinces in 1993. A community is defined as a village in rural areas and/or a township in urban areas. Subsequent waves of the survey sought to re-interview all households in IFLS1, as well as any households that had split-off.

The IFLS contains rich information on individual, household, and community characteristics. Individual characteristics include age, education, marital status, employment, as well as complete pregnancy history for women between the ages of 15 and 49. The following anthropometric measures were also taken: height, weight, blood pressure, and lung capacity. Household characteristics include household size, household expenditure, and asset ownership.

Almost 90 percent of the second wave survey (IFLS2) in our sample was conducted between June and December 1997, 72 percent of which was conducted while the fires were burning between June and November 1997. Therefore, IFLS2 allows us to analyze the immediate effects of the fires on children's outcomes. IFLS3, conducted in 2000, makes it possible for us to analyze the short-term effects of the fires 3 years later. IFLS4 and IFLS5 conducted in 2007 and in 2014, respectively, enable us to examine longer-term outcomes including cognitive test scores and lung capacity measures.

We restrict the sample to children born between 1995 and 2000. We then divide the sample into two groups: affected children (born before and during the fires) and unaffected

children (born after the fires). All of these children are old enough for us to examine their longer term outcomes. We further restrict the sample to children whose height measurements were taken in 2000, 2007, and 2014. Since height measurements were take during every IFLS wave, this restriction allows us to consistently compare children's outcomes over time. Although migration can be an affected household's response to the fires, because the decision to migrate is endogenous, we include migrants from our analyzed sample[4] . Altogether, the sample includes 2,371 children born between 1995 and 2000. Among these children, 8 percent were born in districts that experienced the 1997 fires.

Outcomes In this study, children's outcomes come from the individual surveys, the anthropometric,and cognitive measures. Since stunting captures the cumulative effect of negative health shocks on children's physical growth, the short-term outcome we examine is children's height for age in 2000, when the children in our sample are under 5 years of age. Trained enumerators measured children's height and weight in each wave of the survey. We used the WHO standard to generate z-scores for children's height for age as a measure of children's health status. The indicator for stunting takes the value 1 if a child's height for age is more than 2 standard deviations below the standardized mean.

Our longer term outcomes are children's height, lung capacity, and cognitive function. Because height has been commonly used to capture the cumulative effects of health shocks and is a marker of long-term adult well-being, we use children's height in 2007 and 2014 as our first longer term health outcome [Currie and Vogl, 2013, Case and Paxson, 2008].

As our second longer-term outcome, the lung capacity measure captures peak respiratory flow, which indicates a person's maximum speed of expiration/exhalation in liters per minute. This measure shows whether there are any obstructions in the respiratory pathways. For this study, we use the IFLS' measures of lung capacity taken from respondents older than 9 years of age. [5] For the children in our sample, lung capacity is only available in 2007

[4]Approximately 4 percent of children born in both affected and non-affected districts moved between 1997 and 2000. When migrants are excluded, the estimates are qualitatively similar

[5]In 2007 (wave 4), this measure is not available for children born later than 1999.

and 2014. In each survey, three measures were taken for each respondent. Following the literature, we use the maximum of the three measurements [Silwal and McKay, 2014].

The IFLS administers cognitive testing on children and adult respondents between the ages of 7 and 24 years. In this study, we use test scores from 2007 and 2014, when the children are old enough to analyze their longer term outcome. Cognitive function is measured using 2 means: (1) the Raven progressive matrices, which were designed to assess abstract reasoning (such as spatial and problem-solving skills), and (2) the mathematics test, which determines a respondent's numerical ability. Children between 7 and 14 years old were given 12 questions from the Raven's test and 5 mathematics questions. Each child's score is the percentage of correct answers to the Raven and mathematics tests.[6] We combine these scores as our cognitive outcome. [7] We also create a z-score for children in each age group in 2007.

Pollution shock Pollution data come from TOMS (version 8). The daily aerosol index from TOMS has been shown to be correlated with levels of TSP [Torres et al., 2002]. TOMS measures daily aerosol in one degree latitude and 1.25 degree longitude grids. Adjacent grid points are approximately 175 kilometers apart. For this study, we use the available aerosol index from July 1996 to December 2005. The index ranges from 0 to almost 6 during the fires. We use the monthly average of the aerosol index for each grid and match these grids to the nearest IFLS district centroid. Indonesia experienced district proliferation over the years, so we use IFLS district centroids in 1997. The land area of Indonesian districts range anywhere from under 20 kilometers squared to 45,000 kilometers squared. A range of 2 to 9 IFLS districts were matched, depending on size, to one TOMS grid, with an average of 2.9 districts per TOMS grid. Following Frankenberg et al. [2005], our exposure variable takes the value 1 if the aerosol index for the district exceeded 1.5 for more than 3 days in a given month. Figure 1 shows the average exposure to air pollution for children born between 1995 and 2005. The average aerosol index in the first two years of life ranges from 0.05 to

[6]The cognitive measure was restricted to children who completed both the cognitive and mathematics components.

[7]We separate the tests into the Raven and mathematics and find qualitatively similar estimates.

1. Average exposure is similar across cohorts in affected and unaffected areas pre and post 1997. Children residing in affected districts were exposed to a higher level of pollution in 1997. Children who were born in 1998 were exposed to more pollution in-utero (in 1997) in the affected areas (panel A). Similarly, children who were born in 1997 and were between 0 and 12 months old in that year were exposed to higher pollution levels in the affected areas (panel B). Likewise, children who were between 13 and 24 months old in 1997 (those born in 1996) were also exposed to higher pollution in the affected areas.

Potential mechanisms We also explore potential mechanisms behind the short and longer-term effects of exposure to the fires. In the 1997 survey, the IFLS collected information on respiratory problems. An indicator for respiratory problems takes the value 1 if the mother or primary care giver reported the child (aged 3 years of younger) experienced any difficulty breathing or any coughing in the month prior to the second wave of the survey. Since the survey was conducted immediately after the fires, the indicator captures the immediate effect of the fires on young children's health, which is one potential channel for children's weaker respiratory function in the longer term.

Other potential mechanisms underlying the in-utero effects come from the impacts of the fires on expectant mothers' health. Therefore, we analyze mother's respiratory systems based on their lung capacity. We also study whether pregnant women's nutrition may have been disrupted by looking at mother's body mass index (BMI), which is defined as a person's weight in kilograms divided by her height in meters squared. In addition, we explore effects of the shock on the child's birth weight since it is considered a measure that summarizes prenatal investments in-utero [Bharadwaj et al., 2010, Rosenzweig and Zhang, 2009]. Lastly, we also examine the effects on parental responses through breastfeeding and fertility decisions.

3.2 Summary statistics and balance

Table 1 presents our sample characteristics. Panel A shows children's outcomes in 2000, 3 years after the fires. Panels B and C present children's outcomes in 2007 and 2014, 10 and 17 years after the fires respectively. Children who were exposed to the fires were on average shorter for their age (height-for-age z-score of -1.6 vs. -2.1), and a higher fraction of exposed children were stunted (50 percent vs. 40 percent) in wave 3 of the survey in 2000. The average height in 2007 (wave 4) of exposed children is 133 centimeters and the average of those who were not exposed (who are generally younger than exposed children) is 127 centimeters. The log lung capacity of children who were exposed and not exposed is similar at about 5.3, 10 years after the fires. For cognitive development, children who were exposed to the fires correctly answered 73 percent of the test questions, and children who were not exposed answered 69 percent of the questions correctly. Seventeen years after the fires, children who were exposed and not exposed are about the same height, have the same lung capacity, and achieve the same test scores.

Table 2 presents the differences in the observed characteristics of exposed and non-exposed children. These differences are adjusted for district fixed effects and child's year and month of birth fixed effects. All standard errors are clustered at the community level. The gender ratio is balanced in our sample, which is consistent with earlier work that finds no differential infant mortality by gender [Jayachandran, 2009].[8] Exposed and non-exposed children largely share similar household characteristics. For instance, the average household size and mother's education are similar across both groups; per capita household expenditure in 1997 is comparable across both groups; and poverty status, which we define as the bottom two quintiles of the distribution of per capita expenditure, is also similar across the two groups. The difference in another wealth indicator–land ownership– is also small and not statistically significant across the exposed and non-exposed groups. One major concern regarding the pollution due to the fires is that rural residents may have been affected

[8]Mother's age at birth is also balanced across the exposed and non-exposed groups.

more than urban residents, but our analyzed sample has a similar share of urban and rural residents across the affected and non-affected groups. Another selection concern is the endogenous response to migrate immediately after the fire or in the subsequent years. We find that the affected and non-affected groups have a similar probability of migration.

3.3 Estimation strategy

We exploit the temporal and geographical variation of the 1997 fires as a natural experiment. We employ a difference-in-differences approach to estimate the following model:

$$y_{idmy} = \delta_0 inutero_{idmy} + \delta_1 age1_{idmy} + \delta_2 age2_{idmy} + \beta X_i + \tau_y + \mu_m + \rho_d + \epsilon_{idmy}$$

where y_{idmy} is the outcome of interest for individual i, born in district d at month m and year y. The variable $inutero$ captures exposure to the 1997 fires in-utero, $age1$ captures exposure between 0 to 12 months, and the variable $age2$ captures exposure between 13 and 24 months. As explained above, exposure to the fires is characterized by being exposed to an aerosol index greater than 1.5 for more than 3 days during the month. We include the following household characteristics: urban residence, an indicator for the bottom two quintiles of per capita expenditure, and household size. In addition, we control for the following individual characteristics: male child, mother's age at delivery, and mother's education. We include year and month of birth fixed effects, τ_y and μ_m respectively, to take into account the common shocks that affect children born in the same month and year. We include district fixed effects, ρ_d, to take into account time invariant district characteristics. Standard errors are clustered at the community level (enumeration area).

4 Results

4.1 Short term outcomes

We begin by analyzing children's short-term outcomes in 2000 in Table 3. Early life negative health shocks can affect children's physical development, which is captured here by height-for-age and stunting. We find that children who were exposed to the fires in-utero are on average 0.3 standard deviations shorter than unexposed children (columns 1-2). Children's exposure to the fires in their first and second year of life is also detrimental, and the magnitudes are meaningful but not statistically significant. Fortunately, in spite of exposed children's lower average height-for-age, exposed children are not significantly more likely to be stunted (columns 3-4).[9] These results suggest that children's health is affected in the short term, but it is not so severely affected that exposure to the fires causes stunted growth.

4.2 Longer term outcomes

Early life negative health shocks disrupt children's human capital formation in the short term, while there are two potential trajectories in the longer term. Children may be able to catch up, or parents may engage in mitigating behavior to ensure exposed children are not affected in the medium and long-terms. Alternatively, children may fail to catch up and continue to be disadvantaged in the medium and long-term. Therefore, whether children's health continues to be affected due to exposure to acute pollution in early life poses an empirical question. We analyze children's medium-term outcomes 10 years post exposure (wave 4, in 2007) in Table 4 and 17 years post exposure (wave 5, in 2014) in Table 5. We begin with physical health status as measured by height and lung capacity, followed by cognitive function as measured by test scores.

[9] Figure 2 (Panel A) shows the distribution of height for age z-score in 2000, 3 years post exposure. We find that the height for age z-score distribution of children who were exposed to the fires in utero is different from the distribution of children who were not exposed to the fires at the 10 percent level, but it appears that differences are not driven by changes in stunting.

Ten years post exposure, we find that children who were exposed to the fires in-utero are on average 0.3 standard deviations (3.1 centimeters) shorter than children who were not exposed to the fires (columns 1-2).[10] Children exposed to the fires in the first year of life are also shorter, but the effects are not statistically significant. Similarly, there is no significant effect on height for children who were exposed in their second year of life.[11] These results suggest that children's physical development was affected 3 years after exposure, and the effect on height persists 10 years after exposure.

We expect exposure to air pollution to affect children's respiratory function; although, it is unclear whether the effect would persist 10 years after exposure. We find that children who were exposed to the fires in utero and during their first 2 years of life have lower lung capacity than children who were not exposed to the fires (columns 3-4). The effects are between 6 and 9 percentage points, suggesting that the negative effect on respiratory function persists 10 years after the fires.

On the other hand, we find no statistically significant effect on cognitive function as measured by age-adjusted cumulative test score (cols. 5-6). Another supporting piece of evidence that corresponds with no effect on cognitive function is that children exposed to the fires are not delayed in starting school. These results imply that other factors might have mitigated the effects of the health shocks on cognitive function, or that the link between exposure to air pollution and cognitive function is not particularly strong in this case.

We analyze the effects of early life exposure to air pollution 17 years post-exposure in Table 5. As adolescents, children exposed to the fires in utero are likely to be 0.2 standard deviations (1.8 centimeters) shorter (columns 1-2).[12] These results are consistent with the persistent health effects of negative shocks in early life. We find a small and not statistically

[10] Figure 2 (Panel B) shows the distribution of height in 2007, 10 years post exposure. We find that the height distribution of children who were exposed to the fires in utero is different from the distribution of children who were not exposed to the fires at the 10 percent level.

[11] Using the CDC's height-for-age z-score for older children, we find that the height-for-age z-score of children who were exposed to the fires in-utero is also lower than children who were not exposed to the fires.

[12] Figure 2 (Panel C) shows the distribution of height in 2014, 17 years post exposure. While children who were exposed to the fires in utero are on average shorter, we find that the height distribution of children who were exposed to the fires in utero is similar to the distribution of children who were not exposed to the fires.

significant effect on respiratory function 17 years post exposure (columns 3-4). Similar to our earlier results, we find no significant effect on children's cognitive function (columns 5-6) or the national examination test score in mathematics.[13] Yet, the link between exposure to acute pollution and respiratory function does not persist beyond 10 years post exposure. The change in the effect on respiratory function between 10 and 17 years post exposure may be due to children outgrowing respiratory symptoms. Our results are consistent with the literature that has found a gradual reduction in the incidence of wheezing and asthma-related symptoms as children transition into early adulthood [Strachan et al., 1996, Piippo-Savolainen and Korppi, 2008, Bisgaard and Bønnelykke, 2010].

4.3 Heterogeneous treatment effects

We explore heterogeneous treatment effects by gender, mother's education, and urban residence. With regard to gender, the fragile male hypothesis argues that males are more vulnerable to shocks in early life compared to females [Kraemer, 2000]. According to this hypothesis, we expect males to be more affected by air pollution. Table 6 presents the short-term (column 1) and longer term effects of exposure to air pollution on males and females (columns 2-4 correspond to outcomes 10 years post exposure, columns 5-7 correspond to outcomes 17 years post exposure). We find no evidence of heterogeneity by gender 3 years after exposure. In the longer term, males who were exposed to the fires in utero have lower lung capacity 10 years post exposure; this effect, however, loses statistical significance 17 years post exposure. There is no evidence of gender selective mortality immediately after the fires [Jayachandran, 2009]. Likewise, there appears to be no evidence for heterogeneous effects by gender in the longer term.

To explore the role of socio-economic status, we analyze heterogeneity by mother's education in Table 7. The variable low education takes the value 1 if the child's mother has primary education or lower. We find no significant heterogeneous effects by mother's education in the

[13] These results are consistent with a US-based study of children with asthma that finds no significant difference in test scores between children with and without asthma [Silverstein et al., 2001].

short term, even though mother's education is generally associated with protective health effects. In the longer term, we find that the negative effects of exposure to the fires early in life on health outcomes 10 years later are stronger for children with mothers who received low levels of education. However, the effect does not persist 17 years post exposure. The results suggest that maternal education has only some mediating effects.

In this study, we find limited heterogeneity by urban residence (Table 8). Children residing in urban areas who were exposed to the fires in their first year of life are on average shorter 17 years post exposure. On the other hand, children from urban areas who were exposed in-utero score 10 percentage points higher on the cognitive test. Since urban residence is typically associated with protective health effects, this result is unexpected [Fink et al., 2014]. In our setting, urban residents generally have better access to infrastructure and health care; therefore, we expect urban residents to be able to better engage in mitigating behavior. In spite of the disparity in urban and rural access to health care services, children residing in urban and rural areas who were exposed to the fires were equally affected. A closely related issue concerns households engaged in agriculture, since the drought that was associated with the El Niño might have affected these households more severely than non-agricultural households. Similar to our findings on urban residence, we find no evidence of heterogeneity by agricultural status (Table 9). Taken together, these results suggest that socio-economic characteristics do not seem to mitigate the persistent health effects of the fires.[14]

We also explore differential effect by chronic exposure to indoor air pollution. We include

[14] We also analyze heterogeneous treatment effects by poverty in table 13, defined as the bottom two quintiles of per capita expenditure. This corresponds to an average per capita expenditure of 98,000 *Rupiah* per month (in 2000 *Rupiah*, approximately USD 10). When we use the lowest quintile of per capita expenditure, we find worse outcomes for exposed children. This is consistent with earlier work by Jayachandran [2009] that finds larger effects in poorer communities. We also analyze the role of the local economy, and in particular the 1997 economic crisis, in table 14 by controlling for the change in unemployment in the child's year of birth. We find that children who were exposed to the fires in-utero have poorer health outcomes three and ten years post-exposure, and the estimated effect sizes are similar to our earlier results, thereby suggesting that the effects cannot be attributed to the economic crisis. We also explore the possibility of unemployment, income loss from any economic shock, or crop loss (due to the El Niño drought) in the household in 1997 (tables 15, 16, and 17 respectively) and find similar effects on children's health, thereby suggesting that effects cannot be attributed to household economic shocks.

an interaction term between outdoor air pollution from the fires and indoor pollution from wood stove– a major source of indoor air pollution in Indonesia and elsewhere (table 18). We find that children who are exposed to the smoke from wood burning stoves at home are likely to have poorer health outcomes and test scores. However, we find no significant heterogeneous effects by exposure to indoor air pollution from wood burning stoves. This is consistent with earlier results that suggest that the effects of air pollution on early life mortality can be attributed to the fires, not exposure to indoor air pollution from wood burning stoves [Jayachandran, 2009].

4.4 Potential channels

In this section, we explore the channels and parental responses to the negative shock of air pollution. We begin by exploring the effects of the fires on health markers likely to respond immediately after the shock. During the fires, we expect air pollution to affect infants and pregnant mothers' respiratory system. Indeed, we find that children who were exposed to the fires in their first two years of life are 15 to 20 percentage points more likely to experience respiratory problems in 1997, immediately after exposure to the fires (Table 10, column 1). The estimates are not statistically significant for children who were exposed in-utero (although we only have information on a small number of children who were exposed in-utero in the 1997 survey).

Since exposure to the fires has been shown to affect adult health [Frankenberg et al., 2005], we specifically explore how exposure to the fires affected maternal health. The negative shock to expectant mothers may be a plausible channel underlying the effects found for children exposed in utero, so we analyze whether the mother's respiratory system, particularly her lung capacity, was affected by air pollution, which, in turn, may affect fetal development, including the child's respiratory system [Margulies, 1986, Murphy et al., 2005, Sbihi et al., 2016]. We find that pregnant women exposed to the 1997 fires experience a 9 percentage point decrease in their lung capacity, which may explain the respiratory problems among

children who were exposed in-utero. We do not expect the fires to affect women's other health outcomes, such as nutritional status (Table 10, column 3). Nevertheless, we explore nutritional problems by analyzing the effects of the fires on expectant mothers' body mass index (BMI). We find that the difference in expectant mothers' BMI in affected and non-affected areas is not statistically significant, suggesting that women were able to obtain sufficient nutrition to gain weight during pregnancy. Taken together, these results imply that the fires affect respiratory function, but not the nutritional status of expectant mothers. These results are consistent with maternal and children's respiratory systems as part of the channels behind children's subsequent health outcomes.

Next, we explore whether exposure to the fires affected birth weight (column 4) and parental responses through breastfeeding and fertility decisions (columns 5-6).[15] Birth weight is often used in the economic literature as a proxy for health endowments at birth that may reflect prenatal inputs and capture the effects of exposure to air pollution in-utero [Bharadwaj et al., 2010, Rosenzweig and Zhang, 2009]. In this case, we do not find evidence that exposure to the fires affected child's birth weight. This finding is in line with our previous result that there were no significant effect of air pollution from the fires on maternal nutrition as proxied by BMI.

Regarding parental responses through breastfeeding and birth spacing, it is possible that mothers who were less healthy due to the fires would have to stop breastfeeding earlier than healthy mothers. On the other hand, it is probable that mothers might increase breastfeeding to improve their children's immune system. Additionally, birth spacing may increase if women's health were affected by the fires, or they may decide to wait longer before conceiving another child to invest more on the child experiencing the negative health shock. We find no statistically significant effect on breastfeeding behavior or birth spacing. We also explore whether affected children are more likely to have younger siblings and we find no statistically

[15]Low birth weight (birth weight $<$ 2,500 grams) is an important health outcome because it is correlated with later outcomes, but not all children were weighed at birth. Moreover, birth weight is based on mother's recall and there is measurement error in the reporting of birth weight.

significant effect. These results suggest that parents do not alter their fertility decisions in response to a negative health shock to their children.

4.5 Falsification test and Robustness

Our main threat to identification is that exposure to pollution may be spurious and confounded with unobserved shocks or trends. In this section, we provide some evidence that validate our empirical strategy. To begin, negative shocks several months before conception should not have a causal impact on birth outcomes. Table 11 presents 'falsification regressions' that include exposure to air pollution six months before conception as a way to check if there were spurious differential trends between children in places more and less affected by the pollution shock. If our estimated effects of in-utero and early childhood exposure to the fires on children's human capital outcomes in the short and long-term were biased by the presence of omitted variables or a trend, then these placebo regressions may show significant effects as well. Our results show no impact of these prior exposures and no change in the magnitude of the effects of exposure in utero and at ages 1 and 2 years.

We explore different thresholds of the aerosol index for sensitivity, and show that our results are robust to alternative definitions of the shock. At a higher threshold of 1.75 (Table 12, Panel A), we find persistent effects on height among children who were exposed to the fires in-utero at 3, 10, and 17 years post-exposure (although only 4.5 percent of the sample were exposed to the higher pollution threshold). We also find that children's respiratory function is affected 10 years after exposure, but the results are not significant 17 years after exposure. We find no significant effects on cognitive test 10 or 17 years after exposure. Using a lower threshold of aerosol index of 1.25 (Panel B), we find qualitatively similar results to our earlier estimates.[16] Exposure to an aerosol index of 1.25 for at least 3 days in a month in early life continues to be associated with persistent health effects. Children who were exposed to a lower pollution threshold in-utero are about 3 centimeters shorter 10

[16] When we use an even lower threshold, an aerosol index of 1.00, the results are qualitatively similar, but most of the effects are not statistically significant.

years post exposure, which is similar to our earlier finding. The negative effects of in-utero exposure on height 17 years after the shock are negative and similar in magnitude to the our main specification, albeit they are not statistically significant. According to these robustness checks, the health effects persist even when children are exposed to lower pollution levels, suggesting the importance of the longer-term effects of pollution.

4.6 Selection issues

One important concern is that exposure to the fires affected families' fertility and migration decisions, which could confound our estimates of exposure to early-life pollution. In this section, we address these concerns by looking at family migration and fertility responses to exposure to the 1997 fires.

An issue of importance is that women who became pregnant during the fires in affected areas may be different from other women. Column 1 in table 19 explores the relationship between pregnancy status during the 1997 fires and living in affected districts. Overall, there is no evidence of differences in the likelihood of becoming pregnant between places affected and not affected by the fires. Also, to explore whether fertility responses may be different across subgroups, we interact living in an affected areas with several socio-demographic characteristics such as a woman's education and family size. Again, we find no evidence of differential fertility response to the fires.

Next, one may be concerned that exposure to the fires influenced families' migration decisions and that such migration responses may have changed the socio-demographic composition of families living in places more or less affected by the fires, thereby confounding the true effect of exposure to air pollution. In columns 2 and 3 of Table 19, we examine whether the probability of migrating between 1997 and 2000 or the subsequent years was different in places affected by the fires. We do not find evidence that migration responses were different between districts exposed and not exposed to the fires.

5 Discussion

Early life exposure to air pollution has been shown to adversely affect children's outcomes. Jayachandran [2009] finds that the fires were associated with a smaller birth cohort, suggesting fetal death and infant mortality. In light of her findings, our results on lower lung capacity and height among children who were exposed to air pollution in-utero could be taken as a lower bound, since these children survived childhood. In spite of the persistent effects on children's health outcomes, we find no significant effects on children's cognitive function. The results imply that either the link between pollution and cognitive function is not particularly strong in this case, or that parents engage in compensating behavior that mitigate the effects. More work on the link between air pollution and cognitive function would shed some light on the channels between air pollution and children's cognitive development.

We find that the effects on children's respiratory system persist 10 years after exposure. Exposure to the Indonesian fires can be compared to the effects of other forms of outdoor pollution and even indoor pollution such as second-hand smoke and biomass fuels. Estimated levels of particulate matter (PM_{10}) associated with second-hand smoke range between 60 $\mu g/m^3$ outdoor and 210 $\mu g/m^3$ indoor [Cunningham et al., 1994, Gilliland et al., 2001]. Our findings are comparable to the effects of exposure to second-hand smoke on children's respiratory function, which ranges between 3 and 5 percent [Gilliland et al., 2001]. This result thereby suggests that the effect of exposure to acute air pollution is persistent and comparable to chronic exposure to secondhand smoke. Furthermore, estimated levels of PM_{10} associated with biomass fuels range between 1000 and 2000 $\mu g/m^3$ at peak indoor concentration [Fullerton et al., 2008]; these levels are comparable to the pollution levels associated with the 1997 fires. While the pollution levels were comparable, the effects of chronic exposure through a wood burning stove combined with exposure to the fires are not statistically significant, suggesting that the acute exposure to air pollution affects children more adversely in this case.

We find no heterogeneity by mother's education or urban residence. These results high-

light the indiscriminate health effects of early life exposure to acute air pollution. The lack of protective effects of maternal education and urban residence indicate the importance of air pollution as an environmental concern that access to infrastructure that typically accompanies urban residence does not appear to mitigate. Further research on air pollution and avoidance behavior in developing countries will allow us to explore the role of information and socio-economic status in mitigating the effects of pollution on long term human capital outcomes [Bharadwaj et al., 2014].

The persistent effects on children's height may be associated with later earnings [Thomas and Strauss, 1997, Schultz, 2002, Persico et al., 2004, Case and Paxson, 2008, Vogl, 2014]. If we use the estimated height premium of 8 percent for an additional 10 centimeters in physical stature for Indonesia [Sohn, 2015], and that this premium is not mediated by differences in cognitive function [LaFave and Thomas, 2013], the estimated effect of 2 centimeters lower height from exposure to the fires would translate to 1.6 percent lower earnings for the affected population. Based on our sample, 7 percent of surviving children under 2 years were affected by the fires. Therefore, we assume that this fraction of the workforce would be about 2 centimeters shorter, and earn less once they join the labor market. Extrapolating these estimates, at Indonesia's current GDP of USD 868.2 billion and 7 percent of the workforce affected by the fires, the effect of lower height on earnings translates to 1 billion lower GDP (0.1 percent of GDP) annually. These estimates suggest that the hidden costs of air pollution can be long lasting.

6 Conclusion

This paper addresses the link between environmental and health policies in a low middle-income country. Specifically, we find that the adverse effects of acute exposure to air pollution on children's health are indiscriminate and are not limited to children from poor households or rural households. Our results suggest that young children exposed to the fires would be

affected for years to come. Since the annual occurrence of forest fires in Indonesia continues to be a policy challenge for Southeast Asia, including the most recent 2015 fires that were similarly exacerbated by the El Niño weather phenomenon, interventions that reduce the occurrence of these fires are necessary. Even with imperfect enforcement, more feasible policy options would involve developing interventions that encourage avoidance behavior to limit exposure to air pollution.

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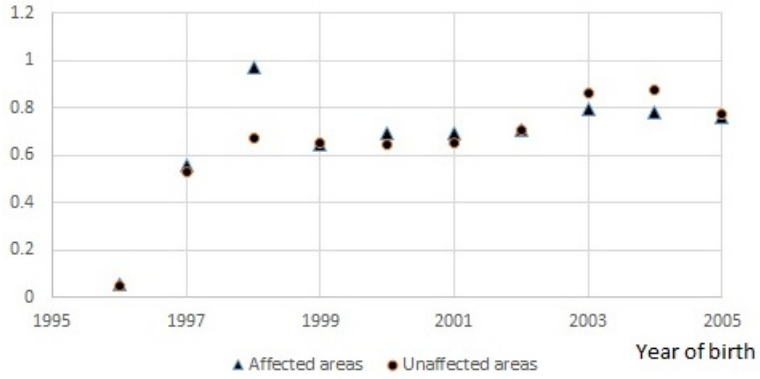
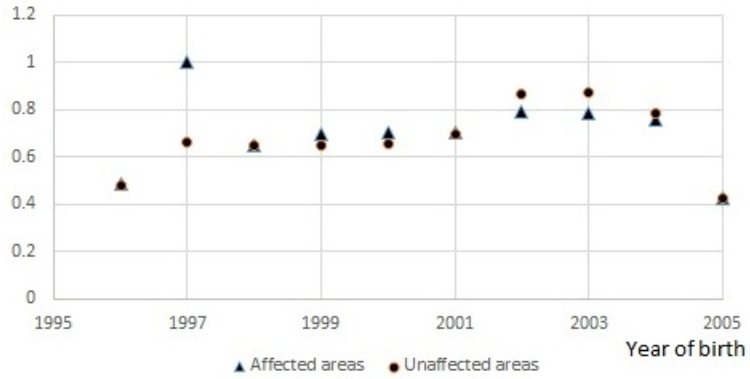
Tables and Figures

Figure 1: Average aerosol index in early life

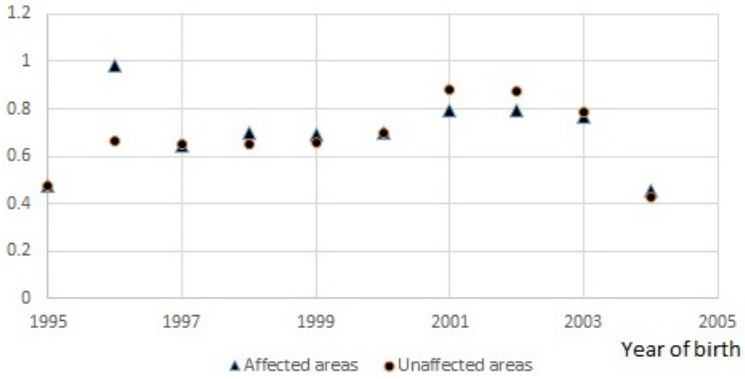
Panel A. Average aerosol index in utero



Panel B. Average aerosol index at 0-12 months



Panel C. Average aerosol index at 13-24 months



Notes: Affected takes the value 1 if the child's district of birth had an aerosol index of more than 1.5 for at least 3 days in a month. The aerosol index ranges from 0 to almost 6 in our sample.

Table 1: Summary statistics

	(1)	(2)	(3)	(4)
	Non-exposed		Exposed	
	Mean	N	Mean	N
Panel A. Short term outcomes: 3 years post fires				
Height for age	-1.567	2,147	-2.098	177
	(1.590)		1.192	
Stunted	0.403	2,147	0.508	177
	(0.491)		0.501	
Panel B. 10 years post fires				
Height (cm)	126.857	2,147	132.590	177
	(10.224)		9.828	
Lung capacity (log)	5.289	1,242	5.304	173
	(0.287)		0.257	
Total test score	0.689	1,684	0.733	154
(fraction correct)	(0.176)		0.170	
Panel C. 17 years post fires				
Height (cm)	157.143	2,147	157.286	177
	(8.120)		8.261	
Lung capacity (log)	5.840	2,136	5.905	176
	(0.268)		0.275	
Total test score	0.739	1,789	0.766	150
(fraction correct)	(0.178)		0.147	

Notes: Exposed takes the value 1 if the child's district of birth had an aerosol index of more than 1.5 for at least 3 days in a month. The aerosol index ranges from 0 to almost 6 in our sample. Children's respiratory problem in the past four weeks prior to the survey is reported by the mother or primary caretaker. Stunted takes the value 1 if a child is less than 2 standard deviations below average for height-for-age. Lung capacity is the maximum of the three readings taken in the survey. IFLS2 was conducted during or immediately after the fires in 1997, IFLS3 was conducted 3 years after the fires in 2000, IFLS4 was conducted 10 years after the fires in 2007, IFLS5 was conducted 17 years after the fires in 2014.

Table 2: Balance test: differences in the characteristics of exposed and non-exposed children

	(1) Male	(2) Household size	(3) Mother's education: primary	(4) Poor household	(5) Own land	(6) Urban	(7) Moved by 2000	(8) Moved
In-utero	0.045	-0.402	0.042	-0.019	-0.011	0.012	-0.029	-0.027
	(0.073)	(0.421)	(0.065)	(0.075)	(0.081)	(0.057)	(0.019)	(0.019)
0-12mo	0.023	0.429	-0.002	0.053	0.001	0.059	0.000	0.004
	(0.086)	(0.449)	(0.071)	(0.065)	(0.071)	(0.041)	(0.025)	(0.025)
13-24mo	0.036	-0.125	0.017	-0.032	0.084	0.004	-0.007	-0.025
	(0.087)	(0.423)	(0.073)	(0.078)	(0.074)	(0.046)	(0.032)	(0.027)
Obs.	2,371	2,371	2,371	2,371	2,371	2,371	2,371	2,371
R-sq.	0.075	0.152	0.248	0.217	0.170	0.618	0.338	0.340

Notes: Mother's primary education takes the value one if she has primary education or lower. Poverty takes the value one if the household is in the bottom two quintiles of total per capita expenditure in 1997 (1 USD~Rp. 10,000). The regressions include district, children's year and month of birth fixed effects. All standard errors are clustered at the community level (enumeration area). * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 3: Short term outcomes: 3 years post exposure

	(1)	(2)	(3)	(4)
	Height for age z-score		Stunted	
In-utero	-0.497*	-0.515**	-0.019	-0.018
	(0.262)	(0.253)	(0.071)	(0.072)
0-12mo	-0.334*	-0.326*	0.060	0.060
	(0.181)	(0.183)	(0.060)	(0.060)
13-24mo	-0.196	-0.205	0.103	0.107
	(0.194)	(0.189)	(0.074)	(0.072)
Observations	2,371	2,371	2,371	2,371
R-squared	0.221	0.232	0.169	0.176
HH char.	N	Y	N	Y
Mean of	-1.567		0.403	
non-exposed	(1.590)		(0.491)	

Notes: Respiratory problem in wave 2 (1997) reported by mothers or primary caretakers. Height for age z-score measured in wave 3 (2000). Stunted takes the value one if child's height-for-age z-score is more than two standard deviations below the mean. Mother's primary education takes the value one if she has primary education or lower. Per capita expenditure is in 2000 Rupiah (1 USD~Rp. 10,000 in 2000). The regressions include district, children's year and month of birth fixed effects. All standard errors are clustered at the community level (enumeration area). * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 4: Children's outcomes in 2007: 10-years post exposure

	(1)	(2)	(3)	(4)	(5)	(6)
	Height		Lung capacity		Cognitive test	
In-utero	-3.130***	-3.151***	-0.085*	-0.085*	0.013	0.042
	(1.192)	(1.170)	(0.045)	(0.045)	(0.152)	(0.150)
0-12mo	-0.336	-0.205	-0.057	-0.054	0.080	0.071
	(1.136)	(1.139)	(0.036)	(0.037)	(0.164)	(0.162)
13-24mo	0.571	0.545	-0.099**	-0.101**	0.030	0.040
	(1.025)	(1.002)	(0.047)	(0.047)	(0.178)	(0.174)
Observations	2,371	2,371	1,439	1,439	1,876	1,876
R-squared	0.624	0.635	0.405	0.411	0.165	0.188
HH char.	N	Y	N	Y	N	Y
Mean of non-exposed group	126.857 (10.224)		5.289 (0.287)		0.689 (0.176)	

Notes: The maximum of 3 measures of lung capacity is used. Age-adjusted test score is the age-adjusted z-score of the fraction correct response on both the Raven and mathematics tests. Mother's primary education takes the value one if she has primary education or lower. Per capita expenditure is in 2000 Rupiah (1 USD~Rp. 10,000 in 2000). The regressions include district, children's year and month of birth fixed effects. All standard errors are clustered at the community level (enumeration area). * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 5: Children's outcomes in 2014: 17 years post exposure

	(1)	(2)	(3)	(4)	(5)	(6)
	Height		Lung capacity		Cognitive test	
In-utero	-1.833*	-1.850*	-0.025	-0.023	0.010	0.012
	(1.064)	(1.084)	(0.030)	(0.030)	(0.031)	(0.029)
0-12mo	-0.790	-0.683	0.007	0.009	0.004	0.008
	(1.009)	(1.011)	(0.035)	(0.035)	(0.023)	(0.024)
13-24mo	-0.478	-0.486	-0.011	-0.011	-0.023	-0.020
	(1.099)	(1.099)	(0.031)	(0.031)	(0.025)	(0.025)
Observations	2,371	2,371	2,359	2,359	1,979	1,979
R-squared	0.452	0.459	0.472	0.477	0.166	0.189
HH char.	N	Y	N	Y	N	Y
Mean of non-exposed	157.143		5.840		0.739	
group	(8.120)		(0.268)		(0.178)	

Notes: The maximum of 3 measures of lung capacity is used. Test score is the the fraction correct response on both the Raven and mathematics tests. Mother's primary education takes the value one if she has primary education or lower. Per capita expenditure is in 2000 Rupiah (1 USD~Rp. 10,000 in 2000). The regressions include district, children's year and month of birth fixed effects. All standard errors are clustered at the community level (enumeration area). * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 6: Heterogeneous treatment effects by gender

	(1) Height for age	(2) Height	(3) Lung capacity	(4) Test score	(5) Height	(6) Lung capacity	(7) Test score
Male	-0.138**	-0.729**	0.082***	-0.029	9.361***	0.317***	-0.014
	(0.065)	(0.293)	(0.015)	(0.046)	(0.273)	(0.009)	(0.010)
In-utero	-0.665**	-2.976*	-0.104*	0.028	-1.617	-0.045	0.047
	(0.312)	(1.699)	(0.059)	(0.202)	(1.071)	(0.048)	(0.029)
0-12mo	-0.340	0.189	-0.011	0.130	-1.134	0.011	0.021
	(0.231)	(1.550)	(0.045)	(0.213)	(1.258)	(0.038)	(0.024)
13-24mo	-0.314	1.050	-0.088*	-0.008	-1.039	-0.032	-0.005
	(0.219)	(1.282)	(0.051)	(0.220)	(1.285)	(0.036)	(0.029)
In-utero x Male	0.291	-0.274	0.046	0.040	-0.514	0.045	-0.063
	(0.346)	(1.966)	(0.065)	(0.252)	(1.657)	(0.060)	(0.044)
0-12mo x Male	0.008	-0.784	-0.098*	-0.119	0.953	-0.007	-0.019
	(0.288)	(1.690)	(0.054)	(0.211)	(1.382)	(0.058)	(0.031)
13-24mo x Male	0.229	-1.043	-0.033	0.099	1.142	0.044	-0.029
	(0.246)	(1.925)	(0.072)	(0.291)	(1.603)	(0.052)	(0.039)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.233	0.635	0.413	0.189	0.459	0.477	0.191

Notes: All analyses include district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 7: Heterogeneous treatment effects by mother's education

	(1) Height for age	(2) Height	(3) Lung capacity	(4) Test score	(5) Height	(6) Lung capacity	(7) Test score
Mother's education: primary	-0.126	-1.047***	-0.002	-0.239***	-0.171	-0.021*	-0.059***
	(0.078)	(0.360)	(0.019)	(0.055)	(0.346)	(0.011)	(0.011)
In-utero	-0.312	-2.210	-0.128**	0.035	-1.264	-0.052	0.010
	(0.284)	(1.477)	(0.055)	(0.183)	(1.171)	(0.035)	(0.039)
0-12mo	-0.353	-0.866	0.002	0.114	-1.325	0.013	0.017
	(0.249)	(1.629)	(0.046)	(0.191)	(1.284)	(0.038)	(0.029)
13-24mo	0.053	2.443	-0.108*	0.151	0.627	0.013	-0.053
	(0.289)	(1.545)	(0.064)	(0.209)	(1.512)	(0.033)	(0.038)
In-utero x Low maternal education	-0.389	-1.783	0.097	0.034	-1.154	0.063	0.002
	(0.367)	(2.050)	(0.073)	(0.247)	(2.016)	(0.054)	(0.056)
0-12mo x Low maternal education	0.043	1.108	-0.114*	-0.100	1.143	-0.015	-0.013
	(0.315)	(1.836)	(0.064)	(0.253)	(1.628)	(0.065)	(0.033)
13-24mo x Low maternal education	-0.446	-3.249*	-0.008	-0.211	-1.856	-0.049	0.054
	(0.343)	(1.968)	(0.068)	(0.280)	(1.660)	(0.050)	(0.040)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.233	0.636	0.413	0.189	0.459	0.477	0.190

Notes: Low education takes the value one if mother's education is primary or lower. All analyses include district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 8: Heterogeneous treatment effects by urban residence

	(1) Height for age	(2) Height	(3) Test score	(4) Lung capacity	(5) Height	(6) Lung capacity	(7) Test score
Urban	0.259**	1.022**	0.020	-0.133*	0.989**	0.000	-0.001
	(0.103)	(0.456)	(0.022)	(0.079)	(0.422)	(0.013)	(0.011)
In-utero	-0.668**	-3.594***	-0.065	0.014	-2.340*	-0.010	-0.013
	(0.317)	(1.352)	(0.058)	(0.176)	(1.333)	(0.036)	(0.034)
0-12mo	-0.345	0.325	-0.053	-0.046	0.259	0.011	0.003
	(0.223)	(1.357)	(0.049)	(0.202)	(1.243)	(0.045)	(0.026)
13-24mo	-0.171	0.526	-0.079	-0.024	-0.010	-0.010	-0.023
	(0.209)	(1.169)	(0.055)	(0.219)	(1.397)	(0.040)	(0.026)
In-utero x Urban	0.465	1.425	-0.069	0.162	1.432	-0.042	0.102*
	(0.442)	(2.265)	(0.083)	(0.279)	(1.911)	(0.063)	(0.055)
0-12mo x Urban	0.055	-1.579	0.004	0.356	-2.772	-0.004	0.021
	(0.294)	(2.071)	(0.069)	(0.261)	(1.695)	(0.061)	(0.044)
13-24mo x Urban	-0.065	0.153	-0.071	0.188	-1.325	-0.006	0.017
	(0.325)	(1.812)	(0.084)	(0.365)	(1.746)	(0.050)	(0.053)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.233	0.635	0.412	0.189	0.460	0.477	0.191

Notes: All analyses include district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 9: Heterogeneous treatment effects by agricultural participation

	(1) Height for age	(2) Height	(3) Test score	(4) Lung capacity	(5) Height	(6) Lung capacity	(7) Test score
Agricultural	-0.265***	-0.689	-0.051**	-0.124	-0.837*	-0.015	0.005
household	(0.099)	(0.429)	(0.024)	(0.082)	(0.432)	(0.013)	(0.012)
In-utero	-0.261	-2.085	-0.065	0.102	-1.072	0.017	-0.005
	(0.290)	(1.639)	(0.054)	(0.239)	(1.271)	(0.038)	(0.042)
0-12mo	-0.462*	-0.251	-0.036	0.090	-1.472	0.016	0.017
	(0.238)	(1.348)	(0.054)	(0.250)	(0.991)	(0.040)	(0.036)
13-24mo	-0.324	0.377	-0.109*	0.147	-1.053	-0.011	-0.007
	(0.256)	(1.473)	(0.061)	(0.266)	(1.588)	(0.036)	(0.027)
In-utero x Agri	-0.454	-0.502	0.063	-0.065	-0.786	-0.040	0.049
	(0.569)	(2.266)	(0.073)	(0.287)	(1.821)	(0.063)	(0.060)
0-12mo x Agri	0.325	1.616	-0.030	0.128	2.392	0.037	-0.077
	(0.414)	(2.115)	(0.092)	(0.316)	(2.227)	(0.077)	(0.052)
13-24mo x Agri	0.332	1.526	0.053	0.102	2.087	0.019	0.016
	(0.296)	(1.955)	(0.085)	(0.365)	(1.753)	(0.063)	(0.045)
Observations	2,002	2,002	1,222	1,590	2,002	1,993	1,679
R-squared	0.261	0.650	0.422	0.212	0.465	0.482	0.214

Notes: All analyses include district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 10: Potential channels

	(1) Any breathing problem	(2) Mother's lung capacity	(3) Mother's BMI	(4) Breast-fed >6mo	(5) Birth spacing
In-utero	-0.003	-0.0518**	0.0487	-0.026	-0.218
	(0.071)	(0.0244)	(0.053)	(5.498)	(0.123)
0-12mo	0.149*			0.016	1.842
	(0.080)			(0.054)	(6.092)
13-24mo	0.105*			-0.013	-2.803
	(0.062)			(0.046)	(8.248)
Observations	956	3,720	3,927	2,205	1,845
R-squared	0.200	0.330	0.042	0.263	0.171

Notes: Mother's primary education takes the value one if she has primary education or lower. Per capita expenditure is in 2000 Rupiah (1 USD~Rp. 10,000 in 2000). All analyses include district fixed effects. All standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 11: Pre-conception exposure to pollution in affected and unaffected areas

	(1) Height for age	(2) Height	(3) Lung capacity	(4) Test score	(5) Height	(6) Lung capacity	(7) Test score
Exposed in 6mo. pre-conception	0.015	0.530	-0.078	0.143	-0.066	-0.007	0.012
	(0.366)	(1.022)	(0.053)	(0.183)	(1.032)	(0.034)	(0.036)
In-utero	-0.519**	-3.297***	-0.091**	0.012	-1.832	-0.021	0.009
	(0.252)	(1.269)	(0.046)	(0.155)	(1.154)	(0.030)	(0.028)
0-12mo	-0.323*	-0.110	-0.089*	0.101	-0.695	0.008	0.010
	(0.189)	(1.154)	(0.048)	(0.162)	(1.051)	(0.035)	(0.023)
13-24mo	-0.203	0.620	-0.140**	0.069	-0.496	-0.012	-0.018
	(0.186)	(0.992)	(0.055)	(0.169)	(1.109)	(0.031)	(0.025)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.232	0.635	0.412	0.189	0.459	0.477	0.190

Notes: The indicator 'affected' takes the value one if the child's district of birth had an aerosol index of more than 1.5 for at least 3 days in a month in 1997. * p<0.1, ** p<0.05, *** p<0.01.

Table 12: Alternative aerosol threshold

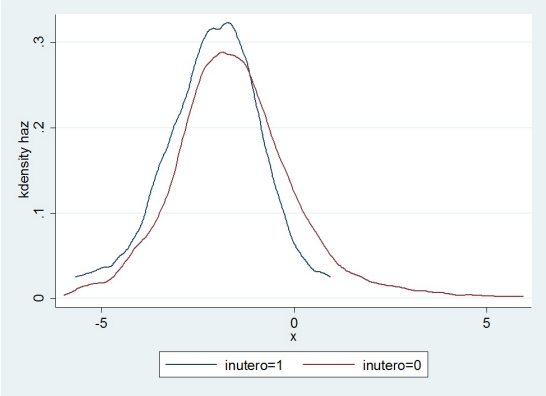
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Height for age z-score	Height	Lung capacity	Cognitive test	Height	Lung capacity	Cognitive test
Panel A. Aerosol index > 1.75							
In-utero	-0.852**	-4.540***	-0.117**	0.157	-3.505***	-0.047	0.023
	(0.340)	(1.362)	(0.052)	(0.175)	(1.121)	(0.034)	(0.034)
0-12mo	-0.185	-1.337	-0.056	0.077	-1.328	-0.006	0.022
	(0.217)	(1.220)	(0.041)	(0.198)	(1.103)	(0.045)	(0.027)
13-24mo	-0.289	-0.586	-0.136***	0.107	-1.752	-0.040	-0.030
	(0.243)	(1.242)	(0.052)	(0.204)	(1.266)	(0.038)	(0.029)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.233	0.636	0.412	0.189	0.461	0.477	0.190
Panel B. Aerosol index > 1.25							
In-utero	-0.486*	-3.021***	-0.093**	0.004	-1.749	-0.015	-0.000
	(0.260)	(1.085)	(0.044)	(0.143)	(1.065)	(0.028)	(0.029)
0-12mo	-0.349*	-0.588	-0.067*	0.057	-0.489	-0.011	0.016
	(0.199)	(1.097)	(0.035)	(0.162)	(0.949)	(0.035)	(0.023)
13-24mo	-0.215	0.441	-0.110**	-0.058	0.001	-0.018	-0.011
	(0.183)	(0.958)	(0.045)	(0.181)	(1.084)	(0.030)	(0.026)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.233	0.635	0.412	0.188	0.459	0.477	0.189

Notes: All analyses include district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Appendix

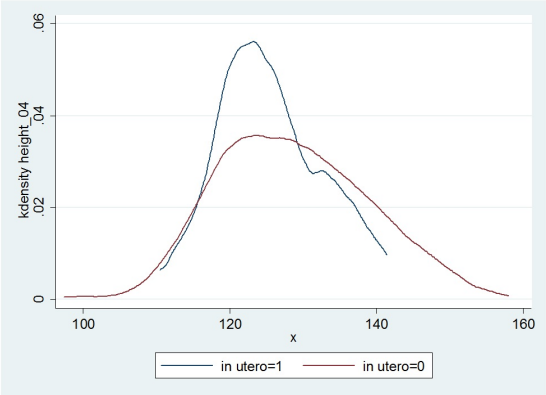
Figure 2: Distribution of children's height

Panel A. Height for age z-score 3 years post exposure



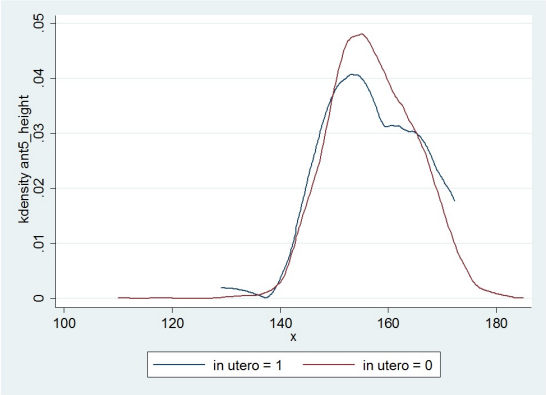
Kolmogorov-Smirnov test p-value 0.065.

Panel B. Height in 2007: 10 years post exposure



Kolmogorov-Smirnov test p-value 0.084

Panel C. Height in 2014: 17 years post exposure



Kolmogorov-Smirnov test p-value 0.496

Notes: In utero takes the value 1 if the child's district of birth had an aerosol index of more than 1.5 for at least 3 days in a month. The aerosol index ranges from 0 to almost 6 in our sample.

Table 19: Selective fertility and migration

	(1)	(2)	(3)
	Pregnant during the fire	Moved between 1997-2000	Moved
Affected	0.0456	-0.193**	-0.160*
	(0.0350)	(0.087)	(0.086)
Mother's education: primary	-0.00249	-0.039***	-0.037***
	(0.0104)	(0.013)	(0.013)
Affected x Low education	0.00760	0.009	0.002
	(0.0343)	(0.027)	(0.027)
Household size $>$ 6	0.00978	-0.048***	-0.050***
	(0.00943)	(0.012)	(0.012)
Affected x Large household	-0.0209	0.004	0.015
	(0.0246)	(0.028)	(0.026)
Poor household	-0.0216**	-0.006	-0.004
	(0.00982)	(0.012)	(0.012)
Affected x Poor	-0.0329	0.009	-0.004
	(0.0237)	(0.033)	(0.031)
Urban	-0.0283**	0.006	0.007
	(0.0111)	(0.022)	(0.022)
Affected x Urban	0.0169	0.060	0.039
	(0.0240)	(0.055)	(0.056)
Observations	3,927	2,363	2,363
R-squared	0.051	0.323	0.324

Notes: Mother's primary education takes the value one if she has primary education or lower. Per capita expenditure is in 2000 Rupiah (1 USD~Rp. 10,000 in 2000). All analyses include district fixed effects. All standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 13: Heterogeneous treatment effects by poverty

	(1) Height for age z-score (2000)	(2) Height	(3) Lung capacity (2007)	(4) Cognitive test	(5) Height	(6) Lung capacity (2014)	(7) Cognitive test
Poor household	-0.175**	-1.454***	-0.045**	-0.158**	-0.985***	-0.030***	-0.019*
	(0.076)	(0.356)	(0.019)	(0.067)	(0.340)	(0.011)	(0.010)
In-utero	-0.261	-1.425	-0.085	-0.039	-1.531	-0.012	0.035
	(0.242)	(1.366)	(0.052)	(0.168)	(1.191)	(0.034)	(0.038)
0-12mo	-0.343*	-0.319	-0.068	0.190	-1.070	-0.000	-0.027
	(0.190)	(1.384)	(0.044)	(0.182)	(1.292)	(0.042)	(0.034)
13-24mo	-0.023	1.783	-0.080	0.005	-0.293	-0.016	-0.022
	(0.235)	(1.526)	(0.055)	(0.186)	(1.351)	(0.039)	(0.030)
In-utero x Poor	-0.610	-4.141**	0.011	0.164	-0.713	-0.028	-0.049
	(0.495)	(1.889)	(0.072)	(0.271)	(1.530)	(0.061)	(0.055)
0-12mo x Poor	0.051	0.335	0.034	-0.253	0.876	0.022	0.072**
	(0.317)	(1.642)	(0.062)	(0.246)	(1.647)	(0.046)	(0.037)
13-24mo x Poor	-0.423	-2.865	-0.047	0.042	-0.343	0.016	0.022
	(0.291)	(2.111)	(0.070)	(0.260)	(1.445)	(0.050)	(0.036)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.233	0.636	0.412	0.189	0.459	0.477	0.191
Poorest	0.083	-0.246	0.016	-0.045	-0.383	0.002	0.011
	(0.109)	(0.469)	(0.024)	(0.079)	(0.455)	(0.016)	(0.014)
In-utero	-0.341	-2.529*	-0.079*	-0.024	-1.647	-0.003	0.018
	(0.225)	(1.327)	(0.047)	(0.167)	(1.254)	(0.034)	(0.033)
0-12mo	-0.417**	-0.647	-0.051	0.062	-1.012	-0.006	-0.005
	(0.176)	(1.215)	(0.037)	(0.167)	(1.083)	(0.037)	(0.030)
13-24mo	-0.119	1.112	-0.084*	0.126	-0.276	0.010	-0.015
	(0.200)	(1.102)	(0.051)	(0.175)	(1.163)	(0.033)	(0.027)
In-utero x Poorest	-0.743	-2.373	-0.021	0.393	-0.666	-0.079	-0.028
	(0.636)	(2.435)	(0.107)	(0.349)	(2.771)	(0.072)	(0.060)
0-12mo x Poorest	0.385	1.893	-0.018	-0.006	1.433	0.066	0.047
	(0.415)	(1.654)	(0.077)	(0.338)	(1.802)	(0.055)	(0.042)
13-24mo x Poorest	-0.360	-3.524*	-0.142*	-0.776*	-1.068	-0.128*	-0.008
	(0.476)	(1.924)	(0.086)	(0.436)	(2.111)	(0.068)	(0.053)
Observations	2,371	2,371	1,439	1,876	2,371	2,359	1,979
R-squared	0.234	0.636	0.412	0.191	0.459	0.478	0.190

Notes: Poverty defined as the bottom two quintiles of per capita expenditure. Poorest defined as the lowest quintile of per capita expenditure. All analyses include household characteristics, as well as district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 14: Robustness: The effect of the local economy

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Height for age z-score	Height	Lung capacity	Cognitive test	Height	Lung capacity	Cognitive test
	(2000)		(2007)			(2014)	
In-utero	-0.507**	-3.121***	-0.085*	0.042	-1.799*	-0.021	0.012
	(0.254)	(1.175)	(0.045)	(0.150)	(1.087)	(0.030)	(0.029)
0-12mo	-0.333*	-0.120	-0.055	0.078	-0.633	0.015	0.007
	(0.183)	(1.128)	(0.037)	(0.161)	(1.005)	(0.035)	(0.024)
13-24mo	-0.204	0.536	-0.100**	0.038	-0.496	-0.012	-0.020
	(0.189)	(1.007)	(0.047)	(0.174)	(1.102)	(0.031)	(0.025)
Change in unemployment	-0.003	-0.127	0.001	-0.003	-0.061	-0.009**	0.000
	(0.031)	(0.133)	(0.007)	(0.020)	(0.125)	(0.004)	(0.004)
Observations	2,369	2,369	1,437	1,875	2,369	2,357	1,978
R-squared	0.234	0.636	0.412	0.189	0.461	0.479	0.189

Notes: Change in unemployment is measured at the province level in the child's year of birth. All analyses include household characteristics, as well as district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 15: Robustness: The effect of household unemployment in 1997

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Height for age z-score	Height	Lung capacity	Cognitive test	Height	Lung capacity	Cognitive test
	(2000)		(2007)			(2014)	
In-utero	-0.531**	-3.199***	-0.088*	0.015	-1.939*	-0.023	0.007
	(0.256)	(1.201)	(0.046)	(0.152)	(1.093)	(0.031)	(0.029)
0-12mo	-0.372**	-0.343	-0.056	0.052	-0.766	0.009	0.006
	(0.183)	(1.121)	(0.036)	(0.158)	(1.000)	(0.035)	(0.023)
13-24mo	-0.213	0.429	-0.099**	0.007	-0.749	-0.013	-0.029
	(0.195)	(1.026)	(0.048)	(0.178)	(1.113)	(0.032)	(0.025)
Household head	-0.009	0.683	0.047	-0.047	0.771	0.017	-0.012
unemployed	(0.128)	(0.675)	(0.029)	(0.096)	(0.566)	(0.021)	(0.017)
Observations	2,331	2,331	1,411	1,848	2,331	2,319	1,949
R-squared	0.235	0.637	0.418	0.189	0.463	0.479	0.197

Notes: Household unemployment is a self reported indicator, measured at the household level in 1997. All analyses include household characteristics, as well as district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 16: Robustness: The effect of any household income loss due to economic shocks in 1997

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Height for age z-score	Height	Lung capacity	Cognitive test	Height	Lung capacity	Cognitive test
	(2000)		(2007)			(2014)	
In-utero	-0.518**	-3.153***	-0.083*	0.018	-1.848*	-0.021	0.003
	(0.257)	(1.195)	(0.046)	(0.150)	(1.088)	(0.030)	(0.030)
0-12mo	-0.345*	-0.274	-0.060	0.022	-0.679	0.007	0.006
	(0.186)	(1.137)	(0.038)	(0.160)	(1.016)	(0.035)	(0.023)
13-24mo	-0.209	0.465	-0.093*	0.009	-0.607	-0.011	-0.030
	(0.194)	(1.034)	(0.049)	(0.176)	(1.116)	(0.032)	(0.024)
Income	-0.094	-0.715	0.015	-0.087	-0.535	0.012	0.001
loss	(0.135)	(0.629)	(0.027)	(0.124)	(0.578)	(0.017)	(0.015)
Observations	2,286	2,286	1,389	1,811	2,286	2,274	1,910
R-squared	0.236	0.637	0.414	0.188	0.462	0.478	0.201

Notes: Household income loss is a self-reported indicator, measured at the household level in 1997. All analyses include household characteristics, as well as district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 17: Robustness: The effect of any crop loss in 1997

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Height for age z-score (2000)	Height	Lung capacity (2007)	Cognitive test	Height	Lung capacity (2014)	Cognitive test
In-utero	-0.511**	-3.141***	-0.084*	0.017	-1.828*	-0.023	0.003
	(0.256)	(1.196)	(0.046)	(0.151)	(1.092)	(0.031)	(0.030)
0-12mo	-0.343*	-0.263	-0.060	0.023	-0.670	0.006	0.006
	(0.184)	(1.133)	(0.038)	(0.161)	(1.016)	(0.035)	(0.023)
13-24mo	-0.195	0.484	-0.096*	0.009	-0.570	-0.013	-0.031
	(0.193)	(1.032)	(0.049)	(0.178)	(1.120)	(0.032)	(0.024)
Crop loss	-0.133	-0.333	0.024	-0.008	-0.441	0.022	0.011
	(0.097)	(0.568)	(0.027)	(0.092)	(0.512)	(0.015)	(0.014)
Observations	2,286	2,286	1,389	1,811	2,286	2,274	1,910
R-squared	0.236	0.636	0.415	0.188	0.462	0.478	0.201

Notes: Crop loss is a self reported indicator, measured at the household level in 1997. All analyses include household characteristics, as well as district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.

Table 18: Heterogeneous treatment effects by use of wood stove

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Height for age z-score (2000)	Height	Lung capacity (2007)	Cognitive test	Height	Lung capacity (2014)	Cognitive test
Wood stove	-0.131	-1.066***	-0.005	-0.222***	-0.496	0.000	-0.013
	(0.092)	(0.363)	(0.019)	(0.070)	(0.399)	(0.012)	(0.010)
In-utero	-0.222	-2.715*	-0.108*	-0.031	-1.494	-0.043	0.016
	(0.255)	(1.397)	(0.056)	(0.210)	(1.197)	(0.038)	(0.032)
0-12mo	-0.249	-0.961	-0.070	-0.107	-1.325	-0.010	-0.011
	(0.218)	(1.241)	(0.044)	(0.236)	(1.063)	(0.039)	(0.035)
13-24mo	-0.112	1.165	-0.143**	0.205	-0.878	0.013	-0.013
	(0.287)	(1.292)	(0.059)	(0.238)	(1.364)	(0.033)	(0.038)
In-utero x Wood stove	-0.767	-1.074	0.059	0.246	-0.933	0.066	-0.017
	(0.550)	(1.892)	(0.079)	(0.269)	(1.670)	(0.054)	(0.055)
0-12mo x Wood stove	-0.068	1.778	0.022	0.273	1.556	0.029	0.037
	(0.321)	(1.639)	(0.059)	(0.307)	(1.338)	(0.056)	(0.033)
13-24mo x Wood stove	-0.139	-1.125	0.074	-0.341	0.796	-0.043	-0.018
	(0.341)	(1.753)	(0.066)	(0.288)	(1.784)	(0.052)	(0.038)
Observations	2,320	2,320	1,412	1,834	2,320	2,308	1,936
R-squared	0.235	0.636	0.410	0.195	0.460	0.476	0.197

Notes: All analyses include household characteristics, as well as district, children's year and month of birth fixed effects. Standard errors are clustered at the community level (enumeration area). * p<0.1, ** p<0.05, *** p<0.01.