# Dust and Death: Evidence from the West African

Harmattan\*

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#### Abstract

Using two decades of data from twelve low-income countries in West Africa, we show that dust carried by *harmattan* trade winds increases infant and child mortality. Health investments respond to dust exposure, consistent with compensating behaviors. Despite these efforts, surviving children still exhibit negative health impacts. Our data allow us to investigate differential impacts over time and across countries. We find declining impacts over time, suggesting adaptation. Using national-level measures of macroeconomic conditions and health resources, we find suggestive evidence that both economic development and public health improvements have contributed to this adaptation, with health improvements playing a larger role.

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# 1 Introduction

Environmental factors impact important outcomes related to economic growth and development – including conflict, mortality, disease, and income – in profound ways (Dell et al., 2012; Hsiang et al., 2013; Maccini and Yang, 2009). The links between environment, health, and the ability of people to adapt are particularly salient in low-income countries. First, health is more fragile in these contexts, implying that small changes in environmental conditions could have large impacts on health outcomes. Second, coping mechanisms for adjusting to extreme weather events and other environmental shocks are fewer than in other countries. For example, while migration was a major response to the American Dust Bowl (Hornbeck, 2012), in many low-income countries, individuals do not migrate during seasonal floods despite high returns to migration (Bryan et al., 2014). Third, as low-income countries raise their standards of living, it is critical to examine whether economic growth is accompanied by increased adaptation, mitigating the impacts of environmental shocks.<sup>1</sup> Finally, climate change is likely to exacerbate preexisting inequalities in income, health, and human capital. Examining the effects of weather-related events on such outcomes is therefore crucial in the low-income country context.

This paper presents a large-scale empirical analysis of the impacts of *in utero* dust exposure in Western Africa on mortality and health in early childhood. Our analysis spans two decades, from 1986 to 2006, allowing us to examine evidence suggestive of adaptation responses. The data cover 12 countries, allowing for an exploration of the effects of dust exposure across countries with different economic conditions and public health systems. Our study focuses on a specific source of dust-related pollution in the African Sahel: a yearly trade wind that blows from the Sahara towards the Gulf of Guinea, known locally as the *harmattan*. It carries with it a large amount of dust, mostly quartz, clay, colloids, and fine mica flakes (Besancenot et al., 1997).<sup>2</sup> Recent *harmattan* activity in Nigeria has sparked

<sup>&</sup>lt;sup>1</sup>This has been shown recently in the United States with regards to the adoption of air conditioning as a means to mitigate temperature shocks (Barreca et al., 2016).

<sup>&</sup>lt;sup>2</sup>Abdurrahman and Taqi (1982) give the composition of Saharan dust as silica (50%), alumina (10%), lime

enquiry into whether global warming could be partly responsible for changes in the timing and intensity of the dust storms (Gambrell, 2010).

Our research is aided by the availability of data on dust concentrations taken from NASA satellite data and extrapolation verified by on-the-ground measurements. Our specific measure of dust is PM2.5, or particulate matter whose size is less than 2.5 microns. Our focus on outcomes in early childhood is motivated by research emphasizing this critical period of child development (Cunha and Heckman, 2007). We find compelling evidence that *in utero* exposure to dust affects early life survival. Additional exposure of 10  $\mu g/m^3$  of PM2.5 during each month of gestation on average decreases infant survival by 6.45 percentage points, which is substantial relative to the sample average infant mortality rate of 11.2%.

However, between 1986 and 2006, the effect of PM2.5 dust on mortality shows a steady decline; the effect more than halves during this time period. This suggests that these impacts have been attenuated by adaptation, by the spread of public health services, or by technological advances, among other potential explanations. Using national-level measures on macroeconomic conditions and public health infrastructure, we find evidence that dust impacts are smaller in countries with more developed economies and greater health resources, with health improvements seemingly the more important moderator. At the individual level, we find that non-first-born children are less susceptible to the deleterious effects of dust, and are also more likely to receive compensatory investments at birth and in early life. These results suggest some role for parental "experience" and investments in adaptation, in line with recent work demonstrating that parental investments react to early childhood health shocks (Adhvaryu and Nyshadham, 2016; Almond and Mazumder, 2013; Bharadwaj et al., 2013). Accordingly, we interpret our main results as the effect of dust exposure net of any compensating investments.

<sup>(5%),</sup> ferric oxide (4%), and other salts (5%). It is our understanding that the dust is composed primarily of materials relating to the Earths crust (see e.g. Zhou et al. (2013)). It is possible that man-made forms of pollution have become a larger component of the dust phenomenon over time. In Ibadan, for example, Akinlade et al. (2015) find some evidence of man-made pollution as part of the overall dust composition. However, this is unlikely to affect our main results, as we control for year-by-month fixed effects in all our regression specifications.

It should be noted that our analysis shows the reduced form relationship between the *harmattan* and infant and child mortality across multiple countries and over time. We are not able to distinguish the relative importance of various candidate mechanisms for which there is evidence of links to the *harmattan*, such as damage to infrastructure, reduced rainfall, crop output reduction, or changes to the disease environment (Adefolalu, 1984; Adetunji et al., 1979; Archibong et al., 2017; Besancenot et al., 1997).

Our empirical approach addresses the standard concerns faced by studies that examine the role of pollution exposure on mortality.<sup>3</sup> Our baseline empirical specification includes survey cluster fixed  $\times$  calendar month effects. Clusters approximate villages and are defined uniquely for every wave of the survey. Intersecting these with calendar month dummies means that we control flexibly for seasonal patterns that we allow to differ for every cluster in the data. We include country  $\times$  year  $\times$  month fixed effects that allow for different seasonal patterns by country, as well unobserved heterogeneity at the level of year-by-month-bycountry cells. That is to say, we compare two children from the same survey cluster exposed to varying levels of dust by virtue of being born at different times, over and above any unobserved shocks to mortality that vary by the intersection of country, month, and year. We further add controls for rainfall and temperature during the *in utero* period to isolate the impacts of dust exposure from the impacts of weather phenomena. Our results are also robust to the inclusion of alternative country  $\times$  month of birth  $\times$  year of birth fixed effects and dust point fixed effects, as well as an alternative mother fixed effects approach, in which we compare siblings born at different times, and hence exposed to different dust levels, but who otherwise share similar family and location characteristics. Finally, we check that mother and child characteristics are not affected by dust exposure due to selective fertility.

This paper contributes to the growing body of work in economics on the health impacts

<sup>&</sup>lt;sup>3</sup>A major concern, for example, is sorting. Individuals might sort into areas with lower dust prevalence and characteristics that are associated with sorting could affect early childhood mortality. Similarly, fertility in Africa is seasonal (Dorélien, 2016). Children born in different months of the year could also be exposed to other seasonal variation or environmental agents, which could drive part of the correlation of dust exposure with mortality. There can also be broad secular trends at the national level with declining infant mortality that might affect our results.

of pollution exposure in the developing world (Arceo-Gomez et al., 2016; Ebenstein, 2012; Ebenstein et al., 2017; Greenstone and Hanna, 2014; Heft-Neal et al., 2018; Jayachandran, 2009).<sup>4</sup> Our data, across many West African countries and spanning two decades, allow us to expand the external validity of existing findings. Similarly, our focus on a naturally occurring source of pollution allows our results to be uncontaminated by policies that might coincide with environmental regulations and similar sources of identifying variation.

Another advantage of considering such a broad region over a long time period, using data that record multiple parental investments, is that we are able to test the degree to which people and institutions adapt to the adverse effects of dust exposure. In this regard, our paper is related most closely to Barreca et al. (2016). Even in the case of the health effects of environmental shocks for which there is a large literature (temperature), and in developed country contexts where avoidance behaviors can be reliably measured, existing knowledge on adaptation is quite limited (Deschenes, 2014). Studies on the mortality effects of pollution in poor countries can compare impacts to those found in rich countries and identify socioeconomic gradients, but cannot track adaptation over time or measure avoidance behavior. In other cases, diminishing mortality has not been due to better mitigation, but rather to improved air quality (Arceo-Gomez et al., 2016). As Arceo-Gomez et al. (2016) note, the effects of pollution may be more severe at the higher levels found in poor countries, and the costs of avoidance behavior may be greater. This is true of many types of environmental shocks to health (Burgess et al., 2014; Dell et al., 2014; Kudamatsu et al., 2012).

It is not obvious, therefore, in our context that there should be diminishing effects of shocks over time akin those found in the developed world.<sup>5</sup> Our results show that the effects of fetal dust exposure on the survival of West African children have weakened dramatically in recent years. We show that economic development and health-related improvements, such as higher immunization rates and per capita health expenditure and lower disease prevalence,

<sup>&</sup>lt;sup>4</sup>There exists a similar literature on health consequences of pollution in richer countries. See, for example, Currie and Schwandt (2016); Jia and Ku (2015).

<sup>&</sup>lt;sup>5</sup>Adhvaryu et al. (2015), for example, find no evidence that the effect of *in utero* temperature exposure on adult mental health in Africa has improved over time.

are associated with smaller impacts of dust on infant and child mortality, suggesting potential mechanisms for this decline.

# 2 Background

Broadly, the mechanisms connecting fetal dust exposure to infant and child mortality can be classified as *biological* or *economic*. Biological mechanisms can themselves be divided roughly into indirect effects operating through the health of the mother, direct effects while in the womb, and greater vulnerability to later health insults. Pregnant mothers who inhale dust are at risk of having particulate matter enter their lungs, which can affect the operation of several organ systems (DeFranco et al., 2015). Dust inhaled during the harmattan in particular can carry harmful elemental particles, including heavy metals and trace metals, and can lodge deep in the lungs (Uduma and Jimoh, 2013). Pathways for direct effects in *utero* include interrupted placental development, fetal growth restriction, susceptibility to pre-term birth, heart defects, and reduced weight gain. Ritz and Wilhelm (2008) suggest several effects of air pollution both at birth and afterwards, making children more vulnerable. These include higher risks of: infant death; brain, respiratory, and digestive problems in early life; and heart disease and diabetes in adulthood. It has also been noted in the literature that the harmattan is correlated with outbreaks of meningitis (Besancenot et al., 1997). While the mechanisms remain unclear, the seasonal dust dries the lips and irritates the nasopharyngeal mucosa.

West African populations are generally aware of seasonal correlations between health outcomes and the *harmattan*. Eighteenth-century European observers on the Ghanaian coast observed that fine dust would settle on the skin, giving it a whitish color. Dryness that accompanied the dust harmed vegetables and trees, facilitated the spread of fires, damaged furniture, and irritated the eyes, nostrils, palate, and lips, though the *harmattan* also coincided with declines in infections and epidemics such as smallpox (Dobson and Fothergill, 1781). Several studies have suggested that seasonal dust and dryness might aggravate asthma, carry disease vectors, dry the skin, irritate the throat and eyes, produce catarrh, lead to coughing and bronchitis, and even give rise to sinusitis, pneumonia, and respiratory infections (Abdurrahman and Taqi, 1982; Adefolalu, 1984; Quagraine and Boschi, 2008). Websites and newspapers in West Africa note the existence of these seasonal health issues, as well as other problems such as the greater prevalence of fires, and provide tips on keeping one's skin hydrated.<sup>6</sup> Experts, medical staff, and other popular sources of advice suggest individuals take preventive measures such as wearing thick clothing, staying inside, keeping their food covered, washing their hands more regularly, tending fires more carefully, and washing their faces more often.<sup>7</sup> Whether this popular knowledge translates into preventive action is less clear; for many, the *harmattan* is simply thought of as a nuisance (Adefolalu, 1984), though the seasonal cold may make business start later in the morning.<sup>8</sup> Libraries in Nigeria cope with the dust by closing windows, dusting library materials, and attacking the insects blown in with the dust (Ezennia, 1989).

Economic mechanisms are those that affect the prices, returns, or budget constraints faced by West Africans. Dust storms may reduce incomes generally by damaging infrastructure, disrupting flights, reducing rainfall, increasing the frequency of fires, and affecting crop outputs (e.g. Adetunji et al. (1979); Jenik and Hall (1966)), though there is some evidence that the *harmattan* aids soil fertility at the fringes of the Sahara (Adefolalu, 1984). Some of the most well-known airplane crashes in Nigerian history have occurred during the *harmattan* (Abdurrahman and Taqi, 1982). Reduced parental health may impact labor productivity, and hence income. If infant health is initially harmed by fetal dust exposure, it may reduce the returns to complementary parental investments or, conversely, increase the urgency of interventions that might ameliorate these effects (Cunha et al., 2010). Seasonal increases in

<sup>&</sup>lt;sup>6</sup>http://www.thisdaylive.com/articles/tips-to-keeping-your-skin-hydrated-during-the-harmattan/ 106015/, http://allafrica.com/stories/201311180452.html, http://www.thisdaylive.com/ articles/asthma-and-the-harmattan/128687/.

<sup>&</sup>lt;sup>7</sup>http://www.aitonline.tv/post-harmattan\_haze\_\_avoiding\_the\_dry\_season\_blues, http:// howtotellagreatstory.com/2012/11/the-story-of-harmattan-part-1-it-chillsdriesalters-lifestyle/.

<sup>&</sup>lt;sup>8</sup>http://allafrica.com/stories/201311180452.html

patient visits may create problems of congestion and shortages in public health systems that have limited capacity. Alternatively, dust storms may either increase the costs of accessing health services, or may prompt a public health response that makes these services more available.

# **3** Empirical strategy

To test for the effects of dust exposure during the *in utero* phase on neonatal, infant, and child mortality, we use ordinary least squares (OLS) to estimate the following equation:

$$Mortality_{icvym} = \beta Dust_{cvym} + x'_{icvym}\gamma + \delta_{cvm} + \eta_{cym} + \epsilon_{icvym}.$$
 (1)

Here, *Mortality*<sub>icvtm</sub> is an indicator for child *i*, born in month *m* in year *y*, whose mother is surveyed in cluster *v* in country *c*. *v* can loosely be thought to index "villages." Neonatal mortality is death within the first month of life. Infant mortality is within the first twelve months, and child mortality is within the first 60 months. In the main results,  $Dust_{cvym}$  is the level of dust pollution recorded at the dust point closest to *v* in the 9 months preceding month *m* in year *y*. Thus, we assume a gestation period of 9 months for all births.<sup>9</sup> We use a cumulative exposure measure, summing the dust reading for each month during gestation.  $\beta$  is the coefficient of interest, and we expect it to be positive.

 $x_{icvym}$  is a vector of controls. In our preferred specification, it includes average rainfall 9 months prior to birth, average temperature 9 months prior to birth, the squares of these terms, the interaction between average rainfall and temperature, child birth order, child is a female, child is a multiple birth, mother's age at birth, mother's age at birth squared, mother's years of education, and mother's religion. We acknowledge the concern that dust

<sup>&</sup>lt;sup>9</sup>This is unfortunately not a directly testable assumption in our data; however, we do show robustness to using the full year of exposure prior to birth and controlling for exposure in the year after birth.

pollution could itself affect levels of rainfall and temperature, and so controlling for these could lead to overadjustment bias (Schisterman et al., 2009). Further, the data we have only allow us to control for monthly averages, and so we cannot control for degree-day measures that could capture more subtle nonlinearities. For these reasons, many of the results in our paper are reported both with and without these weather controls.

We also include two additional and important sets of fixed effects. First, cluster × calendar month of birth fixed effects ( $\delta_{cvm}$ ) account for general seasonal variation in birth outcomes that may vary across survey clusters. These also absorb any unobserved drivers of mortality that happen to correlate with dust over space, since they sum to a cluster fixed effect. Second, country × year-of-birth × month-of-birth fixed effects ( $\eta_{cym}$ ) account for time-specific shocks such as economic crises and disease outbreaks. These also account for other possible unobserved trending variables that may vary over time by country. These may include, for example, supply-side health initiatives such as immunization campaigns.

The identification in this paper is accordingly derived from variation in PM2.5 exposure across children from the same survey cluster whose exposure to dust *in utero* was determined by differential birth timing, but cannot be explained away as the effect of other country-byyear-by-month-specific patterns of unobserved heterogeneity, by village-specific patterns of seasonality, or by very flexible functions of rainfall and temperature. We cluster standard errors by dust point; as a robustness exercise we show that clustering by administrative regions has little effect on the results.

We also estimate several alternative versions of the above model. For example, to study heterogeneity in impacts of dust on mortality, we include interactions of dust exposure with a dummy for the child not being the first born to the mother as a measure of parental experience. We perform similar heterogeneity tests using a gender dummy and a wealth index measure. Additionally, to study how the effect of dust on mortality has changed over time, we include the interaction of time trends with the dust variables. Finally, we interact dust with national-level measures of economic development (e.g., GNI, poverty gap, % of GDP derived from agriculture, tractors per land area) and health infrastructure (e.g., per capita health expenditure, % health expenditure out of pocket, maternal mortality rates, child anemia rates, DPT immunization rates, rural access to safe water) from the World Bank World Development Indicators to study the degree to which these aggregate indicators moderate the impact of dust on mortality.

As discussed above, the physiological pathway by which dust can directly or biologically impact child health and mortality has been established; however, it is possible that dust exposure can, in addition, affect incomes, parental investments, and other behavioral responses, and by way of these mediative factors indirectly impact mortality outcomes further. Accordingly, in the above models, we interpret  $\beta$  as the *composite* effect of dust on mortality, reflecting the sum of any direct biological impacts and the impacts of any subsequent reinforcing or mitigating investments or behaviors on the part of the parents.

To examine the mediating role of parental investment responses, we also estimate a version of equation (1) in which we replace the dependent variable with measures of earlylife and at-birth investments as well as health measures later in childhood. To the extent that we find evidence of impacts of dust on these parental investment responses, our interpretation of the effect of dust on mortality in our main results, as well as any effect on later childhood health among survivors, is *net* of these behavioral responses to dust exposure.

# 4 Data

In this section, we describe the data sources used in the analysis. Additionally, where necessary, we describe the construction of the variables of interest.

### 4.1 Dust

We take data on dust from the International Research Institute for Climate and Society at Columbia University.<sup>10</sup> These data are available for the period from August 1985 to December 2006 at latitude-longitude intervals of  $1.25^{\circ}$  by  $1.25^{\circ}$ . These will allow us to compute fetal dust exposure for children born between April 1986 and December 2006. Constructed using NASA satellite measurements, the dust data report hourly concentrations of  $PM_{2.5}$ , or particulate matter smaller than 2.5 micrometers. We convert these data to monthly format in order to merge them with the Demographic and Health Survey (DHS) datasets described below. Particulate concentrations are reported in hundreds of micrograms per cubic meter.

These data are the result of the NMMB/BSC Dust model, which was developed at the Barcelona Supercomputing Center in collaboration with the NASA Goddard Institute for Space Studies and the National Centers for Environmental Protection (Pérez et al., 2011). This model simulates soil dust aerosol emission, which has been validated using existing satellite and *in situ* data over this region (García-Pando et al., 2014; Pérez et al., 2011).<sup>11</sup> Additional information on these data is presented in Stanley (2013). This dust model also predicts PM10 pollution; hence, while we primarily use PM2.5 as our main dust exposure variable, we also show our main results using PM10 as the dust variable.

## 4.2 Demographic and health survey data

Our principal outcomes of interest are taken from 128 DHS datasets from West Africa. These have been collected from several West African countries since the mid-1980s. We use all standard West African DHS surveys for which geographic coordinates are available; these coordinates are needed for merging with the data on dust. Our sample, then, includes Benin,

<sup>&</sup>lt;sup>10</sup>The data can be downloaded from http://iridl.ldeo.columbia.edu/home/.nasa\_roses\_a19/.Dust\_model/.dust\_mon\_avg/.dust\_pm25\_sconc10\_mon/

<sup>&</sup>lt;sup>11</sup>The correlation between the soil dust aerosol component of this model and aerosol optical depth (AOD) is around 0.6 (Ceccato et al., 2014; Pérez et al., 2011).

Burkina Faso, Ghana, Guinea, the Ivory Coast, Liberia, Mali, Niger, Nigeria, Senegal, Sierra Leone, and Togo.

The data come in four formats:

- 1. The *Individual Recodes* survey women who are aged between 15 and 49. These nationally representative cross-sections contain several variables that we use. These include the woman's year of birth, her level of education, whether she lives in a rural area, her age, her occupation, the occupation of her partner, and her self-reported religion.
- 2. The *Births Recodes* are complete birth histories of the women surveyed in the individual recodes. We use these data for our main results. In particular, we make use of the child's year and month of birth, birth order, an indicator for a multiple birth, a dummy for female, and the length of the child's life.
- 3. The *Children's Recodes* are similar to the births recodes, but contain a larger amount of information about a smaller number of children. Women are asked about births in the previous five years. In addition to controls available in the births recodes, these include early life investments such as vaccinations and breastfeeding. Additional prenatal investments are also recorded, including care from doctors and the circumstances of the child's birth.
- 4. The *Geographic Datasets* record latitude and longitude coordinates for the survey clusters in the data. We use these coordinates to merge children to the nearest point in the dust data, assigning each child the level of dust of the nearest point. These are shown in Figure 1.

### 4.3 Matching DHS to dust data

Figure 1 shows how DHS clusters are matched to their closest dust grid points. Shaded small dots are DHS clusters (the variation in colors represents how much average dust exposure these clusters have), while the black larger dots are the dust points on the grid.

Figure 1: DHS clusters and dust points



The small, square dots are DHS survey clusters. These are plotted in different shades of grey that represent deciles of mean dust, ranging from dark (most dust) to light (least dust). The larger, round, black dots are dust points.

### 4.4 Country-level World Bank Indicators

As discussed above, we introduce World Development Indicators from the World Bank open access data archives to investigate the degree to which these aggregate factors moderate dust impacts on mortality. These data are often recorded only intermittently for each country in our primary dataset. Accordingly, we impute missing values for each variable with the nearest future value recorded. This allows us to use as many observations as possible. We check that all results using these data are robust to restriction to only unimputed data as well. The indicators used fall into two broad categories: economic development (GNI, poverty gap, % of GDP derived from agriculture, tractors per land area) and health infrastructure (per capita health expenditure, % health expenditure out of pocket, maternal mortality rates, child anemia rates, DPT immunization rates, rural access to safe water).



Figure 2: Infant Mortality by Country

Graph shows infant death by year of birth and country. Infant mortality calculated from raw DHS data without adjustment. Country abbreviations are as follows: BF = Burkina Faso, BJ = Benin, CI = Ivory Coast, GH = Ghana, GN = Guinea, LB = Liberia, ML = Mali, NG = Nigeria, NI = Niger, SL = Sierra Leone, SN = Senegal, TG = Togo.

# 4.5 Summary statistics

Table 1 shows the summary statistics used in this paper. Infant mortality over the births in our data during the 1986-2006 period for all the countries considered averages 11.2%. Child mortality is relatively high in this region, even by the standard of developing countries. However, as Figure 2 suggests, most countries in our data set have experienced substantial declines in infant mortality over this period. Figure 2 also suggests substantial heterogeneity in infant mortality across West African nations: some of the lowest rates are seen in Ghana and Senegal (averages of 7.7% and 8.1% respectively), while the highest averages are seen in Mali and Niger (13.8% and 13.3% respectively).



Graphs show PM2.5 concentrations averaged for the sample at the year x month level, showing raw levels as well as residuals derived from removing fixed effects as indicated in the figure.

The average monthly PM2.5 concentration is around  $44\mu g/m^3$  over the entire sample.<sup>12</sup> To put this in perspective, the average annual PM2.5 concentration in Southern California was 20 in 1999 (EPA), while the recent estimates from New Delhi put its annual PM2.5 concentration at 153 (WHO 2014). However, this average measure hides substantial within-year variation in PM 2.5. As Figure 3 suggests, there are stark seasonal patterns in PM2.5, with the highs typically observed January through April. Despite anecdotal evidence that the *harmattan* is becoming more severe over time or that it now reaches parts of West Africa that were unaffected within living memory (e.g. Abdurrahman and Taqi (1982)), Figure 3 shows that for the sample period (1986-2006) there appears to be no obvious yearly trend

<sup>&</sup>lt;sup>12</sup>That is, 4.11 × 100 (Table 1 reports 100s of  $\mu g/m^3$ )  $\times \frac{1}{9}$  (i.e. per month *in utero*).

in PM2.5 levels.

Table 1 also shows some of the key characteristics of mothers in our sample. Mothers overall have low levels of education (an average of 1.6 years), and are young. The average age at the child's birth is nearly 26 years, but note that the average birth order observed in the data is 3, suggesting women typically have 3 children by age 26 in the sample. We show robustness to controlling for these characteristics in our main results as attributes such as maternal education and age can have associations with child health.

# 5 Results

Table 2 shows the effects of *in utero* dust exposure on neonatal (within 28 days of birth), infant (within a year of birth), and child (within 5 years of birth) mortality. The results indicate that there is little evidence of impacts of dust on mortality in the first month, but there are substantial effects on both infant and child death. Comparing results across columns, we find that including controls for rainfall and temperature exposure over the same period increase the magnitude and significance of the impacts of dust exposure; however, the inclusion of additional controls for child and mother characteristics has little impact on the effect size and precision, suggesting that it is unlikely that omitted variables correlated with dust exposure are driving our results (Altonji et al., 2005).<sup>13</sup>

We focus our interpretation of magnitudes now on the results for infant mortality as the interpretation of the coefficients for child mortality are similar. Column 6 of Table 2 shows that the effect of a one standard deviation increase in *in utero* PM2.5 leads to a 1.09 percentage point increase in infant mortality, or roughly 9.7% of the mean.<sup>14</sup> The following thought experiment helps to put the magnitude of the estimate impacts in perspective: con-

<sup>&</sup>lt;sup>13</sup>In additional results (not reported), we control for the number of battle deaths during the *in utero* period within 100km of the child's survey cluster as reported by the UCDP Geo-referenced Event Dataset (UCDP GED) version 2.0. This has little effect on the results. Battle deaths data only begin in 1989, which reduces the sample.

 $<sup>^{14}</sup>$ To see this, multiply the standard deviation of cumulative dust exposure from Table 1 (1.52) by the coefficient (7.170) and divide by 10 to convert from deaths per 1,000 to percentage points.

sider reducing exposure to levels deemed acceptable by the EPA's PM2.5 national standards in the US  $(15\mu g/m^3)$ . This would reduce total exposure over an entire pregnancy from a mean of 411  $\mu g/m^3$  to 135  $\mu g/m^3$ , reducing infant mortality by 1.98 percentage points, or 17.8% of the mean.<sup>15</sup>

We can put these results in the context of other interventions or environmental shocks considered in the literature as well. Our estimates are smaller than some in the literature, but still comparable. These include the 3.27 percent decline in American infant mortality due to reductions in the use of bituminous coal for heating (Barreca et al., 2016); the 3.5 percentage point increase in mortality due to a six-month malaria epidemic (Kudamatsu et al., 2012); the 1.8 percentage point effect of democratization in Africa (Kudamatsu, 2012); the elasticity of rural infant mortality with respect to aggregate income of -0.33 in India (Bhalotra, 2010); or the 7.3% annual mortality increase due to a one standard deviation increase in high temperature days in India (Burgess et al., 2014).

On the other hand, our estimates are of equal or larger magnitude to those found in a few closely related studies. The effect we find of a one standard deviation increase in dust exposure is similar to the 1.2 percent effect on cohort size of wildfires in Indonesia (Jayachandran, 2009). While Arceo-Gomez et al. (2016) find that a 100  $\mu g/m^3$  increase in PM10 in Mexico City increases deaths by 0.23 per 1000, our results suggest that the same increase in PM2.5 would increase fetal death by 7.170 per 1000. In Appendix Table A1, we replace PM2.5 measures with PM10. We find a corresponding effect of 1.444 that compares directly to their estimate of 0.23.<sup>16</sup> Our effect is also larger than the 0.24 to 0.40 deaths per thousand brought on by a 1% decline in GDP in poor countries (Baird et al., 2011) or the insignificant effect of air regulation in India (Greenstone and Hanna, 2014).

In the bottom panel of Table 2, we check that in utero dust exposure is indeed the

<sup>&</sup>lt;sup>15</sup>To see this, multiply the reduced dust exposure (276) by  $\frac{1}{100}$ , since dust is measured in 100s of  $\mu g/m^3$  in the regression. Multiply this by the coefficient 7.170 and divide by 10 to convert from deaths per 1,000 to percentage points.

 $<sup>^{16}</sup>$  More precisely, they report that a 1  $\mu g/m^3$  increase in PM10 in Mexico city increases deaths by 0.23 per 100,000.

driving force behind our main results and is not simply a proxy for dust experienced after birth by including dust exposure after birth as an additional control. When using neonatal or infant mortality as an outcome, we control for dust in the first year of life (columns 1-6). When using child mortality as an outcome, we control for dust in the first five years of life (column 7-9). We use fixed blocks of time to compute this exposure, even for children who die before the block of time has ended. In both cases, the main effect of dust *in utero* remains significant and of nearly identical magnitude to those in the top panel. The effect of dust during the first year of life on infant mortality is statistically insignificant. Controlling for PM2.5 during the first 60 months of life does not diminish the effect of *in utero* dust on child mortality, but does have its own independent effect on the probability that a child dies.<sup>17</sup>

### 5.1 Mechanisms

While it is beyond the scope of this study, and beyond the limitations of the available data, to disentangle all possible mechanisms, we can use heterogeneous treatment impacts and the responses of additional outcomes to help reduce the set of plausible explanations and to understand some possible policy implications regarding the mitigation of dust effects.

#### 5.1.1 Heterogeneous response

Table 3 explores heterogeneity in the impacts of dust on infant and child mortality. In the top panel, we interact dust exposure with a dummy for the child not being the first born to the mother. This serves as a proxy for parental experience. If we believe that parents with greater experience might be better able to respond to dust exposure with impact mitigating behaviors, we would expect that impacts would be smaller for later born children. Indeed, we find that impacts of dust on mortality are almost completely offset for later-born children.

Next, we interact *in utero* dust exposure with gender. We find that the dust impacts are larger for girls, but only with respect to child mortality. Given that medical research suggests

 $<sup>^{17}</sup>$ We have tested whether in utero dust exacerbates the effects of dust after birth. The interaction is positive but insignificant (not reported).

that male fetuses are more fragile than females, gender heterogeneity in the biological impacts of dust are unlikely to produce these results (Gualtieri and Hicks, 1985; Kraemer, 2000). Gender-biased survival could be contributing to the observed heterogeneity. That is, if only relatively healthy boys survived to birth (or past the first year of life), this could explain their relatively better post-birth outcomes (see Dagnelie et al. (2018) for an example). However, we find no evidence of this; we show below in Appendix Table A8 that there is no evidence that dust impacts the gender composition of births. Finally, gender heterogeneity in child mortality could be due to gender-biased parental investments in early-life. We show evidence below of compensating investments that offset the effects of dust exposure, but in additional heterogeneity results (reported in Appendix Table A9) we find no evidence of compensatory investment responses being weaker for girls.

In the next three panels, we include additional interaction terms with variables that proxy for maternal experience. In particular, we include mother's age, mother's education, and dust exposure of the first-born sibling. The maternal experience interpretation we give above is supported in the sign of the interaction between maternal age and dust, which is negative and significant. It is not, however, confirmed in the interactions of maternal education or dust exposure of the older sibling. Both interactions are small and statistically insignificant.

We also report results for heterogeneity by wealth. Though we find no evidence of heterogeneity by wealth in infant mortality, the effect of dust on child mortality is significantly reduced for children whose households are wealthier, according to the DHS's wealth index.<sup>18</sup> These results are consistent with wealthier households being more able to respond to dust shocks with compensatory investments, with these investments more likely to be reflected in later childhood mortality.

Finally, we interact dust exposure with a rural dummy. We find that the response of

<sup>&</sup>lt;sup>18</sup>This index is made available in the DHS data and is not constructed by the authors. It aggregates the assets owned by the household using factor analysis. A greater score on the index indicates ownership of more items such as radios or motorcycles. We lose sample size in this specification because it is missing for a large number of observations.

infant mortality to a given amount of dust exposure is greater in rural areas. That is, the interaction of rural status and dust in utero is positive. This suggests that, though urban areas might be more polluted, offsetting factors such as better access to public health investments dominate in these places.

### 5.2 Behavioral responses and other outcomes

Having established a reduced form effect of PM2.5 on infant mortality and its robustness, we next document whether this effect is mediated by behavioral responses of parents. The top panel of Table 4 examines whether dust exposure *in utero* affects subsequent parental investment choices. We find that greater dust exposure leads to greater health investments, particularly in early life.

Parental investment responses to dust exposure can arise from two primary sources. First, this could be a result of greater access to health care after a high dust period. If high dust periods are followed by government or NGO intervention to increase the availability of services such as health centers, this could explain part of the effect. The second explanation could arise from the idea that parents invest more in weaker children in the post natal period. This "compensatory" or mitigating behavior contrasts with the "reinforcing" behavior of parents found in some developing country settings where parents have limited resources and choose to invest only in children with the highest "potential" (Adhvaryu and Nyshadham, 2016; Almond and Mazumder, 2013; Bharadwaj et al., 2017).<sup>19</sup>

The middle panel of Table 4 examines these behavioral responses further and provides more direct evidence about the relative importance of "access," relative to post-birth parental responses. We find less evidence of impacts on prenatal and at-birth investments, such as antenatal visits for pregnancy and delivery in a hospital. We only find significant impacts on BCG vaccination which need not be administered at birth, but rather can be administered

<sup>&</sup>lt;sup>19</sup>Our results are suggestive of compensatory investment, but our data do not allow us to evaluate whether parents respond specifically to the shock that affects the child (in this case, dust exposure) or to revealed child health after birth (such as low birth weight or bouts of illness).

up to 6 weeks after birth. Since the prenatal and at-birth investment decisions are made before the child is born, this would capture the effect of dust events on access to health centers that provide vaccinations and on access to doctors who can attend to the delivery. Rather, it is postnatal investments (vaccinations) that respond in Table 4. This is suggestive of parents undertaking compensatory investments once child quality is revealed, rather than facing greater access to investments or anticipating health effects to which they will later need to respond.

Even with compensating investments after birth, it is possible that the impacts of dust exposure will not be completely offset and will still result in lower health even if the child survives. This is explored in the bottom panel of Table 4, where we examine the role of *in utero* dust exposure on various measures of early childhood health among survivors. Many of these measures, notably weight for height, weight for age, and height for age represent measures of nutrition and respond negatively. Birthweight responds negatively, but is estimated less precisely due to the smaller available sample for this outcome.

Lastly, we check whether the evidence of smaller impacts of dust on mortality among later born children presented in Table 3 is in part driven by stronger compensatory investment responses by these more experienced parents. Table 5 reports estimates of heterogeneous impacts of *in utero* dust exposure on subsequent parental investment responses and health measures for surviving children. Indeed, we find that compensatory investment responses are stronger for non-first-born children. Furthermore, we find some suggestive evidence of compensatory responses in prenatal and at-birth investments as well, consistent with parents being more able to predict adverse impacts of dust exposure after accumulating experience with previous children. Nevertheless, we do not find evidence that these stronger compensatory responses lead to better subsequent health outcomes among later-born surviving children.

# 5.3 Effects over time and across countries

Having established the basic result that dust exposure is harmful to children and that direct biological impacts are only partially offset by parental responses, we next exploit the fact that our data spans 20 years between 1986-2006 to see how the impact of dust on mortality has changed over time.





Equation (2) is estimated for each year and the coefficients on dust and associated confidence intervals are plotted.

Figure 4 shows estimates of the effects of dust on infant mortality for each year in our sample. Because this prevents us from including some base controls such as year fixed effects or trends, we estimate a more parsimonious specification:

$$Mortality_{icvym} = \beta_y Dust_{cvym} + x'_{icvym} \gamma + \delta_{cm} + \phi_{ct} + \epsilon_{icvym}.$$
(2)

Each point on Figure 4 is a regression coefficient of the effect of *in utero* exposure on infant

mortality for a given year, or  $\beta_y$ . For each estimate, the sample includes only children born in that year.  $\phi_{ct}$  is a fixed effect for the DHS survey conducted in year t in country c; in our baseline regression, this is absorbed by the fixed effects for survey clusters. Other variables and parameters are as in our baseline specification.

Figure 4 shows a clear pattern of a decline in the impact of dust on mortality. The impact of dust appears to decrease by more than half over this time period. Table 6 confirms these effects by showing an interaction of dust exposure with a linear time trend; the interaction is negative, statistically significant, and shows a rapidly diminishing effect. This finding suggests adaptation, other supply-side health responses, or health technology improvements over time that have helped attenuate these negative impacts.

A more direct test of this is in Figure 5, in which we interact dust with various countrylevel World Development Indicators from the World Bank, in sequence. We investigate the degree to which two groups of macroeconomic factors moderate the impacts of dust on mortality: economic development (GNI, poverty gap, % of GDP derived from agriculture, tractors per land area) and health infrastructure (per capita health expenditure, % health expenditure out of pocket, maternal mortality rates, child anemia rates, DPT immunization rates, rural access to safe water). Figure 5 shows that countries with higher economic development (GNI), more mechanized agriculture (tractors), higher health expenditure, and greater public health resources (DPT immunization) exhibit smaller impacts of dust on mortality. On the other hand, countries with a higher out of pocket share of health expenditure, a larger poverty gap, and higher child anemia rates exhibit larger impacts of dust on mortality. The indicator variables are standardized across countries and time to make coefficients more comparable. That is, GNI, maternal mortality, access to safe water, and health expenditure have the strongest moderating effects for one standard deviation changes.<sup>20</sup>

To better compare the relative importance of economic factors and health infrastructure measures in moderating impacts of dust on mortality, we construct mean effects measures

 $<sup>^{20}\</sup>mathrm{Analogous}$  regression results are presented in Tables A11 and A12.



#### Figure 5: Individual macroeconomic moderators

Coefficients on the interaction between dust and each macroeconomic moderator variables and associated confidence intervals are plotted. Regressions for each moderator variable are run in sequence including one moderator variable as a main effect and interacted regressors at a time. Dependent variable is infant mortality.

for each group and use these in the same heterogeneity specifications. The mean effects measures are standard normal transformations of linear indices of the variables in each set of factors, with exacerbating measures reoriented to allow the mean effects to be interpreted as summarily mitigating measures. That is, the economic mean effect measure is constructed by adding GNI and tractors and subtracting poverty gap and agricultural intensity, with all variables having been normalized to be N(0, 1) before aggregation. Table 7 shows that, while both improvements in economic and health factors are associated with smaller impacts of dust on mortality, health factors are more strongly mitigative. In fact, in columns 1 and 4 we see that, when including both economic and health mean effects in the same regression,

Results from full specification regressions of infant mortality on dust and interaction with each national-level variable in sequence. Coefficients represent contribution to dust impact on deaths per 1000 children of one SD change in (imputed) national-level variable.

health factors continue to significantly mitigate dust impacts, but economic factors no longer seem to matter. These results suggest that improvements in health infrastructure might help to alleviate impacts of dust exposure; perhaps, more so even than general economic development. It is possible that improvements in health infrastructure contributes to the attenuation in dust impacts on mortality over time depicted in Figure 4 and Table  $6.^{21}$ 

Lastly, we demonstrate robustness of the analysis of impacts over time to functional form, ensuring that secular trends in mortality as a whole are not driving this evidence of adaptation. We collapse the data to the region  $\times$  month of birth  $\times$  year of birth level, and construct a dependent variable equaling the natural log of the proportion of live births who died in infancy (0-1 years) or childhood (0-5 years). We regress this variable on our regressor of interest (*in utero* dust exposure) as well as the same controls used in the baseline analysis. The coefficient on dust exposure then tells us the percent impact on mortality (as opposed to the impact expressed in percentage points that results from the baseline specification). We also interact the dust exposure variable with the child's year of birth to test for differential impacts across cohorts (i.e., across time). The main advantage of doing the time analysis in this modified specification is that we are able to test whether the effect of dust on mortality was changing in percentage terms. This is important because, while we know that the impact of dust is declining over time in levels, the overall rates of infant and child mortality also declined dramatically during this period. Thus, it is not clear that there was a *percentage* decline in the impact from the baseline analysis; but the present analysis allows us to test precisely for this differential percentage impact. Note that the year of birth is normalized to 0 for the first cohort in our sample for ease of interpretation.

Table 8 displays the results of this analysis. With no additional controls, there is a positive (3.8 percent) but insignificant effect of *in utero* dust exposure on infant mortality; the impact on child mortality is slightly larger, about 5.5 percent, and significant at the 5 percent level. When country by year trends are added, precision in the main effect specification diminishes,

 $<sup>^{21}{\</sup>rm We}$  unfortunately cannot test for this specifically, given that we have limited data on changes in these macroeconomic conditions over time.

but in specifications capturing impacts over time, for both infant and child mortality, there is a large and significant impact (9-10 percent) on both infant and child mortality in early cohorts. This impact declines considerably with time. The linear time interaction shown here indicates that for each additional year, the impact on mortality declines by more than half a percent. These results confirm the hypothesis that not only is the size of the mortality impact declining over time, but it is declining in percentage terms as well, despite the fact that mean infant and child mortality rates declined across most of the countries studied during our sample period.

### 5.4 Additional Checks

#### 5.4.1 Robustness checks

In Table 9, we consider alternative sets of fixed effects. Our aim is to show that restricting identification to narrowly-defined comparison groups continues to yield our main results, even if we change the definition of the group used. In column 1, we include fixed effects for country  $\times$  month of birth, for year of birth, and for DHS Cluster, alongside country linear time trends. In column 2, we add country-specific quadratic time trends to this. In column 3, we add DHS survey fixed effects (specific to country and year of survey) and replace DHS cluster  $\times$  month fixed effects with dust point ID fixed effects. Column 4 adds country  $\times$  year of birth  $\times$  month of birth fixed effects along with dust point ID fixed effects and DHS survey fixed effects for mothers, alongside fixed effects for country  $\times$  year of birth and country  $\times$  month of birth. Finally, in column 6, we add cluster  $\times$  month of birth effects. Overall, the results are of the same sign and magnitude as our main results and remain, in general, statistically significant.

We perform several additional tests for robustness in the Appendix. The sign and significance of our results are largely replicable when we replace PM2.5 measures with PM10 measures in Appendix Table A1. Appendix Table A1 is organized the same way as Table 2 (omitting the middle columns with weather controls but no additional controls) and shows that PM10 exposure *in utero* leads to greater infant and child mortality. The magnitudes are smaller. This is to be expected, as PM2.5 is known to cause greater harm (Schwartz and Neas, 2000).<sup>22</sup>

In Appendix Table A2 we show that alternative definitions of *in utero* dust exposure provide similar results. It is possible, for example, that it is the intensity of exposure rather than its mean that matters (e.g. Hansman et al. (2015)). High dust exposure measured instead as the number of months in which dust exposure was at least one standard deviation above the cluster average predicts a statistically significant increase in both infant and child mortality. Similar results are obtained if the dust level during the month of greatest exposure during pregnancy or the log of accumulated dust are used. Controlling for dust variability (the standard deviation of monthly dust exposure *in utero*) does not render our main result on accumulated dust insignificant; nor does controlling for the dust level during the month of greatest exposure.

In Appendix Table A3, we account for the fact that the spatial resolution of the dust data is more coarse than that in the DHS in two ways. First, we weight observations by the inverse of the distance to the nearest dust point. Second, rather than joining each DHS cluster to the nearest point in the dust data, we use bilinear interpolation to assign it a weighted average of the dust recorded at the four dust points that surround it. Both exercises give results very similar to our baseline.

In Table A4, we show that our results are not dependent on a linear specification. Using deciles of dust exposure shows that, relative to the omitted, lowest-exposure decile, higher treatments of *in utero* dust raise mortality, particularly in the three greatest deciles. Appendix Table A5 explores the sensitivity of the results to excluding survey clusters in which dust exposure is greatest or lowest. Throughout, our results remain stable. The effect is larger in the clusters where mean dust exposure is greater, consistent with the possibility that mothers exposed to greater dust over their lifetimes are more vulnerable to exposure *in* 

 $<sup>^{22}</sup>$ The strong correlation between PM2.5 and PM10 dust prevents us from disentangling their relative effects in the same empirical specification (Brook et al., 1997).

*utero*, or with the possibility that *in utero* dust exposure makes children more vulnerable to later dust exposure in childhood.

In Appendix Table A6, we disaggregate dust exposure by trimester. The magnitude and significance of dust exposure in the first trimester are greater than exposure for the second and third trimesters. This is consistent with other studies that have found larger effects on mortality, birth weight, and later-life outcomes for insults occurring early in pregnancy (Almond and Mazumder, 2011; Camacho, 2008). This also suggests that nutrition is not the sole source of the effects we find, as fetal health is particularly sensitive to nutrition in the final trimester (Almond et al., 2011).

In Appendix Table A7, we show that our results are robust to clustering by administrative region as recorded in the DHS, rather than the nearest point in the dust data. These regions are typically second-level administrative units such as provinces and states. Indeed, standard errors are largely indistinguishable using this approach.

In Table A10, we replicate the main results removing one country at a time from the sample. The coefficient estimates are fairly stable in terms of magnitude and precision, with the exceptions of the removal of Nigeria and Niger, both of which cause the estimates to shrink slightly (though these are not significantly different from the other estimates).

The countries in our sample were not all surveyed the same number of times, nor were they all surveyed in the same years. However, this creates only minor changes in sample composition over time, because our main data consists of retrospective birth histories. A child in our data may be born in the survey year or in any prior year. In Figure A1 in the appendix, we show the share of births in each year that come from each of the countries in our data. It is clear from this figure that there is not much evidence of major changes in the sample over time. There are two minor exceptions to this. First, since the most recent survey we can use from Togo took place in 1998, this country ceases to be a part of the dataset after that year. Second, the most recent DHS surveys from Nigeria have had large sample sizes, and so that country is a growing share of the sample in more recent years. Because the location we code for the child is based on the mother's location at the time of the survey, this could introduce potential measurement error in dust exposure. In Table A13 in the appendix, we restrict the sample to mothers who have never moved from their current place of residence – a question that is asked directly in the DHS. This restricted sample yields similar results in magnitude to our baseline results, although with less precision. The loss of precision is as expected, since the sample size is nearly halved. This suggests, first, that selective out-migration or measurement error based on using current GIS data from the DHS do not bias our results. Second, it is evident that the effect of dust exposure for those incapable of migration as an avoidance behavior is not different from that of the whole sample.

While our principal specifications account for birth order, they do not account for sibship size. In Table A14 in the appendix, we take three different approaches to address this. First, we restrict the data to only first births, since these pose no issues with replacement fertility. Second, we include sibship size fixed effects. Third, we add an interaction between sibship size and dust exposure. This table broadly suggests that sibship size considerations are not a major factor in explaining our results. Restricting the sample to first births lowers precision due to a drastic reduction in sample size.

#### 5.4.2 Selective fertility and survival to birth

Two forms of sample selection might be contributing to some portion of our results: selective survival of only some fetuses to term, and selection of the types of parents whose fertility coincides with variations in dust exposure. The DHS do not collect information on miscarriages in a way that would permit us to directly measure whether death *in utero* responds to dust exposure. However, the fact that our estimated effects are similar when controlling for mother fixed effects in Table A9 above suggests that selection by parental characteristics is unlikely to explain our results.

Further, we perform analyses similar to those in Buckles and Hungerman (2013), demon-





The top figure shows the percentage of total births that occur in a given month within each quintile of dust clusters. The bottom figure shows the average education of mothers whose births occur in a given month within each quintile of dust clusters.

strating that neither parental characteristics nor child characteristics correlate with dust. These include child gender, child birth order, whether the child is a twin or other multiple birth, the mother's age, and the mother's education (as well as dummies for less-than-primary school completion and less-than-university completion). In particular, we report estimates of (1) with these variables as outcomes in Appendix Table A8, though without controlling for individual or maternal characteristics. We find no evidence that dust exposure predicts pre-determined characteristics, making selective fertility an unlikely explanation of our results.

Finally, in Figure 6, we provide additional evidence that selective fertility does not explain our results. First, we plot the relative frequency of births in each month, separately for clusters defined by quintiles of mean dust. Second, we plot the average education of mothers for the births recorded in each month. This too is by quintile of mean dust exposure. In the first figure, it is clear that there is a seasonal pattern to fertility in the data, but it is not one that differs across regions heavily affected by dust pollution and those that are not. Indeed, the pattern in births across the year appears nearly identical for the highest and lowest mean dust clusters with median dust clusters showing the strongest seasonality, though largely similar to other quintiles. In the second case, though there are static differences in education of mothers across clusters, these are netted out by geographic fixed effects in all of our regression specifications. Importantly, there is no evidence of seasonal variations in the characteristics of mothers, let alone any differential seasonality that might correlate with dust.

# 6 Conclusion

This paper shows that PM2.5 dust in West Africa has an economically meaningful and statistically significant impact on infant mortality during the years 1986-2006. While many papers have studied the impacts of *in utero* pollution exposure on early life health, our paper makes additional contributions to this literature.

First, by examining these effects in the context of developing countries, we highlight

the greater vulnerability of people with fewer resources to adopt avoidance behavior against dust. Second, we examine parental responses, both in the prenatal and in the post natal stages to show evidence suggesting that dust pollution in this context can be mitigated in part by investments shaped by compensating parental behaviors or direct access to health centers and doctors. Third, we examine the effects of dust over time and by country-level measures of macroeconomic conditions and public health infrastructure.

We find a steady decline in the impacts of dust over time. In the absence of a reduction in dust itself, this suggests that people and countries are adapting over time in some way to attenuate the harmful effects of dust. The analysis of country-level indicators as moderators of the dust impact suggests that this attenuation in impacts may be due to economic development and improvements in public health infrastructure, with health-related improvements exhibiting a stronger mitigating role. We also find evidence at the individual level that implies some role for parental experience in behavioral adaptation. We find that children born to more experienced parents (that is, non-first-born children) are less susceptible to the deleterious effects of dust and are also more likely to receive compensatory investments at birth and in early life.

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Tab	Table 1. Summary Statistics									
	(1)	(2)	(3)	(4)	(5)					
_	Mean	s.d.	Min	Max	Ν					
Outcomes										
Died neonatal X 1000	54.6	227	0	1,000	615,187					
Died as child X 1000	172	377	0	1,000	615,187					
Died as infant X 1000	112	315	0	1,000	615,187					
Treatment										
Dust L0 through L8 (cumulative) in 100s of micrograms/metercube	4.11	1.52	0.55	17.3	597,267					
Environment Controls										
Mean Dust (in 100s of micrograms/metercube)	0.44	0.13	0.18	1.36	615,187					
SD Dust	0.30	0.073	0.16	0.94	615,187					
Child Controls										
Birth order	3.74	2.44	1	18	615,187					
Multiple	0.035	0.18	0	1	615,187					
Female	0.49	0.50	0	1	615,187					
Mother Controls										
Mother Years of Education	1.63	3.35	0	22	614,827					
Mother Age	25.9	6.60	9	50	615,187					

		Table 2. Impacts of Dust on Mortality									
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)		
	Neona	tal Mortality	X 1000	Infar	nt Mortality X	1000	Child	Child Mortality X 1000			
				1	n-utero shoc	k					
Dust L0 through L8	1.979	1.927	2.455	6.236***	6.449***	7.170***	12.648***	13.121***	13.675***		
	(1.553)	(1.575)	(1.594)	(2.107)	(2.160)	(2.198)	(2.527)	(2.596)	(2.674)		
Observations	581,847	581,487	581,487	581,847	581,487	581,487	581,847	581,487	581,487		
Mean of Dependent Variable		54.35			111.34			170.70			
	In-utero shock and exposure after birth										
Dust L0 through L8	1.584	1.104	1.992	2.543	2.012	3.319	5.999**	5.548*	7.254**		
	(2.006)	(1.953)	(1.958)	(2.546)	(2.508)	(2.545)	(2.930)	(2.883)	(2.912)		
Dust Year After Birth	-0.149	-0.154	-0.164	1.068	1.281	1.251	3.135	3.545	3.550		
	(1.759)	(1.707)	(1.711)	(2.295)	(2.242)	(2.257)	(2.807)	(2.760)	(2.773)		
Observations Mean of Dependent Variable	474,051	473,789 56.50	473,789	474,051	473,789 116.50	473,789	474,051	473,789 170.70	473,789		
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes		
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes		
Demographic controls	No	Yes	Yes	No	Yes	Yes	No	Yes	Yes		
Weather controls	No	No	Yes	No	No	Yes	No	No	Yes		

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by dust point in parentheses, unless otherwise indicated. All regressions are OLS. Demographic controls are birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated. Weather controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared.

Table 3. Heter	ogeneous Impact	s of Dust on Morta	ality					
	(1) (2) (3) (4)							
	Infant Morta	ality X 1000	Child Mortality X 1000					
	Birth order							
Dust X Not First Born	-12.812***	-12.888***	-12.287**	-12.445**				
	(4.325)	(4.326)	(5.001)	(5.004)				
Dust L0 through L8	8.231***	8.928***	14.267***	14.784***				
	(2.302)	(2.326)	(2.687)	(2.746)				
Observations	581,487	581,487	581,487	581,487				
Mean of Dependent Variable	111.34	111.33	170.70	170.70				
		Fen	nale					
Dust X Female	0.435	0.436	1.654**	1.659**				
	(0.600)	(0.600)	(0.748)	(0.749)				
Dust L0 through L8	6.232***	6.953***	12.298***	12.850***				
	(2.218)	(2.250)	(2.690)	(2.772)				
Observations	581,487	581,487	581,487	581,487				
Mean of Dependent Variable	111.34	111.33	170.70	170.70				
		Mothe	r's Age					
Dust X Mother's Age	-0.196***	-0.195***	-0.217***	-0.213***				
	(0.061)	(0.061)	(0.072)	(0.072)				
Dust L0 through L8	11.135***	11.821***	18.311***	18.765***				
	(2.619)	(2.623)	(3.110)	(3.142)				
Observations	581,487	581,487	581,487	581,487				
Mean of Dependent Variable	111.34	111.33	170.70	170.70				
		Mother's	s Ed Level					
Dust X Mother's Education Level	0.033	0.027	-0.110	-0.118				
	(0.124)	(0.124)	(0.152)	(0.152)				
Dust L0 through L8	6.428***	7.151***	13.190***	13.756***				
	(2.159)	(2.196)	(2.586)	(2.664)				
Observations	581,487	581,487	581,487	581,487				
Mean of Dependent Variable	111.34	111.33	170.70	170.70				
		Dust exposur	re of first child					
Dust X Dust experienced by 1st child	-0.160	-0.130	-0.498	-0.476				
	(0.453)	(0.457)	(0.589)	(0.593)				
Dust L0 through L8	8.983**	8.800**	18.957***	17.936***				
	(4.266)	(4.265)	(5.797)	(5.777)				
Observations	239,521	239,521	239,521	239,521				
Mean of Dependent Variable	111.34	111.33	170.70	170.70				
		We	alth					
Dust X Wealth	-0.752	-0.796	-3.088***	-3.160***				
	(0.702)	(0.695)	(0.886)	(0.876)				
Dust L0 through L8	8.699***	9.297***	13.997***	14.215***				
	(2.456)	(2.511)	(2.922)	(3.070)				
Observations	372,788	372,788	372,788	372,788				
Duct V Dural	0.070**	Rural	Area	0 700*				
Dust X Rurai	2.870***	2.997***	2.571"	2.723"				
Durat I O there use I O	(1.358)	(1.352)	(1.452)	(1.430)				
Dust Lo through L8	(2.527)	(2.553)	(2.938)	(2.993)				
	. ,	. ,	. ,	. ,				
Observations	581,487	581,487	581,487	581,487				
Mean of Dependent Variable	109.51	109.50	168.48	168.48				
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes				
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes				
Demographic controls	Yes	Yes	Yes	Yes				
Weather controls	No	Yes	No	Yes				

Table 4	. Impacts of Dust or	n Early-life Investm	nent Responses		
	(1)	(2)	(3)	(4)	(5)
		E	arly-life investmer	nts	
	No. of Polio	No. of DPT	Received	No. of Total	
	doses received	doses received	Measles	Vaccinations	_
Dust L0 through L8	0.070***	0.069***	0.021***	0.163***	-
	(0.016)	(0.016)	(0.008)	(0.040)	
Observations	112,808	112,110	110,525	109,486	
Mean of dependent variable	1.84	1.74	0.52	4.10	
		At-bin	th investment resp	oonses	
	Received BCG	Received Polio	Prenatal Doctor	Homo Dolivon	Doctor Attended
	Vaccination	0 dose	Visit	Home Delivery	Delivery
Dust L0 through L8	0.017***	0.016***	-0.001	0.000	0.000
	(0.003)	(0.004)	(0.001)	(0.002)	(0.001)
Observations	109,486	112,565	95,790	84,685	118,879
Mean of dependent variable	0.72	0.46	0.08	0.56	0.03
		Ear	y life health outco	mes	
	Weight / Height	Weight / Age	Height / Age		
	(percentile)	(percentile)	(percentile)	Birthweight	_
Dust L0 through L8	-0.934***	-2.100***	-1.630***	-0.010	
	(0.326)	(0.403)	(0.417)	(0.010)	
Observations	84,023	78,138	78,138	23,880	
Mean of dependent variable	34.17	21.26	23.87	3.15	
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes
Weather controls	Yes	Yes	Yes	Yes	Yes
Child recode controls	Yes	Yes	Yes	Yes	Yes

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by dust point in parentheses, unless otherwise indicated. All regressions are OLS. Weather controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared. Child recode controls are birthorder, dummy for whether birth was multiple, child's age in months, and child's sex.

Table 5. Heterogeneous Impacts of Dust on Early-life Investment Responses by Birth Order									
	(1)	(2)	(3)	(4)	(5)				
		Ea	rly-life investmen	ts					
	No. of Polio	No. of DPT	Measles	No. of Total					
	doses received	doses received	Vaccination	Vaccinations					
Dust X Not First Born	0.002	0.001	0.005	0.005					
	(0.009)	(0.009)	(0.003)	(0.019)					
Dust L0 through L8	0.069***	0.069***	0.017*	0.159***					
	(0.019)	(0.019)	(0.009)	(0.046)					
Observations	112,808	112,110	110,525	109,486					
Mean of dependent variable	1.84	1.74	0.52	4.10					

	At-birth investment responses						
	Received BCG	Received Polio	Prenatal Doctor	Homo Dolivon	Doctor Attended		
	Vaccination	0 dose	Visit	Home Delivery	Delivery		
Dust X Not First Born	-0.002	-0.004	0.000	-0.004	0.001		
	(0.003)	(0.003)	(0.002)	(0.003)	(0.001)		
Dust L0 through L8	0.019***	0.019***	-0.001	0.003	-0.000		
	(0.004)	(0.004)	(0.002)	(0.003)	(0.001)		
Observations	112,565	95,790	84,685	118,879	118,437		
Mean of dependent variable	0.72	0.46	0.08	0.56	0.03		
	Eary life health outcomes						
	Weight / Height	Weight / Age	Height / Age	Distance is bet			
			(percentile)	Birtirweigrit	-		
Dust X Not First Born	-0.189	-0.227	-0.068	0.002			
	(0.261)	(0.216)	(0.228)	(0.009)			
Dust L0 through L8	-0.779**	-1.913***	-1.575***	-0.012			
	(0.371)	(0.437)	(0.464)	(0.013)			

	()	( )	()	
84,023	78,138	78,138	23,880	
34.17	21.26	23.87	3.15	
Yes	Yes	Yes	Yes	Yes
Yes	Yes	Yes	Yes	Yes
Yes	Yes	Yes	Yes	Yes
Yes	Yes	Yes	Yes	Yes
	84,023 34.17 Yes Yes Yes Yes Yes	84,02378,13834.1721.26YesYesYesYesYesYesYesYesYesYesYesYesYesYes	84,02378,13878,13834.1721.2623.87YesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYes	84,02378,13878,13823,88034.1721.2623.873.15YesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYesYes

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by dust point in parentheses, unless otherwise indicated. All regressions are OLS. Weather controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared. Child recode controls are birthorder, dummy for whether birth was multiple, child's age in months, and child's sex.

Table 6. Change	Table 6. Changes Over Time in Impacts of Dust on Mortality									
	(1)	(2)	(2)	(3)	(4)	(4)				
	Infan	t Mortality X	Child	Child Mortality X 1000						
Dust X Time (Years)	-0.885***	-0.941***	-0.922***	-1.769***	-1.866***	-1.825***				
	(0.142)	(0.142)	(0.146)	(0.219)	(0.224)	(0.227)				
Dust L0 through L8	10.354***	10.836***	11.398***	20.880***	21.823***	22.041***				
	(2.266)	(2.381)	(2.403)	(2.885)	(3.014)	(3.084)				
Observations	581,847	581,487	581,487	581,847	581,487	581,487				
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes				
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes				
Demographic controls	No	Yes	Yes	No	Yes	Yes				
Weather controls	No	No	Yes	No	No	Yes				
Mean of Dependent Variable		111.34			170.70					

Table 7. Mean Effects of World Bank National Variables as Moderators									
	(1)	(2)	(3)	(4)	(5)	(6)			
	Infa	nt Mortality X 1	000	Chi	Child Mortality X 1000				
Dust X WB Economic Factors Mean Effects	2.786**	-2.676**		1.562	-7.873***				
	(1.402)	(1.120)		(1.905)	(1.576)				
Dust X WB Health Factors Mean Effects	-8.773***		-7.527***	-16.019***		-15.320***			
	(1.359)		(1.137)	(2.168)		(1.888)			
Dust L0 through L8	-0.363	5.706***	0.085	-0.559	5.798***	-0.153			
	(2.427)	(2.118)	(2.401)	(2.413)	(2.160)	(2.384)			
Observations	537,301	563,268	537,301	537,301	563,268	537,301			
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes			
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes			
Demographic controls	Yes	Yes	Yes	Yes	Yes	Yes			
Mean of Dependent Variable	109.71	110.82	109.71	167.84	171.01	167.84			

Table 8: Estimates at the region level								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		In(Infant I	mortality)			In(Child	mortality)	
Dust 9 months prior	0.0384	0.0497	0.0718	0.154**	0.0549**	0.0825*	0.109**	0.195***
Duct 0 months prior V Voor of Birth	(0.0259)	(0.0508)	(0.0505)	(0.0593)	(0.0256)	(0.0475)	(0.0509)	(0.0590)
Dust 9 months prior × rear or Birth		(0.00423)		(0.00289)		-0.00289 (0.00378)		(0.00305)
			Country	X Year X			Coutry X Y	ear X Month
Additional controls	None	None	Mon	th FE	None	None	F	E
Observations	19,129	19,129	19,105	19,105	21,163	21,163	21,149	21,149
R-squared	0.452	0.452	0.690	0.691	0.444	0.444	0.689	0.691

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by region in parentheses, unless otherwise indicated. All regressions are OLS. All regressions include month of birth fixed effects, country fixed effects, region fixed effects, year of birth fixed effects, average temperature and rain the past 9 months and their squares and interactions. The dependent variable is calculated as In(total deaths/number of births) in a given region-month-year cell.

	Table 9. Alternative Fixed Effects									
	(1)	(2)	(3)	(4)	(5)	(6)				
			Infant Mortali	ty X 1000						
Dust L0 through L8	2.581*** (0.847)	2.411*** (0.850)	2.581*** (0.847)	3.244* (1.811)	2.963*** (1.038)	3.083*** (0.957)				
Observations	596,876	596,876	596,876	596,876	557,654	581,487				
Mean of Dependent Variable			111.7	71						
	Child Mortality X 1000									
Dust L0 through L8	4.142*** (1.132)	3.551*** (1.157)	4.142*** (1.132)	8.009*** (2.305)	3.742*** (1.329)	4.291*** (1.228)				
Observations	596,876	596,876	596,876	596,876	557,654	581,487				
Mean of Dependent Variable			171.	7						
Fixed effects	Country X Month of birth, Year of birth, DHS Cluster, Country linear time trends Yeas	Country X Month of birth, Year of birth, DHS Cluster, Country quadratic time trends Yes	Country X Month of birth, Year of birth, DHS Survey, Dust point ID, Country linear time trends Yes	Country X Year of birth X Month of birth, DHS Survey, Dust Point ID Yee	Mother, Year of birth, Country X Month of birth, Country trends Yea	Country X Month of Birth, Birth Year, DHS Cluster, Country X Year of birth time trends, DHS Cluster X Month of Birth Yes				
				. 50	. 50					

Appendix. Not for publication.



Figure A1: Share of each country in births in the data, by year

Each figure shows the percentage of total births that occur in a given country in a given birth year.

Table A1. Impacts of Dust on Mortality (robust to use of PM 10)									
	(1)	(2)	(3)	(4)	(5)	(6)			
	Neonatal Mo	rtality X 1000	Infant Mort	ality X 1000	Child Mort	ality X 1000			
Dust L0 through L8	0.224	0.355	1.266**	1.444***	2.850***	2.998***			
	(0.388)	(0.394)	(0.518)	(0.526)	(0.643)	(0.669)			
Observations	578,851	578,851	578,851	578,851	578,851	578,851			
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes			
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes			
Demographic controls	Yes	Yes	Yes	Yes	Yes	Yes			
Weather controls	No	Yes	No	Yes	No	Yes			
Mean of Dependent Variable	54	.56	111	1.71	17	1.7			

Table A2. Impacts of Alternative Dust M	leasures on Mortality	
	(1) Infant Mortality X	(2) Child Mortality X
	1000	1000
Specification 1		
Number of past 9 months with above average dust + 1SD	4.385***	6.932***
	(1.080)	(1.391)
Specification 2		
Max of dust in the past 9 months	8.564**	11.563**
	(4.188)	(4.921)
Specification 3		
Log of dust in the past 9 months	38.807***	66.934***
	(10.180)	(14.747)
Specification 4		
Dust L0 through L8 (controlling for dust SD L0-L8)	8.643**	15.208***
	(3.329)	(4.390)
SD of dust L0-L8	-13.441	-13.988
	(20.489)	(27.435)
Specification 5		
Dust L0 through L8 (controlling for max dust L0-L8)	8.564***	19.159***
	(3.066)	(3.964)
Observations asd	581,487	581,487
Country X Month of Birth X Year of Birth FE	Yes	Yes
DHS Cluster X Month of Birth FE	Yes	Yes
Demographic controls	Yes	Yes
Weather controls	Yes	Yes
Mean of Dependent Variable	111.71	171.7

Table A3. Impacts of Dust on Mortality - Inverse weighting and bilinear interpolation									
	(1)	(2)	(3)	(4)					
	Infant Mor	tality X 1000	Child Mor	tality X 1000					
Dust L0 through L8	4.993	7.322***	12.937***	14.777***					
	(3.624)	(2.395)	(4.049)	(2.916)					
	Inverse	Bi-linear	Inverse	Bi-linear					
Weights	distance	interpolation	distance	interpolation					
Observations	581,487	581,487	581,487	581,487					
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes					
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes					
Demographic controls	Yes	Yes	Yes	Yes					
Weather controls	Yes	Yes	Yes	Yes					
Mean of Dependent Variable	11	1.71	171.7						

Table A4. Nonlinear effects of dust							
	(1)	(2)					
	Infant Mortality X 1000	Child Mortality X 1000					
Dust L0-L8 decile 2	4.275	8.850***					
	(2.963)	(3.085)					
Dust L0-L8 decile 3	5.228	13.290***					
	(3.634)	(4.115)					
Dust L0-L8 decile 4	10.715**	19.441***					
	(4.237)	(4.898)					
Dust L0-L8 decile 5	10.956**	18.921***					
	(4.972)	(6.069)					
Dust L0-L8 decile 6	12.938**	22.188***					
	(5.140)	(6.330)					
Dust L0-L8 decile 7	18.157***	29.841***					
	(5.932)	(7.326)					
Dust L0-L8 decile 8	19.463***	33.186***					
	(6.561)	(8.076)					
Dust L0-L8 decile 9	18.899***	32.579***					
	(7.203)	(8.948)					
Dust L0-L8 decile 10	22.538***	39.769***					
	(8.441)	(10.881)					
Observations	578,851	578,851					
Country X Month of Birth X Year of Birth FE	Yes	Yes					
DHS Cluster X Month of Birth FE	Yes	Yes					
Demographic controls	Yes	Yes					
Weather controls	Yes	Yes					

Table A5. Sensitivity to Outliers of Impacts of Dust on Mortality							
	(1)	(2)	(3)				
	Neonatal Mortality X 1000	Infant Mortality X 1000	Child Mortality X 1000				
	Dro	p Highest Dust Decile Clust	ers				
Dust L0 through L8	1.110	4.394*	7.534**				
	(2.129)	(2.648)	(3.251)				
Observations	517,730	517,730	517,730				
	Dro	p Lowest Dust Decile Clust	ers				
Dust L0 through L8	2.287	7.028***	13.672***				
	(1.633)	(2.284)	(2.879)				
Observations	518,798	518,798	518,798				
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes				
DHS Cluster X Month of Birth FE	Yes	Yes	Yes				
Demographic controls	Yes	Yes	Yes				
Weather controls	Yes	Yes	Yes				
Mean of Dependent Variable	54.56	111.71	171.7				

Table A6. Impacts of Dust on Mortality by Trimesters								
	(1)	(2)	(3)	(4)	(5)	(6)		
	Neonatal I	Mortality X						
	10	00	Infant Morta	ality X 1000	Child Morta	ality X 1000		
First trimester dust	3.708	3.805	9.091**	9.388**	13.265***	13.453***		
	(2.691)	(2.645)	(4.040)	(4.001)	(4.970)	(4.923)		
Second trimester dust	-0.526	0.152	3.702	4.798	10.072**	10.971**		
	(2.664)	(2.731)	(3.868)	(3.892)	(4.242)	(4.400)		
Third trimester dust	3.038	3.610	6.382*	7.648**	14.707***	16.575***		
	(2.821)	(2.780)	(3.305)	(3.405)	(4.087)	(4.265)		
Observations	581,847	581,487	581,847	581,487	581,847	581,487		
Country X Month of Birth X Year of Birth	Yes	Yes	Yes	Yes	Yes	Yes		
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes		
Controls	No	Yes	No	Yes	No	Yes		
Mean of Dependent Variable	54	.56	111	111.71		171.7		

Table A7. Impacts of Dust on Mortality - clustered at regional level								
	(1)	(2)	(3)	(4)				
_	Infant Morta	ality X 1000	Child Morta	ality X 1000				
Dust L0 through L8	6.236**	7.170***	12.648***	13.675***				
	(2.394)	(2.711)	(4.004)	(4.516)				
Observations	581,847	581,487	581,847	581,487				
Country X Month of Birth X Year of Birth	Yes	Yes	Yes	Yes				
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes				
Controls	No	Yes	No	Yes				
Mean of Dependent Variable	111	1.71	171.7					

Table	A0. Dusi exp			n crinuren a			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Female	Birth Order	Multiple birth	Mother's age	Mother any education	Mother education<= 6	Mother education <=12
Dust L0 through L8	-0.001 (0.001)	-0.010 (0.007)	0.001 (0.001)	0.012 (0.017)	0.001 (0.001)	-0.003*** (0.001)	0.000 (0.000)
Observations	597,267	597,267	597,267	597,267	596,915	596,915	596,915
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rain and Temperature Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes

#### Table A8. Dust exposure and characteristics of children and mothers

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by dust point in parentheses, unless otherwise indicated. All regressions are OLS. Controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared, unless otherwise indicated.

Table A9. Heterogeneous impacts of Dust on Eany-life investment Responses by Gender								
	(1)	(2)	(3)	(4)	(5)			
		Early-life investments						
	No. of Polio	No. of DPT	Measles	No. of Total				
	doses received	doses received	Vaccination	Vaccinations				
Dust X Female	0.013**	0.012**	0.002	0.026**				
	(0.005)	(0.005)	(0.002)	(0.011)				
Dust L0 through L8	0.064***	0.064***	0.020**	0.150***				
	(0.017)	(0.016)	(0.008)	(0.040)				
Observations	112,808	112,110	110,525	109,486				
Mean of dependent variable	1.84	1.74	0.52	4.10				

	At-birth investment responses							
	Received BCG	Received Polio	Prenatal Doctor	Homo Dolivory	Doctor Attended			
	Vaccination	0 dose	Visit	Home Delivery	Delivery			
Dust X Female	0.002	0.003	-0.001	-0.001	0.000			
	(0.002)	(0.002)	(0.001)	(0.002)	(0.001)			
Dust L0 through L8	0.016***	0.014***	-0.000	0.000	0.000			
	(0.003)	(0.004)	(0.002)	(0.002)	(0.001)			
Observations	112,565	95,790	84,685	118,879	118,437			
Mean of dependent variable	0.72	0.46	0.08	0.56	0.03			

Eary life health outcomes

	Weight / Height (percentile)	Weight / Age (percentile)	Height / Age (percentile)	Birthweight	
Dust X Female	-0.211	-0.312**	-0.313*	0.005	
	(0.183)	(0.152)	(0.161)	(0.007)	
Dust L0 through L8	-0.832**	-1.949***	-1.478***	-0.013	
	(0.346)	(0.408)	(0.412)	(0.011)	
Observations	84,023	78,138	78,138	23,880	
Mean of dependent variable	34.17	21.26	23.87	3.15	
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes
Weather controls	Yes	Yes	Yes	Yes	Yes
Child Recode Controls	Yes	Yes	Yes	Yes	Yes

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by dust point in parentheses, unless otherwise indicated. All regressions are OLS. Weather controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared.

#### Table A9. Heterogeneous Impacts of Dust on Early-life Investment Responses by Gender

			Table /	A10: Remov	ina one cour	ntrv at a time	from sample	e					
Infant mortality	(1)	(2) Burkina	(3)	(4) Cote d'	(5)	(6)	(7)	(8)	(9)	(10)	(11) Sierra	(12)	(13)
Excluded Country	None	Faso	Benin	Ivoire	Ghana	Guinea	Liberia	Mali	Nigeria	Niger	Leone	Senegal	Togo
Dust L0 through L8	6.449*** (2.160)	7.145*** (2.222)	6.204*** (2.221)	6.444*** (2.195)	7.104*** (2.247)	6.062*** (2.205)	6.164*** (2.163)	6.841*** (2.274)	1.391 (2.804)	6.888*** (2.237)	6.454*** (2.163)	7.182*** (2.242)	7.054*** (2.201)
Observations	581,487	488,553	557,027	547,809	551,910	544,764	563,268	480,309	480,768	555,520	564,887	497,177	564,365
Child mortality	(1)	(2) Burkina	(3)	(4) Cote d'	(5)	(6)	(7)	(8)	(9)	(10)	(11) Sierra	(12)	(13)
Excluded Country	None	Faso	Benin	Ivoire	Ghana	Guinea	Liberia	Mali	Nigeria	Niger	Leone	Senegal	Togo
Dust L0 through L8	13.121*** (2.596)	13.745*** (2.638)	12.833*** (2.668)	13.159*** (2.643)	13.948*** (2.682)	12.621*** (2.644)	12.813*** (2.598)	13.013*** (2.760)	5.348 (3.363)	13.286*** (2.709)	13.051*** (2.599)	15.946*** (2.632)	13.962*** (2.616)
Observations	581,487	488,553	557,027	547,809	551,910	544,764	563,268	480,309	480,768	555,520	564,887	497,177	564,365

Controls: see Column 3 and footnotes of Table 2. Each regression follows exactly the same specifiction.

Table A11. Interactions with Country level variables								
	(1)	(2)	(3)	(4)				
	Infant Mort	ality X 1000	Child Morta	ality X 1000				
Dust X GNI	-15.404***	-17.989***	-46.552***	-50.115***				
	(5.369)	(5.444)	(6.693)	(6.811)				
Dust X Poverty Gap	3.583**	3.538**	8.923***	9.018***				
	(1.442)	(1.420)	(2.066)	(2.073)				
Dust X Tractors per Land Area	-0.359	-2.738	88.417*	81.876				
	(36.268)	(35.594)	(50.606)	(51.256)				
Dust X % GDP Agriculture	-2.133	-1.474	-2.697	-2.025				
	(1.331)	(1.333)	(1.700)	(1.707)				
Dust L0 through L8	8.024	7.417	7.909	7.767				
	(5.375)	(5.488)	(7.219)	(7.272)				
Observations	563,584	563,268	563,584	563,268				
	Infant Mort	ality X 1000	Child Morta	ality X 1000				
Dust X Health Expenditure per capita	-0.779	-1.233	0.332	-0.171				
	(1.814)	(1.779)	(2.673)	(2.684)				
Dust X Maternal Mortality	9.159	10.475*	30.406***	32.582***				
	(5.916)	(5.816)	(8.290)	(8.322)				
Dust X % Out of Pocket Health Expenditure	-0.448	-0.773	-5.648**	-6.028**				
	(1.889)	(1.870)	(2.721)	(2.747)				
Dust X Safe Water (Rural)	-5.218	-4.673	-7.150	-6.585				
	(3.517)	(3.503)	(4.595)	(4.637)				
Dust X Immunization Rate (DPT)	0.254	-0.037	0.621	0.390				
	(1.487)	(1.446)	(1.847)	(1.839)				
Dust X Anemia Rate (Children under 5)	19.147	20.650*	65.965***	68.791***				
	(11.747)	(11.469)	(17.018)	(16.831)				
Dust L0 through L8	-86.153	-92.501	-283.674***	-296.951***				
	(65.042)	(63.676)	(90.238)	(89.189)				
Observations	537,617	537,301	537,617	537,301				
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes				
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes				
Demographic controls	No	Yes	No	Yes				

Table A12. Mean Effects of World Bank National Variables as Moderators of Adaptation								
	(1)	(2)	(3)	(4)	(5)	(6)		
-	Infant Mortality X 1000			Child Mortality X 1000				
Dust X WB Economic Factors Mean Effects X Time	-0.162	-0.035		0.452	0.185			
	(0.208)	(0.160)		(0.296)	(0.300)			
Dust X WB Health Factors Mean Effects X Time	0.234		0.170	0.157		0.389*		
	(0.160)		(0.131)	(0.232)		(0.229)		
Dust X WB Economic Factors Mean Effects	5.042*	1.982		-2.645	-1.885			
	(2.835)	(2.127)		(3.957)	(3.659)			
Dust X WB Health Factors Mean Effects	-7.817***		-6.028**	-11.943***		-13.483***		
	(2.685)		(2.384)	(3.970)		(3.852)		
Dust L0 through L8	5.101	10.885***	5.065	8.573**	21.342***	8.617**		
	(3.246)	(2.447)	(3.136)	(4.152)	(3.044)	(4.282)		
Dust X Time (Years)	-0.567***	-0.965***	-0.525***	-0.998***	-1.822***	-0.955***		
	(0.202)	(0.157)	(0.193)	(0.262)	(0.237)	(0.278)		
Observations	537,301	563,268	537,301	537,301	563,268	537,301		
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes		
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes		
Demographic controls	Yes	Yes	Yes	Yes	Yes	Yes		
Mean of Dependent Variable	109.71	110.82	109.71	167.84	171.01	167.84		

Table A13. Impacts of Dust on Mortality for Sample of Never Movers						
	(1)	(2)	(3)	(4)		
	Infant Mortality X 1000		Child Morta	lity X 1000		
Dust L0 through L8	5.922*	2.228	13.258***	7.235		
	(3.281)	(3.781)	(4.390)	(4.818)		
Dust Year After Birth		-0.251		0.931		
		(3.955)		(4.631)		
Observations	184,692	152,758	184,692	152,758		
Mean of Dependent Variable	125.93		195.78			
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes		
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes		
Demographic controls	Yes	Yes	Yes	Yes		
Weather controls	Yes	Yes	Yes	Yes		

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by dust point in parentheses, unless otherwise indicated. All regressions are OLS. Demographic controls are birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated. Weather controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared.

Table A14. Impacts of Dust on Mortality - Exploring Sibship size								
	(1)	(2)	(3)	(4)	(5)	(6)		
	Infant Mortality X 1000			Child Mortality X 1000				
Dust L0 through L8	2.548	5.509**	4.205*	9.863	11.534***	6.451**		
	(5.808)	(2.199)	(2.510)	(6.205)	(2.601)	(2.855)		
Dust L0 through L8 X Sibshipsize			0.192			0.695***		
			(0.159)			(0.212)		
Observations Mean of Dependent Variable	87,193	581,487	581,487	87,193	581,487	581,487		
Sample restriction	First births only	Sibship size FE	None	First births only	Sibship size FE	None		
Country X Month of Birth X Year of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes		
DHS Cluster X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes		
Demographic controls	Yes	Yes	Yes	Yes	Yes	Yes		
Weather controls	Yes	Yes	Yes	Yes	Yes	Yes		

#### Table A14. Impacts of Dust on Mortality - Exploring Sibship size

Notes: \*\*\*Significant at 1%, \*\*Significant at 5%, \*Significant at 10%. Standard errors clustered by dust point in parentheses, unless otherwise indicated. All regressions are OLS. Demographic controls are birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated. Weather controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared.