Dust and Death: Evidence from the West African Harmattan*

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Abstract

Using two decades of data from 12 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, indicating 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

Research over the past decade has emphasized the adverse effects of weather fluctuations and other environmental factors on important outcomes related to economic growth and development, including conflict, mortality, disease, and income (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), studies in Bangladesh show that individuals persist in not migrating, even in chronically flood-prone areas (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.[1] 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, then, crucial in the low-income country context.

This paper presents one of the first large scale empirical analyses 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 adaptation responses. Our data covers 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

[1] This has been shown recently in the United States with regards to the adoption of air-conditioning as a mitigating mechanism against temperature shocks (Barreca et al., 2016).

(Besancenot et al., 1997).[2] Recent *harmattan* activity in Nigeria has sparked enquiry into whether global warming could be partly responsible for changes in the timing and intensity of the dust storms (Gambrell, 2010).

The *harmattan* is known to cause widespread damage to infrastructure, disrupt flights, reduce rainfall, and affect crop outputs (e.g., Adefolalu (1984); Adetunji et al. (1979)). It has been associated with outbreaks of meningitis (Besancenot et al., 1997). In this paper, we are not able to distinguish the relative importance of these mechanisms; rather, our main goal is to show the reduced form relationship between the *harmattan* and infant and child mortality across multiple countries and over time. 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 over the past decade emphasizing this critical period of child development (Cunha and Heckman, 2007). Examining the impacts of environmental shocks such as dust on post-birth health indicators is complicated by the fact that parental investments might react to early childhood health shocks (Adhvaryu and Nyshadham, 2016; Bharadwaj et al., 2013b). Indeed, we examine whether early life parental investments in child health such as vaccinations and health facility utilization respond to dust exposure and find that, even in the short run, there appears to be a significant behavioral response that is most likely due to observed health impairments rather than greater access to these resources. Hence, we interpret our main results as the effect of dust exposure net of any compensating parental investments.[3]

We find compelling evidence that exposure to dust *in utero* affects early life survival. Additional exposure of 10 $\mu g/m^3$ of PM2.5 during each month of gestation decreases infant survival by 2.3 percentage points, which is substantial relative to the sample average

[2]Abdurrahman and Taqi (1982) give the composition of Saharan dust as silica (50%), alumina (10%), lime (5%), ferric oxide (4%) and other salts (5%).

[3]See Almond and Mazumder (2013) for a more detailed discussion of reinforcing and compensating parental investments.

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 mitigated 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 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. These results imply some role for individual parental experience in adaptation as well.

Our empirical approach controls for standard concerns faced by studies that examine the role of pollution exposure on mortality. 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, 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.

To control for these factors, our main empirical specification includes survey cluster fixed effects, where clusters approximate villages and are defined uniquely for every wave of the survey. We include country by month fixed effects that allow for different seasonal patterns by country. Our baseline also includes year of birth fixed effects and country-specific linear time trends. That is, 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 year of birth, the typical country-specific monthly pattern of mortality, and the long-run trends in that child's country. 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. In addition, our results are robust to the inclusion of more rigorous country × year of birth × month 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.

1.1 Contribution

This paper is related to the broader literature examining the impacts of environmental factors on health outcomes. Given that health and mortality shape the economic impacts of the *harmattan*, the results of this paper can be related to work examining the health impacts of exposure to weather related changes in income in developing countries as in Maccini and Yang (2009), Adhvaryu et al. (2014), Baird et al. (2011), Burgess et al. (2014), and others. More directly, fine dust inhalation can directly affect health of the fetus and result in lower health at birth, increasing the risk of infant and early childhood mortality. In particular, given that we examine these impacts across countries in Western Africa, our paper relates to some of the emerging literature examining the health impacts of pollution exposure in developing countries (Arceo-Gomez et al., 2015; Ebenstein, 2012; Greenstone and Hanna, 2014).[4] Our paper also contributes to this literature by examining these impacts across a broad set of countries and over two decades. Baseline vulnerability may differ over time and across levels of economic development and health infrastructure, as may the costs of avoidance and recovery behaviors.

Although we are not the first paper to investigate the link between natural air pollution and mortality in a poor country (e.g. Jayachandran (2009)), the existing literature remains small. Our data allow us to expand the evidence on the external validity of existing find-

[4]There exists a similar literature on health consequences of pollution in rich countries. See, for example, Currie and Schwandt (2015).

ings. Similarly, our focus on a naturally occurring source of pollution allows our results to be uncontaminated by other policies that might coincide with environmental regulations and similar sources of identifying variation used in many standard differences-in-differences frameworks. In this sense, our contribution resembles that of Jia and Ku (2015), in a developing country context and over a broader spatial and temporal scope. However, because we focus narrowly on mortality, parental investments, and health outcomes before age five, ours is not a complete accounting of the negative effects of fetal dust exposure, and further research along these dimensions is needed.

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 are adapting to the adverse effects of dust exposure. In this regard, our paper builds on the evidence presented in Barreca et al. (2016) in the context of several developing nations and over a critical period of their growth. This is a literature in which there is a clear need for more empirical research. Even in the case of the health effects of environmental shocks for which there is a large literature (temperature) and in contexts where avoidance behaviors can be reliably measured (rich countries), existing knowledge on adaptation is quite limited (Deschenes, 2014). Many pioneering studies that evaluate the effects of pollution on mortality in developing countries are able to compare the effects to those in rich countries and to identify a socio-economic gradient, 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., 2015). As Arceo-Gomez et al. (2015) 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). As a result, we may not expect diminishing effects of shocks over time like those found in the developed world.[5] We do, however, find evidence that the effects

[5]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.

of fetal dust exposure on the survival of West African children have weakened dramatically in recent years. We also find that more parental experience leads to stronger compensatory responses to dust exposure in terms of early-life investments and smaller dust impacts for non-first-born children. Finally, we show that economic development, and health-related improvements, such as higher immunization rates and per capita health expenditure and lower diseases prevalence, are associated with smaller impacts of dust on infant and child mortality.

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 out-

comes 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.[6] 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.[7] 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.[8] 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

[6] 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/.

[7] 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/.

[8] http://allafrica.com/stories/201311180452.html

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). 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:

$$Mortality_{icvym} = \beta Dust_{cvym} + x'_{icvym}\gamma + \delta_{cm} + \eta_y + \theta_{cv} + \phi_c \times y + \epsilon_{icvym}. \quad (1)$$

Here, $Mortality_{icvtm}$ is an indicator for child i, born in month m in year t, 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.[9] We use a cumulative exposure measure, summing the dust reading for each month during gestation. β 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

[9]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.

terms, the interaction between average rainfall and temperature, child birth order, child 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 also include three additional and important sets of fixed effects, First, country $\times$ calendar month of birth fixed effects (δ_{cm}) account for general seasonal variation in birth outcomes that may vary across countries. Second, year-of-birth fixed effects (η_y) account for time-specific shocks such as economic crises and disease outbreaks. Finally, θ_{cv} captures cluster fixed effects that will absorb any unobserved drivers of mortality that happen to correlate with dust over space. We also include country time trends $\phi_c \times y$ to account for other possible unobserved trending variables that may vary by country-specific birth cohort.

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-specific patterns of seasonality, country-specific time trends, annual shocks that affect the whole of West Africa, or very flexible functions of rainfall and temperature. We cluster standard errors by dust point; as a robustness exercise we will 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, as well as a gender dummy and 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 impacts 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 β 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 early-life 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.[10] These data are available for the period from August 1985 to December 2006 at latitude-longitude intervals of 1.25° by 1.25°. 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

[10]The data can be downloaded from `http://iridl.ldeo.columbia.edu/home/.nasa_roses_a19/.Dust_model/.dust_mon_avg/.dust_pm25_sconc10_mon/`

of $PM_{2.5}$, or particulate matter smaller than 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). 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). 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, 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.

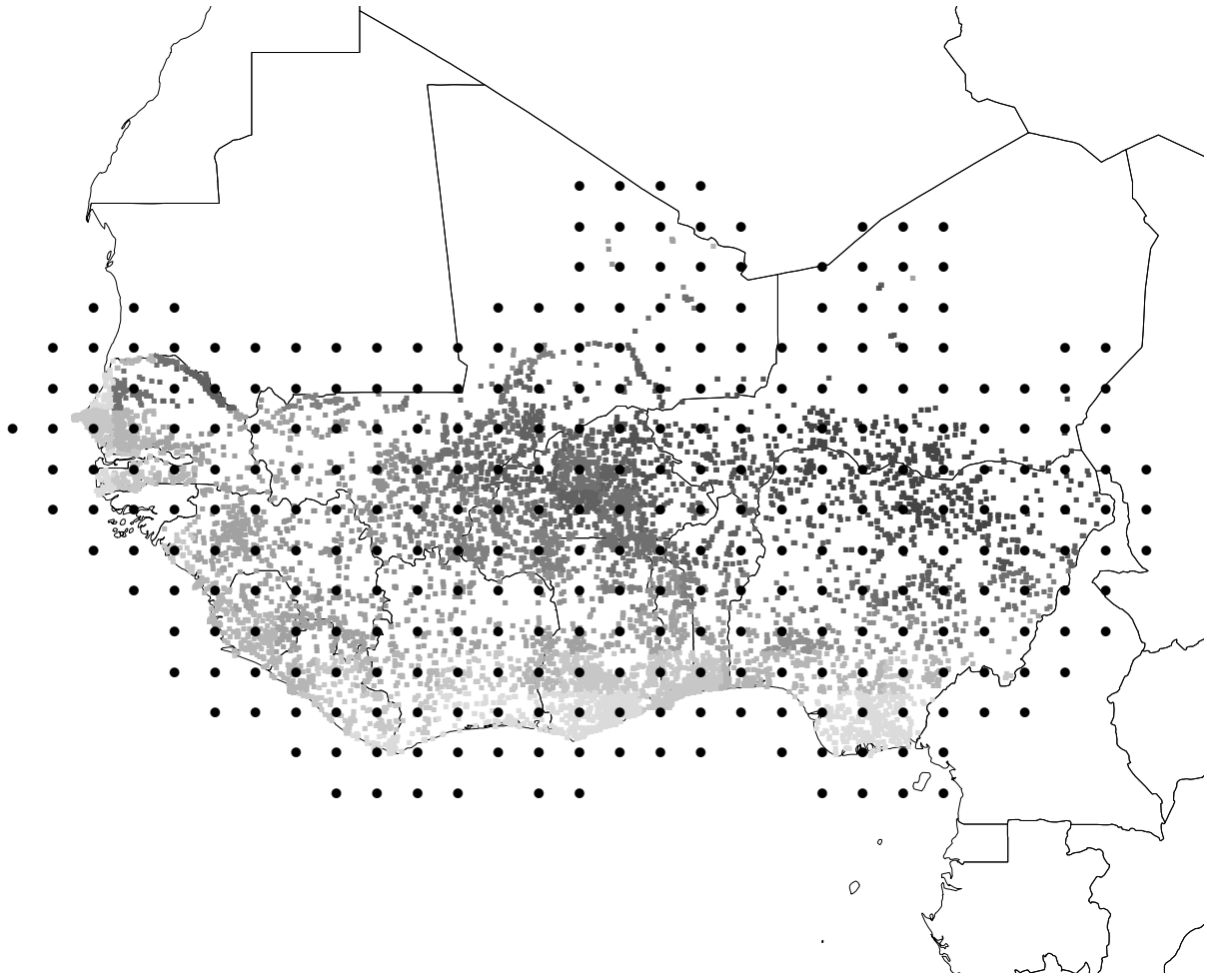
4.3 Matching DHS to dust data

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.

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

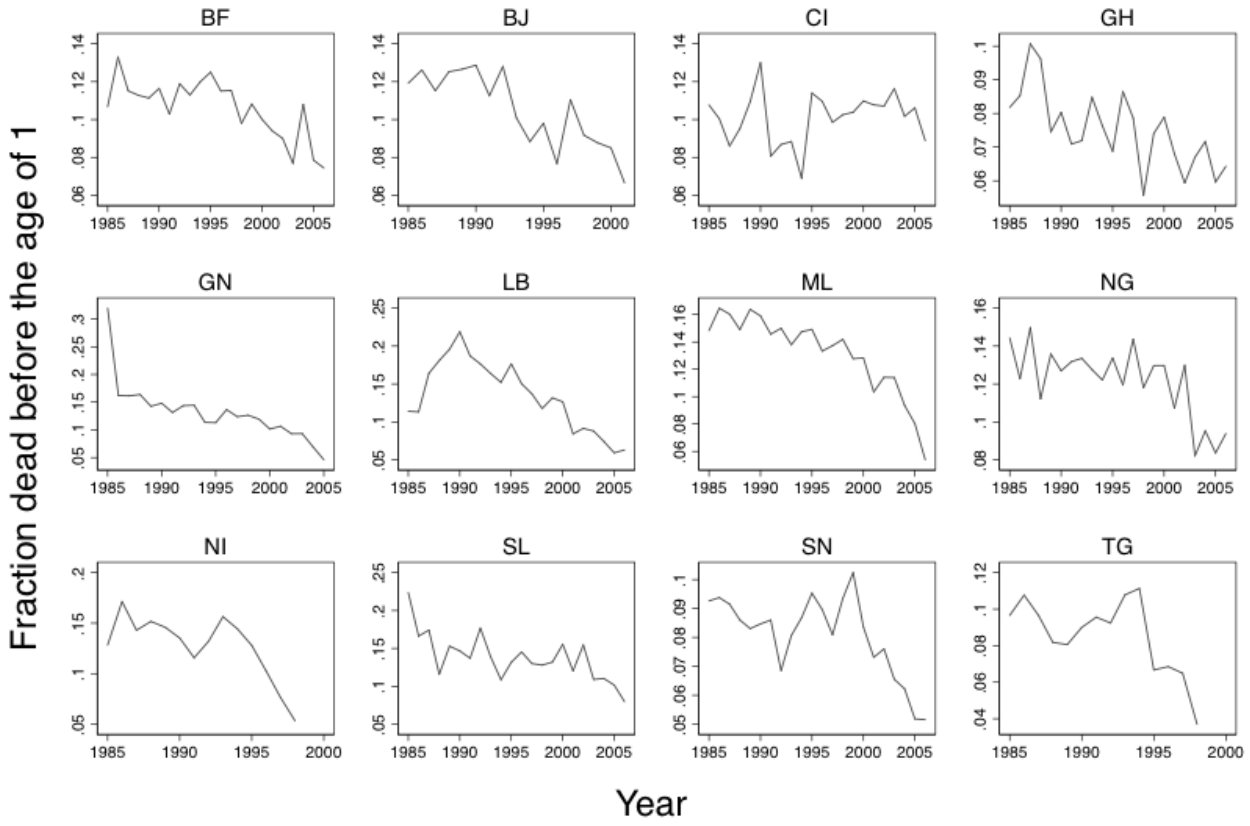
Figure 1: DHS clusters and dust points



The square dots are DHS survey clusters. Shades of grey represent deciles of mean dust, ranging from dark (most dust) to light (least dust). The round dots are dust points.

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 (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).

Figure 2: Infant Mortality by Country

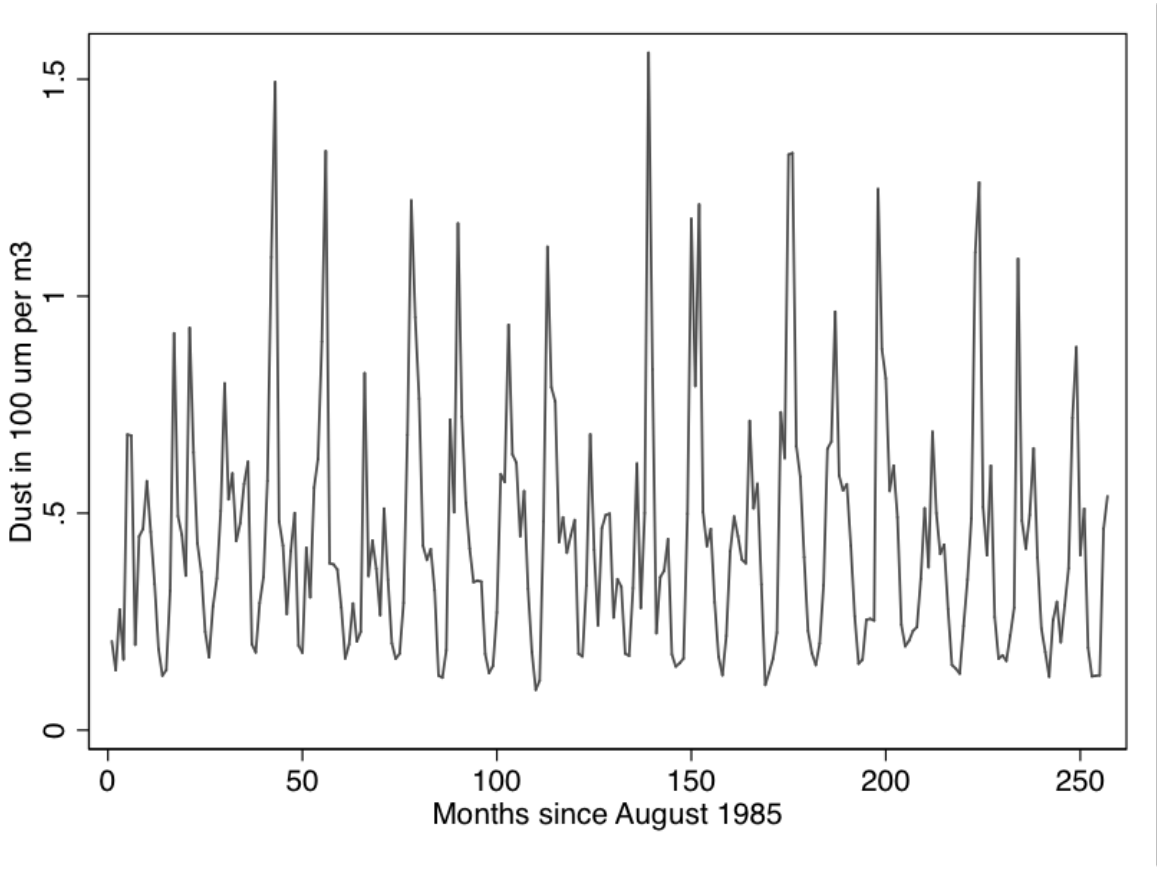


Graph shows infant death by year of birth and country. Infant mortality calculated from raw DHS data without adjustment. Country abbreviations are as follows: BF = Burkina Faso, BJ = Benin, CI = Ivory Coast, GH = Ghana, GN = Guniea, LB = Liberia, ML = Mali, NG = Nigeria, NI = Niger, SL = Sierra Leone,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

Figure 3: PM2.5



Graph shows PM2.5 concentrations averaged for the sample at the year and month level.

The average monthly PM2.5 concentration is around $44\mu g/m^3$ over the entire sample.[11] 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

[11] 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's 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 have 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).[12]

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 0.39 percentage point increase in infant mortality, or roughly 3.5% of the mean.[13] The following thought experiment helps to put the magnitude of the estimate impacts in perspective. Consider re-

[12]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 Georeferenced 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.

[13]To see this, multiply the standard deviation of cumulative dust exposure from Table 1 (1.52) by the coefficient (2.581) and divide by 10 to convert from deaths per 1,000 to percentage points.

ducing exposure to levels deemed acceptable by the EPA's PM2.5 national standards in the US ($15\mu g/m^3$). This would reduce mean exposure from 411 $\mu g/m^3$ over an entire pregnancy to 135 $\mu g/m^3$, reducing infant mortality by 0.71 percentage points, or 6.4% of the mean.[14]

We can put these results in the context of other interventions or environmental shocks considered in the literature as well. Our results are smaller than some other shocks considered 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).

By contrast, 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. (2015) 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 2.581 per 1000. In Appendix Table A2, we replace PM2.5 measures with PM10. We find a corresponding effect of 0.626 that compares directly to their estimate of 0.23.[15] 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 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

[14]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 2.581 and divide by 10 to convert from deaths per 1,000 to percentage points.

[15]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.

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). 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.[16]

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 prescriptions that may mitigate the effects of dust.

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 infant mortality are less than half as large for later born children and impacts on child mortality are roughly two-thirds of that for first born children.

In the middle panel, 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 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;

[16]We have tested whether in utero dust exacerbates the effects of dust after birth. The interaction is positive but insignificant (not reported).

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. (2014) for an example). However, we find no evidence of this; we show below in Appendix Table A9 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 A10) we find no evidence of compensatory investment responses being weaker for girls.

Finally, in the bottom panel, we 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.[17] 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.

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 the 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

[17]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.

of services such as health centers, this could explain part of the effect. A 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., 2013a).[18]

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 *pre natal* and at-brith 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 up to 6 weeks after birth. Since the *pre natal* and at-brith 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 *post natal* 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.

[18] 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).

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 heterogenous 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 *pre natal* 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

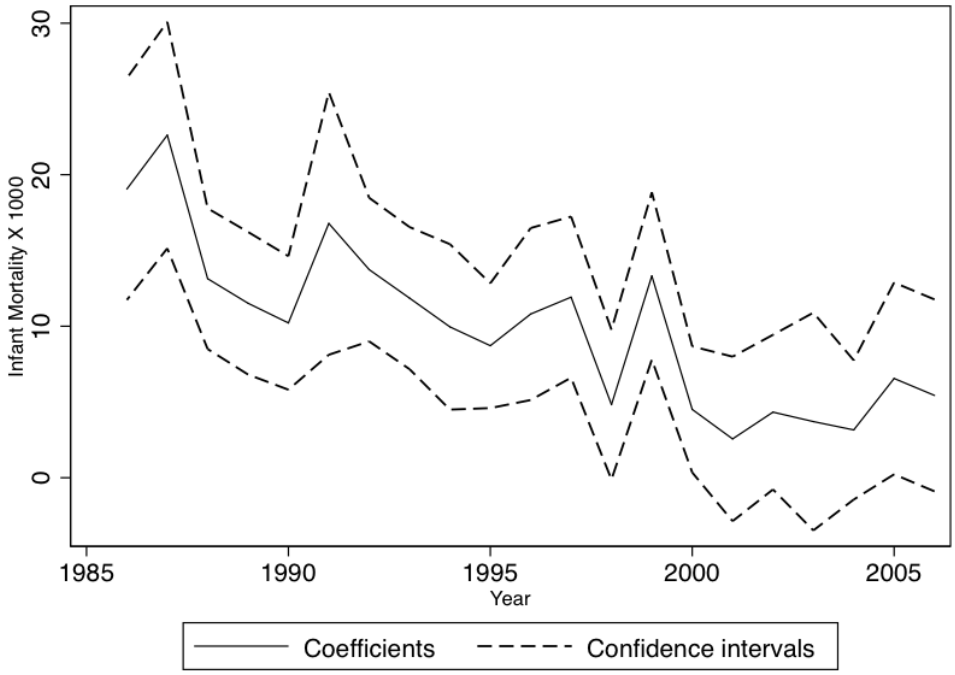
Having established the basic result that dust exposure is harmful to children and that direct biological impacts are only partially offset by parental responses, we next exploit the fact that our data spans 20 years between 1986-2006 to see how the impact of dust on mortality has changed over time.

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

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

Hence, each point on Figure 4 is a regression coefficient of the effect of *in utero* exposure on infant mortality for a given year, or β_y. For each estimate, the sample includes only children born in that year. ϕ_{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.

Figure 4: Effects of dust over time



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

Other variables and parameters are as in our baseline.

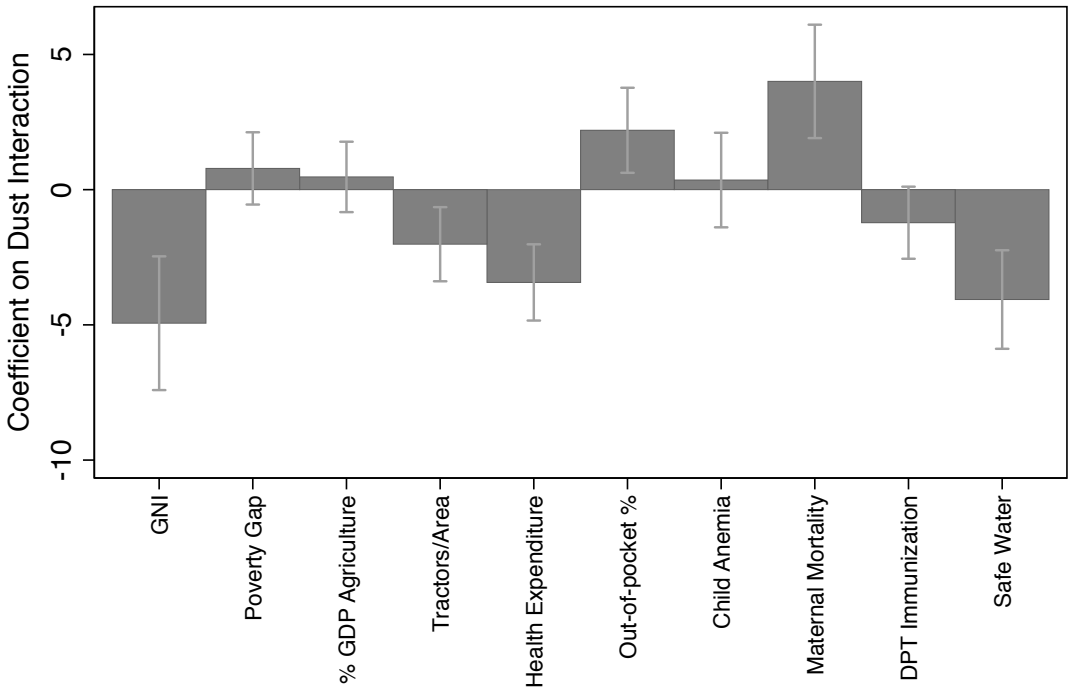
Figure 4 shows a clear pattern of a decline in the impact of dust on mortality. The impact of dust appears to decrease by more than half over this time period. Table 6 confirms these effects by showing an interaction of dust exposure with a linear time trend - the interaction is negative, statistically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

A more direct test of this is in Figure 5, in which we interact dust with various country-level 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 (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). Figure 5 shows that countries with higher economic development (GNI), more mechanized agriculture (tractors), higher health expenditure, greater public health resources (DPT immunization), and greater rural access to safe water exhibit smaller impacts of dust on mortality. On the other hand, countries with higher out of pocket share of health expenditure, higher maternal mortality rates, and to a lesser extent larger poverty gap, greater agricultural intensity, and higher child anemia rates, exhibit larger impacts of dust on morality. 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 SD changes.

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 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, health factors continue to significantly mitigate dust impacts, but economic factors no longer seem to matter. We interpret these results as suggestive evidence of possible policy prescriptions. Improvements in health infrastructure might help to alleviate impacts of dust exposure, more so even than general economic development. In fact, as discussed above, its possible that improvements in health infrastructure contributed to the attenuation in dust impacts on

Figure 5: Individual macroeconomic moderators



Coefficients on the interaction between dust and each macroeconomic moderator variable 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.

mortality over time in our sample. We unfortunately cannot test for this specifically, given that we have limited data on changes in these macroeconomic conditions over time.

We also demonstrate robustness of this analysis 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 equalling 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 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, 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 A1, 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. Note that the preferred specification used to obtain the main results in the paper includes fixed effects for country × month of birth, year of birth, DHS cluster, and country time trends, along with weather and child and mother demographic controls. These main results, first reported in columns 6 and 9 of Table 2, are copied in column 1 of Table A1.

In column 2, we add to the preferred specification DHS survey fixed effects (specific to country and year of survey) and replace DHS cluster fixed effects with the more rigorous dust point ID fixed effects. Column 3 adds rigorous country × year of birth × month of birth FE along with dust point ID FE and DHS survey FE. Finally, in column 4, we restrict comparisons to siblings by including fixed effects for mothers, alongside fixed effects for country × year of birth and country × month of birth. Overall, the results are of the same sign and magnitude as our main results and remain, in general, 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 A2. Appendix Table A2 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).[19]

In Appendix Table A3 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)). The number of months during which dust exposure was greater than the cluster mean correlates positively though insignificantly with infant and child mortality. 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

[19]The strong correlation between PM2.5 and PM10 dust prevents us from disentangling their relative effects in the same empirical specification.

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.

Similarly, in Appendix Table A4, 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.

We use Table A5 to 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 A6 shows 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 utero*, or with the possibility that *in utero* dust exposure makes children more vulnerable to later dust exposure in childhood.

In Appendix Table A7, 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 A8, we show that our results are robust to clustering by administrative

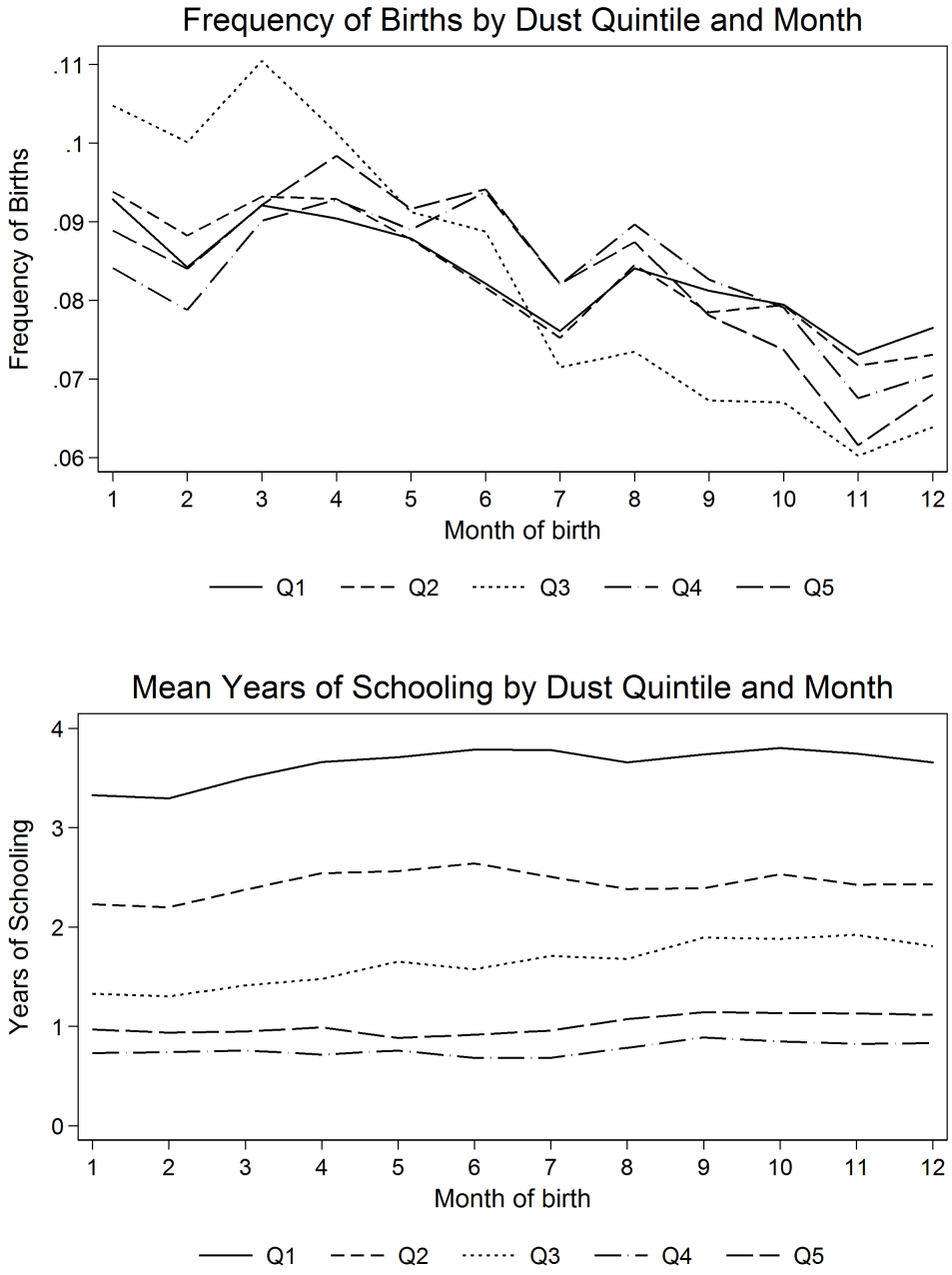
region as recorded in the DHS, rather than the nearest point in the dust data. These regions are typically second-level administrative units such as provinces and states. Indeed, standard errors are largely indistinguishable using this approach. Finally, in additional results (not reported, but available upon request), we restrict our sample to children whose mothers report having lived in their current place of residence their whole lives. Though the effect on infant mortality becomes marginally insignificant ($t = 1.62$), it is nearly identical to the baseline estimate. The effect on child mortality remains significant and is again very close to the baseline estimate. 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 evidence that the effect of dust exposure for those incapable of migration as an avoidance behavior is not different from that of the whole sample.

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 A1 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), demonstrating 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. In particular, we report estimates of (1) with these variables as outcomes in Appendix Table A9, though without controlling for individual or maternal characteristics. We find no evidence that dust exposure predicts predetermined characteristics, making selective fertility an unlikely explanation of our results.

Figure 6: Tests for selective fertility



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.

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 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 suggest that this attenuation in impacts might be due to economic development and improvements in public health infrastructure, with health-related improvements exhibiting a stronger mitigative role.

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Table 1. Summary Statistics

	(1) Mean	(2) s.d.	(3) Min	(4) Max	(5) N
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)
	Neonatal Mortality X 1000			*Infant Mortality X 1000*			*Child Mortality X 1000*		
					In-utero shock				
Dust L0 through L8	0.151	0.321	0.209	2.061**	2.674***	2.581***	3.141***	4.177***	4.142***
	(0.599)	(0.651)	(0.650)	(0.846)	(0.866)	(0.854)	(1.137)	(1.130)	(1.142)
Observations	597,267	597,267	596,915	597,267	597,267	596,915	597,267	597,267	596,915
Mean of Dependent Variable		54.35			111.34			170.70	
					In-utero shock and exposure after birth				
Dust L0 through L8	-0.104	0.310	0.142	1.512	2.363**	2.181**	2.519**	3.769***	3.710***
	(0.754)	(0.822)	(0.813)	(0.998)	(1.049)	(1.020)	(1.162)	(1.107)	(1.123)
Dust Year After Birth	-0.249	-0.099	-0.100	0.205	0.549	0.565			
	(0.654)	(0.664)	(0.647)	(0.920)	(0.921)	(0.880)			
Dust 5 Years After Birth							2.971***	3.028***	3.224***
							(0.446)	(0.453)	(0.470)
Observations	492,640	492,640	492,380	492,640	492,640	492,380	597,267	597,267	596,915
Mean of Dependent Variable		56.50			116.50			170.70	
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Rain and Temperature Controls	No	Yes	Yes	No	Yes	Yes	No	Yes	Yes
Additional 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. Weather controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared. Additional 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.*

Table 3. Heterogeneous Impacts of Dust on Mortality

	(1)	(2)	(3)	(4)	(5)	(6)
	Infant Mortality X 1000			*Child Mortality X 1000*		
			Heterogeneity by Birth Order			
Dust X Not First Born	-3.129***	-3.108***	-2.494***	-2.196*	-2.159*	-1.498
	(0.916)	(0.918)	(0.870)	(1.118)	(1.121)	(1.079)
Dust L0 through L8	4.594***	5.181***	4.613***	4.920***	5.920***	5.368***
	(1.086)	(1.074)	(1.034)	(1.504)	(1.428)	(1.403)
Observations	597,267	597,267	596,915	597,267	597,267	596,915
Mean of Dependent Variable	111.34	111.34	111.33	170.70	170.70	170.70
			Heterogeneity by Gender			
Dust X Female	0.390	0.393	0.492	1.460**	1.467**	1.573**
	(0.580)	(0.580)	(0.567)	(0.703)	(0.703)	(0.685)
Dust L0 through L8	1.855**	2.471***	2.338**	2.406**	3.444***	3.365***
	(0.922)	(0.934)	(0.925)	(1.199)	(1.188)	(1.201)
Observations	597,267	597,267	596,915	597,267	597,267	596,915
Mean of Dependent Variable	111.34	111.34	111.33	170.70	170.70	170.70
			Heterogeneity by Wealth			
Dust X Wealth	0.751	0.676	0.373	-1.267*	-1.449**	-1.824***
	(0.488)	(0.492)	(0.481)	(0.648)	(0.659)	(0.648)
Dust L0 through L8	2.495**	2.958***	2.931***	2.469*	3.527**	3.585**
	(1.063)	(1.056)	(1.047)	(1.453)	(1.435)	(1.432)
Observations	379,500	379,500	379,202	379,500	379,500	379,202
Mean of Dependent Variable	109.51	109.51	109.50	168.48	168.48	168.48
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes	Yes
Weather Controls	No	Yes	Yes	No	Yes	Yes
Additional Controls	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. Weather ontrols are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared. Additional 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.*

Table 4. Impacts of Dust on Early-life Investment Responses

	(1)	(2)	(3)	(4)	(5)
	Early-life investments				
	No. of Polio doses received	*No. of DPT doses received*	*Received Measles*	*No. of Total Vaccinations*	
Dust L0 through L8	0.058***	0.049***	0.022***	0.131***	
	(0.015)	(0.015)	(0.006)	(0.035)	
Observations	143,892	143,191	141,748	140,715	
Mean of dependent variable	1.84	1.74	0.52	4.10	
	At-birth investment responses				
	Received BCG Vaccination	*Received Polio 0 dose*	*Prenatal Doctor Visit*	*Home Delivery*	*Doctor Attended Delivery*
Dust L0 through L8	0.007**	0.005	-0.001	0.002	-0.000
	(0.003)	(0.004)	(0.002)	(0.002)	(0.001)
Observations	143,673	122,422	116,753	151,044	150,636
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 L0 through L8	-1.012***	-1.865***	-1.925***	-0.010	
	(0.311)	(0.367)	(0.327)	(0.008)	
Observations	114,949	109,157	109,157	46,141	
Mean of dependent variable	34.17	21.26	23.87	3.15	
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes
Weather 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 5. Heterogeneous Impacts of Dust on Early-life Investment Responses by Birth Order

	(1)	(2)	(3)	(4)	(5)
	Early-life investments				
	No. of Polio doses received	*No. of DPT doses received*	*Measles Vaccination*	*No. of Total Vaccinations*	
Dust X Not First Born	0.015**	0.009	0.005**	0.028*	
	(0.007)	(0.007)	(0.003)	(0.016)	
Dust L0 through L8	0.046***	0.042**	0.018***	0.108***	
	(0.017)	(0.017)	(0.006)	(0.040)	
Observations	143,892	143,191	141,748	140,715	
Mean of dependent variable	1.84	1.74	0.52	4.10	
	At-birth investment responses				
	Received BCG Vaccination	*Received Polio 0 dose*	*Prenatal Doctor Visit*	*Home Delivery*	*Doctor Attended Delivery*
Dust X Not First Born	0.004*	0.001	0.000	-0.004*	0.002*
	(0.002)	(0.002)	(0.001)	(0.003)	(0.001)
Dust L0 through L8	0.004	0.004	-0.001	0.006*	-0.002
	(0.004)	(0.004)	(0.002)	(0.003)	(0.001)
Observations	143,673	122,422	116,753	151,044	150,636
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 Not First Born	0.014	-0.133	-0.235	0.011	
	(0.156)	(0.157)	(0.173)	(0.007)	
Dust L0 through L8	-1.022***	-1.750***	-1.725***	-0.018*	
	(0.320)	(0.391)	(0.368)	(0.009)	
Observations	114,949	109,157	109,157	46,141	
Mean of dependent variable	34.17	21.26	23.87	3.15	
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	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 6. Changes Over Time in Impacts of Dust on Mortality

	(1)	(2)	(2)	(3)	(4)	(4)
	Infant Mortality X 1000			*Child Mortality X 1000*		
Dust X Time (Years)	-0.547***	-0.543***	-0.579***	-1.132***	-1.124***	-1.186***
	(0.086)	(0.086)	(0.089)	(0.144)	(0.144)	(0.150)
Dust L0 through L8	7.262***	7.863***	8.110***	13.919***	14.915***	15.467***
	(1.204)	(1.235)	(1.256)	(1.782)	(1.915)	(1.990)
Observations	597,267	597,267	596,915	597,267	597,267	596,915
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes	Yes
Weather Controls	No	Yes	Yes	No	Yes	Yes
Additional Controls	No	No	Yes	No	No	Yes
Mean of Dependent Variable		111.34			170.70	

*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 ontrols are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared. Additional 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.*

Table 7. Mean Effects of World Bank National Variables as Moderators

	(1)	(2)	(3)	(4)	(5)	(6)
	Infant Mortality X 1000			*Child Mortality X 1000*		
Dust X WB Economic Factors Mean Effects	0.638	-1.316**		0.775	-1.975**	
	(0.710)	(0.579)		(0.945)	(0.787)	
Dust X WB Health Factors Mean Effects	-3.948***		-3.558***	-6.856***		-6.379***
	(0.726)		(0.612)	(1.251)		(1.063)
WB Economic Factors Mean Effects	-0.656	6.311**		-0.711	8.728**	
	(3.422)	(3.095)		(4.459)	(4.145)	
WB Health Factors Mean Effects	13.872***		13.538***	25.066***		24.692***
	(3.301)		(3.133)	(5.633)		(5.450)
Dust L0 through L8	1.265	2.615***	1.246	1.360	4.208***	1.339
	(0.971)	(0.877)	(0.967)	(1.283)	(1.201)	(1.277)
Observations	551,606	578,351	551,606	551,606	578,351	551,606
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes	Yes
Weather Controls	Yes	Yes	Yes	Yes	Yes	Yes
Additional Controls	Yes	Yes	Yes	Yes	Yes	Yes
Mean of Dependent Variable	109.71	110.82	109.71	167.84	171.01	167.84

*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 the un-interacted main effect, rainfall, temperature, squares of each, rainfall and temperature interacted, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table 8: Estimates at the region level

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	ln(Infant mortality)				ln(Child mortality)			
Dust 9 months prior	0.0384	0.0497	0.0274	0.0909***	0.0549**	0.0825*	0.0360	0.105***
	(0.0259)	(0.0508)	(0.0227)	(0.0314)	(0.0256)	(0.0475)	(0.0224)	(0.0308)
Dust 9 months prior X Year of Birth		-0.00117		-0.00651***		-0.00289		-0.00712***
		(0.00423)		(0.00203)		(0.00378)		(0.00202)
Additional controls	None	None	Country X Year trends		None	None	Country X Year trends	
Observations	19,129	19,129	19,129	19,129	21,163	21,163	21,163	21,163
R-squared	0.452	0.452	0.514	0.515	0.444	0.444	0.504	0.505

*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 ln(total deaths/number of births) in a given region-month-year cell.*

Table A1. Alternative Fixed Effects

	(1)	(2)	(3)	(4)
	Infant Mortality X 1000			
Dust L0 through L8	2.581***	2.395***	2.424	2.892**
	(0.854)	(0.874)	(1.771)	(1.231)
Observations	596,915	578,684	578,684	596,915
Mean of Dependent Variable	111.71			
	Child Mortality X 1000			
Dust L0 through L8	4.142***	3.898***	7.139***	3.736**
	(1.142)	(1.268)	(2.325)	(1.589)
Observations	596,915	578,684	578,684	596,915
Mean of Dependent Variable	171.7			
Fixed effects	Country X Month of birth, Year of birth, DHS Cluster, Country time trends	Country X Month of birth, Year of birth, DHS Survey, Dust point ID, Country time trends	Country X Year of birth X Month of birth, DHS Survey, Dust Point ID	Mother, Country X Year of birth, Country X Month of birth
Weather and Additional 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. Controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table A2. Impacts of Dust on Mortality (robust to use of PM 10)

	(1)	(2)	(3)	(4)	(5)	(6)
	Neonatal Mortality X 1000		*Infant Mortality X 1000*		*Child Mortality X 1000*	
Dust L0 through L8	-0.051	-0.023	0.455**	0.626***	0.826***	1.150***
	(0.149)	(0.163)	(0.217)	(0.213)	(0.314)	(0.306)
Observations	594,735	594,385	594,735	594,385	594,735	594,385
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes	Yes
Controls	No	Yes	No	Yes	No	Yes
Mean of Dependent Variable	54.56		111.71		171.7	

*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, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table A3. Impacts of Alternative Dust Measures on Mortality

	(1) *Infant Mortality X 1000*	(2) *Child Mortality X 1000*
Specification 1		
Number of past 9 months with above average dust	0.450	0.712
	(0.474)	(0.600)
Specification 2		
Number of past 9 months with above average dust + 1SD	1.679**	2.950***
	(0.659)	(0.869)
Specification 3		
Max of dust in the past 9 months	4.188**	5.656**
	(1.810)	(2.302)
Specification 4		
Log of dust in the past 9 months	9.454***	13.174***
	(3.316)	(4.568)
Specification 5		
Dust L0 through L8 (controlling for dust SD L0-L8)	2.305*	3.513*
	(1.390)	(1.938)
SD of dust L0-L8	2.652	6.048
	(9.453)	(13.023)
Specification 6		
Dust L0 through L8 (controlling for max dust L0-L8)	2.515*	4.684**
	(1.306)	(1.811)
Observations	596,915	596,915
Country X Month of Birth FE	Yes	Yes
Birth Year FE	Yes	Yes
DHS Cluster FE	Yes	Yes
Country X Year of birth time trends	Yes	Yes
Controls	Yes	Yes
Mean of Dependent Variable	111.71	171.7

*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, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table A4. Impacts of Dust on Mortality - Inverse weighting and bilinear interpolation

	(1)	(2)	(3)	(4)
	Infant Mortality X 1000		*Child Mortality X 1000*	
Dust L0 through L8	2.769***	2.671***	3.783**	4.349***
	(0.995)	(0.855)	(1.612)	(1.151)
Weights	Inverse distance	Bi-linear interpolation	Inverse distance	Bi-linear interpolation
Observations	596,915	596,914	596,915	596,914
Country X Month of Birth FE	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes
Mean of Dependent Variable	111.71		171.7	

*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, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table A5. Nonlinear effects of dust

	(1)	(2)
	Infant Mortality X 1000	*Child Mortality X 1000*
Dust L0-L8 decile 2	2.205	4.392*
	(2.061)	(2.411)
Dust L0-L8 decile 3	0.739	2.798
	(2.101)	(2.695)
Dust L0-L8 decile 4	6.309***	7.571***
	(2.300)	(2.902)
Dust L0-L8 decile 5	2.261	1.671
	(2.738)	(3.571)
Dust L0-L8 decile 6	2.181	2.423
	(3.012)	(3.662)
Dust L0-L8 decile 7	6.842**	6.611
	(3.156)	(4.033)
Dust L0-L8 decile 8	7.703**	8.666**
	(3.373)	(4.116)
Dust L0-L8 decile 9	6.076	5.656
	(3.715)	(4.688)
Dust L0-L8 decile 10	10.602**	13.375**
	(4.424)	(5.606)
Observations	596,915	596,915
Country X Month of Birth FE	Yes	Yes
Birth Year FE	Yes	Yes
DHS Cluster FE	Yes	Yes
Country X Year of birth time trends	Yes	Yes
Controls	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. Controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table A6. Sensitivity to Outliers of Impacts of Dust on Mortality

	(1)	(2)	(3)
	Neonatal Mortality X 1000	*Infant Mortality X 1000*	*Child Mortality X 1000*
	Drop Highest Dust Decile Clusters		
Dust L0 through L8	-0.227	1.880*	2.035*
	(0.770)	(0.977)	(1.201)
Observations	537,274	537,274	537,274
	Drop Lowest Dust Decile Clusters		
Dust L0 through L8	0.117	2.355**	3.721***
	(0.709)	(0.945)	(1.232)
Observations	537,189	537,189	537,189
Country X Month of Birth FE	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes
Controls	Yes	Yes	Yes
Mean of Dependent Variable	54.56	111.71	171.7

*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, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table A7. Impacts of Dust on Mortality by Trimesters

	(1)	(2)	(3)	(4)	(5)	(6)
	Neonatal Mortality X 1000		*Infant Mortality X 1000*		*Child Mortality X 1000*	
First trimester dust	0.845	0.747	3.363***	3.544***	4.451***	5.026***
	(0.783)	(0.790)	(1.241)	(1.224)	(1.582)	(1.584)
Second trimester dust	-0.825	-0.649	0.991	1.683	2.618*	3.749***
	(0.792)	(0.838)	(1.143)	(1.143)	(1.466)	(1.414)
Third trimester dust	0.582	0.620	2.055*	2.624**	2.542*	3.712***
	(0.911)	(0.952)	(1.090)	(1.103)	(1.362)	(1.358)
Observations	597,267	596,915	597,267	596,915	597,267	596,915
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes	Yes
Controls	No	Yes	No	Yes	No	Yes
Mean of Dependent Variable	54.56		111.71		171.7	

*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, birth order, female, multiple, mother's age, mother's age squared, mother's years of education, and mother's religion, unless otherwise indicated.*

Table A8. Impacts of Dust on Mortality - clustered at regional level

	(1)	(2)	(3)	(4)
	Infant Mortality X 1000		*Child Mortality X 1000*	
Dust L0 through L8	2.061**	2.581***	3.141***	4.142***
	(0.895)	(0.952)	(1.187)	(1.409)
Observations	597,267	596,915	597,267	596,915
Country X Month of Birth FE	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes
Weather and Additional Controls	No	Yes	No	Yes
Mean of Dependent Variable	111.71		171.7	

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

Table A9. Dust exposure and characteristics of children and mothers

	(1) Female	(2) Birth Order	(3) Multiple birth	(4) Mother's age	(5) Mother any education
Dust L0 through L8	-0.001	-0.010	0.001	0.012	0.001
	(0.001)	(0.007)	(0.001)	(0.017)	(0.001)
Observations	597,267	597,267	597,267	597,267	596,915
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes
Rain and Temperature 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. Controls are rainfall, temperature, rainfall and temperature interacted, rainfall and temperature squared, unless otherwise indicated.*

Table A10. Heterogeneous Impacts of Dust on Early-life Investment Responses by Gender

	(1)	(2)	(3)	(4)	(5)
	Early-life investments				
	No. of Polio doses received	*No. of DPT doses received*	*Measles Vaccination*	*No. of Total Vaccinations*	
Dust X Female	0.005	0.005	0.000	0.011	
	(0.004)	(0.004)	(0.001)	(0.009)	
Dust L0 through L8	0.055***	0.047***	0.022***	0.126***	
	(0.016)	(0.016)	(0.006)	(0.037)	
Observations	143,892	143,191	141,748	140,715	
Mean of dependent variable	1.84	1.74	0.52	4.10	
	At-birth investment responses				
	Received BCG Vaccination	*Received Polio 0 dose*	*Prenatal Doctor Visit*	*Home Delivery*	*Doctor Attended Delivery*
Dust X Female	0.000	0.002	-0.000	-0.000	0.001
	(0.001)	(0.002)	(0.001)	(0.001)	(0.001)
Dust L0 through L8	0.007*	0.005	-0.001	0.002	-0.001
	(0.003)	(0.004)	(0.002)	(0.002)	(0.001)
Observations	143,673	122,422	116,753	151,044	150,636
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	-0.080	-0.135	-0.133	0.004	
	(0.120)	(0.119)	(0.118)	(0.005)	
Dust L0 through L8	-0.968***	-1.786***	-1.839***	-0.013*	
	(0.317)	(0.371)	(0.334)	(0.008)	
Observations	114,949	109,157	109,157	46,141	
Mean of dependent variable	34.17	21.26	23.87	3.15	
Country X Month of Birth FE	Yes	Yes	Yes	Yes	Yes
Birth Year FE	Yes	Yes	Yes	Yes	Yes
DHS Cluster FE	Yes	Yes	Yes	Yes	Yes
Country X Year of birth time trends	Yes	Yes	Yes	Yes	Yes
Weather 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.*