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Effect of PM_{2.5} Exposure at the Conception and at the Birth on Child Mortality: Evidence from Asian Countries

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Currently, the most air-polluted areas in the world are in Asia and Africa. Although there are reports that air pollution affects human health, these data are obtained from developed countries and data from developing countries are limited. Extrapolating the data obtained from developed countries to estimate the effect of air pollution on health in developing countries remains a challenge owing to the differences in the other environment such as sanitation and access to health services. A central and policy relevant question is whether the timing to exposure matter and how much it matters. In this study, we examined the effect of the exposure to particulate matter 2.5 (PM_{2.5}), a fine inhalable particle with a diameter of 2.5 micrometers or smaller at conception year, on infant mortality using the multiple-waves of the demographic and health survey data and PM_{2.5} data in Asian countries. Combining multiple waves from Demographic and Health Surveys (DHS) through global positioning system (GPS) information of each cluster, we constructed a cluster-level panel data of child mortality. We mapped satellite-based PM_{2.5} data to the constructed panel data through the GPS information of both cluster data and PM_{2.5} data. We found that a 10 microgram increase in PM_{2.5} in the conception year is associated with a 16% increase in infant mortality, and its effect is statistically significant. Meanwhile, the PM_{2.5} at birth year does not affect the infant mortality. Our estimation results are robust with various specification checks.

Keywords: PM_{2.5} | Air Pollution | Infant Mortality | Asian Countries

According to the World Health Organization(1), 1.7 million children die owing to poor air quality. Central Africa and South Asia are among the areas with the most polluted air quality(2, 3). However, the relationship between air quality and health outcomes in developing countries has less frequently been examined. Most studies that have documented the relationship between air quality and health outcomes were conducted from developed countries—a few studies have measured a similar impact in developing countries (2). Interpreting and applying the knowledge obtained from developed countries to developing countries is often difficult owing to the different attributes between these societies, including mortality rates and other health conditions.

The central question on the effect of air pollution to health, which is important for policy makers, is how much and which timing the exposure to air pollution affects the health outcome. The answer to this question is the key input to design a policy to mitigate the impact of air pollution on the health outcomes. Several studies in epidemiology and public health literature shows that significance of the negative shock in utero on the health condition in later life the first few month of pregnancy while other studies does not find such a pattern. Examining the effect of the quantity and the timing of the exposure to

air pollution on health outcome is needed to design a better preventive policy.

To examine such an effect, a simple cross-sectional or time series analysis can be misleading due to confounding factors. One way to solve such a problem is to construct village-level panel data consisting of a large number of countries. The village-level panel data in multiple-countries allow the researchers to control the fixed effect of the village, and time. The availability of data in multiple countries makes it possible for the researcher to manage the country-year fixed effect, allowing control of different countries with different trajectories on the health outcome while estimating the effect of air pollution(2).

In this study, we examined the effect of the exposure to particulate matter 2.5 (PM_{2.5}), a fine inhalable particle of 2.5 micrometers or smaller in diameter on at the conception and birth on infant mortality. We use the multiple-waves of the demographic and health survey data in Asian countries while utilizing the GPS information of each cluster, we constructed the cluster level panel data of child mortality and mapped the PM_{2.5} data to the constructed panel data.

This study contributes to the literature in three ways. First, we specifically distinguish the effect of PM_{2.5} exposure on child mortality by measuring the exposure in the birth and conception years. Given the uncertainty on the mechanism in which PM_{2.5} affects infant mortality, it is important to distinguish the exposure to PM_{2.5} at conception and at birth.

Significance Statement

This paper examines the effect of particulate matter 2.5, fine inhalable particle with diameters that are 2.5 micrometers and smaller (PM_{2.5}) air pollution on infant mortality using the multiple-waves of the demographic and health survey data and PM_{2.5} data in Asian countries. Combining multiple waves of DHS surveys through GPS information of each cluster, we construct the cluster-level panel data of child mortality. We map satellite-based PM_{2.5} data to the constructed panel data through the GPS information of both cluster data and PM_{2.5} data. We find that 10 µg increase of PM_{2.5} at the conception year is associated with 16 % increase of infant mortality, and its effect is statistically significant while PM_{2.5} at the birth year does not affect the infant mortality. Our estimation results are robust to all robustness specification checks.

There is no conflict of interest to declare

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Although PM2.5 in the birth and conception years are highly correlated, we find that the effect of PM2.5 exposure in the birth year is minimal and not statistically significant. Even when the exposure of PM2.5 at conception is removed from the list of the explanatory variable, the estimated coefficient of the exposure to PM2.5 at birth remains minimal and not statistically significant. An additional robustness check conducted on the infants exposed to PM2.5 at birth for only a few months did not change the results, indicating an important mechanism by which PM2.5 affects human health.

Second, we focused on Asian countries as the pollutant contents of PM2.5 in Asia and Africa are different. It is known that the main content of PM2.5 in Africa is dust, whereas that in Asia comes from industry and agriculture. It is quite interesting that the effects of PM2.5 in Africa and Asia can be different. To precisely estimate the effect of PM2.5, in this research, we focus on Asian countries.

Literature Review

The concentration and spatial distribution of PM2.5 is affected by geographic features, meteorology, and regional variations including temporal parameters (4, 5). Therefore, evaluation of PM2.5 variability needs to incorporate various models (6). (2) investigated the robust relationship between air quality and infant mortality in Africa. The study covers 30 countries, including 990,696 individual birth outcomes across sub-Saharan Africa with satellite-based estimates of the exposure to ambient respirable particulate matter. (7) examined the loss of life expectancy from air pollution compared with other risk factors as a worldwide perspective. Moreover, the authors identified the importance of air quality on loss of life expectancy on a global perspective. To explore the causality between the air quality and cardiovascular respiratory disorders, the authors used the Global Exposure Mortality Model (GEMM), which provided wide coverage of fine PM2.5 at a global scale. (6) analyzed the effect of PM2.5 on child mortality using the aggregated data across 16 Asian countries for 20 years. (7) integrated in an exposure-response function, the results from a recent atmospheric chemistry-general circulation model and health statistics from the WHO Global Health Observatory to estimate mortality from ambient air pollution.

Results

Table 1 shows the summary statistics of the main variables. The overall sample size is 136,052. In our sample, the average infant mortality rate is 5.4% and the average level of PM2.5 is 23.7. Table 2 shows the result of ordinary least squares estimation. In all specifications, the cluster fixed effect, year- and birth month fixed effect, year times country fixed effect, and birth month times country fixed effect were included. To calculate the standard error, it is assumed that the error term is correlated within each cluster over time. In Table 2, the estimated coefficient of PM2.5exposure in the conception year is 0.000984. This implies that the 10-microgram increase in PM2.5 exposure increases the probability that a child may die within one year from birth by 0.984%. As the average mortality rate is 5.3%, a 10-microgram increase in PM2.5exposure in the conception year increases the infant mortality rate by 18.32%. Several control variables, such as water source, rainfall, and nighttime light—as a proxy for income level—were included

Table 1: Summary Statistics

VARIABLES	(2)	(3)	(4)	(5)
	mean	sd	min	max
Infant mortality	0.0537	0.225	0	1
PM25 exposure in birth year	23.72	18.72	0	77.32
PM25 exposure in conception year	23.27	18.36	0	77.32
Total PM 25 exposure	23.52	18.54	0	76.44
Interview year	2,013	4,545	2,004	2,018
Interview month	7.63	3.754	1	12
Partners education (years)	6.103	4.995	0	24
Birth year of child	2,005	4,701	1,999	2,016
Birth month of child	6.506	3.445	1	12
Child gender	0.515	0.5	0	1
Mothers education (years)	5.206	4.915	0	25
Mothers' age	33.07	7.322	15	49
Head of household age	42.84	12.06	14	95
Head of household gender	0.886	0.318	0	1
Household size	6.662	3.123	1	35
Urban/Rural	0.391	0.488	0	1
Conception year	2,005	4,707	1,998	2,016
Year	2,005	4,701	1,999	2,016
Improved water source_dry	0.865	0.342	0	1
Improved water source_wet	0.875	0.331	0	1
Improved toilet	0.54	0.498	0	1
avg nightlight at year of age one	2,006	4,701	2,000	2,017
latitude	15.05	15.06	-9.478	35.83
longitude	100.1	20.36	65.43	127.2
total nightlight	6,327	12,143	-289.6	135,380
Avg nightlight in birthyear	5.607	10.58	-0.0692	61.77
Avg nightlight in conception year	5.731	10.7	-0.051	61.77
Total rainfall	235,891	158,988	-771,526	968,640
Avg rainfall in birth year	1,667	1,124	-5,472	6,821
Avg rainfall in conception year	1,667	1,121	-5,517	6,821
Bangladesh dummy	0.3	0.458	0	1
Cambodia dummy	0.061	0.239	0	1

Notes: Sample size is 136,052.

(Table 2). However, these control variables do not affect the estimated coefficients.

Table 2: The Effect of PM2.5 on Infant Mortality

Dependent Variable VARIABLES	Infant Mortality Dummy			
	(1)	(2)	(3)	(4)
PM25 exposure in conception year	0.000984***	0.00101***	0.00101***	0.00102***
	(0.000344)	(0.000345)	(0.000345)	(0.000346)
Cluster FE	YES	YES	YES	YES
CountryYear FE	YES	YES	YES	YES
BirthmonthCountry FE	YES	YES	YES	YES
InterviewmonthCountry	YES	YES	YES	YES
Individual covariates	YES	YES	YES	YES
Water source	NO	YES	YES	YES
Rainfall	NO	YES	YES	YES
Wealth	NO	NO	YES	YES
Nightlight	NO	NO	NO	YES
Sanitation	NO	NO	NO	NO
Cooking fuel	NO	NO	NO	NO
R-squared	0.047	0.048	0.048	0.048
N	136,052	136,052	136,052	136,052

Clustered robust standard errors in parentheses. Mean infant mortality dummy 0.0537074. *** p<0.01, ** p<0.05, * p<0.1

In Table 3, we separated the sample into male and female infants. The estimated coefficients obtained from the male and female infants are relatively similar to the estimated coefficients in Table 2, suggesting that the effect of PM2.5 is not different between male and female infants. We changed the main explanatory variable from the PM2.5 in the conception year to PM2.5 in the birth year (Table 4). The estimated coefficient of PM2.5 in the birth year is one third of the estimated coefficient in Table 2, which is not statistically significant, suggesting that the PM2.5 in the birth year is not as relevant as the PM2.5 in the conception year. We include both PM2.5 in the conception and birth years as the

Table 3: The Effect of PM2.5 on Infant Mortality for Male and Female Sample

Dependent Variable	Infant Mortality Dummy			
	(1)	(2)	(3)	(4)
Panel A. Male Sample				
PM25 exposure	0.000966*	0.000979*	0.000974*	0.000964*
in conception year	(0.000499)	(0.000502)	(0.000502)	(0.000502)
R-squared	0.064	0.065	0.065	0.065
N	70,108	70,108	70,108	70,108

Panel B. Female Sample				
PM25 exposure	0.000947**	0.000976**	0.000979**	0.00101**
in conception year	(0.000477)	(0.000479)	(0.000479)	(0.000480)
R-squared	0.064	0.065	0.065	0.065
N	65,944	65,944	65,944	65,944

Panel C. Control Variables				
Cluster FE	YES	YES	YES	YES
CountryYear FE	YES	YES	YES	YES
BirthmonthCountry FE	YES	YES	YES	YES
InterviewmonthCountry	YES	YES	YES	YES
Individual covariates	YES	YES	YES	YES
Water source	NO	YES	YES	YES
Rainfall	NO	YES	YES	YES
Wealth	NO	NO	YES	YES
Nightlight	NO	NO	NO	YES
Sanitation	NO	NO	NO	NO
Cooking fuel	NO	NO	NO	NO

Clustered robust standard errors in parentheses. Mean infant mortality dummy 0.0537074. *** p<0.01, ** p<0.05, * p<0.1

key explanatory variables (Table 5). The estimated coefficient of PM2.5 in the conception year was similar to the estimated coefficient of PM2.5 in the conception year, as shown in Table 2. Meanwhile, the estimated coefficients of PM2.5 in the birth year were almost zero, and not statistically significant. This suggests that PM2.5 at conception is more of a determinant of infant mortality than PM2.5 at birth.

Table 4: The Effect of PM2.5 at Birth Year on Infant Mortality

Dependent Variable	Infant Mortality Dummy			
	(1)	(2)	(3)	(4)
VARIABLES				
PM25 at birth year	0.000308	0.000349	0.000348	0.000348
	(0.000396)	(0.000397)	(0.000397)	(0.000399)
Cluster FE	YES	YES	YES	YES
CountryYear FE	YES	YES	YES	YES
BirthmonthCountry FE	YES	YES	YES	YES
InterviewmonthCountry	YES	YES	YES	YES
Individual covariates	YES	YES	YES	YES
Water source	NO	YES	YES	YES
Rainfall	NO	YES	YES	YES
Wealth	NO	NO	YES	YES
Nightlight	NO	NO	NO	YES
Sanitation	NO	NO	NO	NO
Cooking fuel	NO	NO	NO	NO
R-squared	0.046	0.047	0.047	0.048
N	136,052	136,052	136,052	136,052

Clustered robust standard errors in parentheses. Mean infant mortality dummy 0.0537074. *** p<0.01, ** p<0.05, * p<0.1

One concern about our estimation is that the PM2.5 is highly correlated with indoor cooking fuel, such as charcoal, and the high infant mortality is not due to outdoor air conditions, but to polluted air from cooking fuels. In addition, there is a concern that house toilet type is highly correlated with infant mortality, and the type of toilet is correlated with economic activity and PM2.5. We included the cooking fuel and the toilet type as additional control variables (Table 6). The estimated coefficients of PM2.5 in the conception and birth years did not change from the key explanatory variables in Table 5. In Table 6, a 10-microgram increase of PM2.5 in the conception year increases the probability of infant mortal-

Table 5: The Effect of PM2.5 at Conception Year and at Birth Year on Infant Mortality

Dependent Variable	Infant Mortality Dummy			
	(1)	(2)	(3)	(4)
VARIABLES				
PM25 in conception year	0.000984***	0.00101***	0.00101***	0.00102***
	(0.000344)	(0.000345)	(0.000345)	(0.000346)
PM25 in birth year	-0.000191	-0.000150	-0.000150	-0.000154
	(0.000434)	(0.000435)	(0.000435)	(0.000436)
Cluster FE	YES	YES	YES	YES
CountryYear FE	YES	YES	YES	YES
BirthmonthCountry FE	YES	YES	YES	YES
InterviewmonthCountry	YES	YES	YES	YES
Individual covariates	YES	YES	YES	YES
Water source	NO	YES	YES	YES
Rainfall	NO	YES	YES	YES
Wealth	NO	NO	YES	YES
Nightlight	NO	NO	NO	YES
Sanitation	NO	NO	NO	NO
Cooking fuel	NO	NO	NO	NO
R-squared	0.046	0.047	0.047	0.048
N	136,052	136,052	136,052	136,052

Clustered robust standard errors in parentheses. Mean infant mortality dummy 0.0537074. *** p<0.01, ** p<0.05, * p<0.1

Table 6: The Effect of PM2.5 at Conception Year and at Birth Year on Infant Mortality: Controlling Type of Cooking Fuel and Sanitation

Dependent Variable	Infant Mortality Dummy			
	(1)	(2)	(3)	(4)
VARIABLES				
PM25 in conception year	0.00104***	0.00105***	0.00105***	0.00106***
	(0.00038)	(0.00038)	(0.00038)	(0.00038)
PM25 in birth year	-0.000199	-0.00016	-0.000161	-0.000165
	(0.00043)	(0.00044)	(0.00044)	(0.00044)
Cluster FE	YES	YES	YES	YES
CountryYear FE	YES	YES	YES	YES
BirthmonthCountry FE	YES	YES	YES	YES
InterviewmonthCountry	YES	YES	YES	YES
Individual covariates	YES	YES	YES	YES
Water source	NO	YES	YES	YES
Rainfall	NO	YES	YES	YES
Wealth	NO	NO	YES	YES
Nightlight	NO	NO	NO	YES
Sanitation	YES	YES	YES	YES
Cooking fuel	YES	YES	YES	YES
R-squared	0.047	0.048	0.048	0.048
N	136,052	136,052	136,052	136,052

Clustered robust standard errors in parentheses. Mean infant mortality dummy 0.0537074. *** p<0.01, ** p<0.05, * p<0.1

ity rate by 1%. This implies that a 10-microgram increase of PM_{2.5} increases the infant mortality by 19%. In contrast, the effect of PM_{2.5} on birth year was almost negligible and was not statistically significant.

One key observation from Table 5 and Table 6 is that the PM_{2.5} at conception has an effect on infant mortality relative to the PM_{2.5} at birth. There are several concerns for this results. In particular, the PM_{2.5} in the conception year and that in the birth year are highly correlated. Consequently, when the two variables are put together, the estimated coefficient of PM_{2.5} in the birth year becomes negligible, and the effect is not statistically significant. However, such concern does not seem relevant. In Table 3, we included only the PM_{2.5} in the birth year and other control variables. In this case, the estimated coefficient of PM_{2.5} in the birth year was one third and not statistically significant, suggesting that the results shown in Tables 5 and 6 are not driven by the multi-colinearity between PM_{2.5} at conception and birth. Another concern is that our sample included children who were born between January and February. As we measure PM_{2.5} annually, children who were born between January and February were exposed to PM_{2.5} in the birth year for only two months, whereas those children were exposed to PM_{2.5} in the conception year for eight months. Thus, one can assume that the economically and statistically insignificant results might be due to the discrepancy in the duration of exposure to PM_{2.5} between birth and conception years. To factor out this concern, we excluded children who were born between January to March for an additional robustness check. Thus, in our restricted sample, children are exposed to PM_{2.5} in the birth year for at least three months. Our regression analysis (Table 7) revealed that even if we exclude children who were born between January and March, the estimated coefficient of PM_{2.5} in the conception year is economically and statistically significant, whereas the estimated coefficient of PM_{2.5} in the birth year is almost zero and not statistically significant.

Discussion

We examined the effect of PM_{2.5} exposure on infant mortality by constructing a cluster-level panel with microdata while merging the high-resolution PM_{2.5} map created using the information on PM_{2.5} data collected via satellite. We found that a 10-microgram increase in PM_{2.5} in the conception year increases infant mortality by 19%. In contrast, the effect of PM_{2.5} in the birth year is almost zero and not statistically significant. Overall, our results corroborate the findings of the previous literature, showing that PM_{2.5} affects infant mortality. However, quantitatively, our estimates showed a remarkable difference in the effect of timing on exposure to PM_{2.5}. In particular, our estimated coefficient shows that the effect of PM_{2.5} is about twice as large as the estimate obtained from Africa's data. For example, Heft-Neal et al. (2018) reported that a 10-microgram increase in PM_{2.5} increases infant mortality by 10%. Moreover, we found that the timing of PM_{2.5} exposure matters substantially. In our analysis, we found that PM_{2.5} levels in the conception year significantly affect infant mortality, while the effect of PM_{2.5} in the birth year is negligible. There are two reasons that our estimates are larger than the previous estimates. First, the content of PM_{2.5} in Africa is mainly sand dust, whereas in South Asia, PM_{2.5} comes from industry sources, such as emissions

from transportation equipment and power-generating facilities. Second, the infant mortality in Asia is 40% lower than that in Africa. These two factors are likely to contribute to the large effect of PM_{2.5} on infant mortality.

Conclusion

In this study, we assessed the effect of PM_{2.5} exposure on infant mortality. We constructed a worldwide cluster-level panel dataset using the GPS information of each cluster and combined the birth records from the DHS of 50 countries throughout 20 y. We mapped the PM_{2.5} information obtained from satellite to each cluster and year. We found a 10-microgram increase in PM_{2.5}, which is associated with an approximately 20% increase in infant mortality rate in Asian countries relative to the 10% increase in African countries. Further, we found that the effect of PM_{2.5} level in the conception year has an economically and statistically significant effect on infant mortality, while the PM_{2.5} in the birth year has negligible effect. Our results are particularly robust as supported with several specification checks.

Material and Method

A. Data Sets. We use the data from the Demographic and Health Surveys (DHS), nationally representative surveys that are conducted in developing countries (ICF, 1998). The DHS is based on two-stage sampling, in which a number of clusters are first selected from a list of enumeration areas created in a recent population census. Subsequently, 5–10 households were randomly selected in each of the clusters, and women aged 15–49 y were selected from the selected households for interviews on birth record. In many waves of DHS, the enumerator carries global positioning system (GPS) devices to collect geospatial information to identify the central point of each cluster's populated area. For data confidentiality, randomness was added to this GPS information. A 0–2-km and 0–5-km displacement was added to the GPS information in urban and rural areas, respectively. The sample used in our analysis was obtained from the birth record file in the DHS. We included all infants born between 1999–2016.

Our dependent variable was the infant mortality dummy, wherein this dummy variable takes the value of one if the infant died by the 12th month after the birth. Infants who were less than 12 months old and were alive at the time of the interview were excluded as their birth outcome at 12 months was unknown. The key explanatory variable is the cluster in which a corresponding child is born. To calculate the PM_{2.5} of each infant, we use satellite-derived data on PM_{2.5} compiled by NASA (Van Donkelaar et al., 2015). These data were derived from a suite of satellite-based atmospheric optical depth measurement instruments, including the two MODIS instruments, the Multi-Angle Resolution Spectroradiometer (MISR) on Terra and the Sea-viewing Wide Field-of-View Sensor (SeaWiFS) on the SeaStar satellite. These data were combined with aerosol profile measurements from the Cloud-Aerosol Lidar with Orthogonal Polarization (CALIOP) instrument aboard the Cloud-Aerosol Lidar and Infrared Pathfinder Satellite Observation (CALIPSO), and satellite, weather and seasonality data from the GEOS-Chem Chemical transport model to quantify the relationship between column Aerosol Optical Depth (AOD) and surface PM_{2.5} measured at available ground-based

stations. Using this PM2.5map, we calculated the PM2.5 using GPS information of the cluster in which the infant is born. Specifically, we made a circle with a 10-km radius at each cluster. The average PM2.5 levels of each cluster were then calculated.

The temperature and rainfall data at each cluster, which were obtained from Fick and Hijmans (2017), were calculated. To control the annual income level at each village, we used the nighttime light data constructed by the National Oceanic and Atmospheric Administration, National Geophysical Data Center (2015).

B. Data Construction. The datasets in the DHS are cross-sectional. GPS data from the village were obtained to create the village-level panel. If the distance between two villages in different DHS waves is less than 10 km, it is assumed that they are comparable and they are assigned the same village ID. In the base year, 345 village-level clusters were selected for Bangladesh, 259 for Pakistan, 221 for Nepal, 504 for the Philippines, 400 for Timor Leste, and 484 for Cambodia. We selected these countries by criteria of GPS data availability. If GPS data for each survey were present, we used the dataset from that country. With GPS information, we matched the children's data with PM2.5 information using information of the infant's birth timing and GPS information of the village. To distinguish the exposure to PM2.5 during the mother's prenatal period and after birth, we measured two indices of PM2.5. From 1999 to 2016, the birth records from waves of the DHS in Bangladesh, Pakistan, Nepal, the Philippines, Timor Leste, and Cambodia were used. For Bangladesh, we used 2004 as the base, while 2007, 2011, 2014, and 2017 were the waves. For Pakistan, we used 2016 and 2017 as the waves. For Philippines, 2003, 2008, and 2017 waves were used from the DHS. Timor Leste had two waves from 2009 and 2016. Meanwhile, Cambodia had 2005, 2010, and 2014. For Nepal, 2001 and 2006 were used for the data construction.

Satellite-based PM2.5 maps were used to assess the PM2.5 in village-level clusters from 1998 to 2016. A 10-km-radius circle around each cluster-based village was drawn using QGIS buffer, and the average PM2.5 intensity was calculated for each circle. The village and the time fixed effect were considered during the estimation. Subsequently, we used the PM2.5 time series fluctuation at each settlement. Birth mortality was coded for one year in all other birth data. The infant mortality dummy is zero if the child survives 13 months after delivery. The infant mortality dummy is coded as one if the child dies before the 12th month following delivery. According to our identifying assumption, after controlling for country- and cluster-level fixed effects, infant mortality can be attributed to air pollution by PM2.5.

Overall, there were 150,750 observations from all six countries, which were arranged by the number of clusters in each DHS survey. As our main dependent variable of interest is infant mortality, we used birth records from the DHS dataset from the respective country. The GPS data are essential to track the infant mortality by PM2.5 exposure level. By using GPS data, we generated a village-level cluster panel dataset. This panel is balanced, with other explanatory variables, including respondent's education, household size, drinking water source, and head of household's information, such as sex, and education. For our analysis we excluded infants born after 2016 and before 1999 as the PM2.5 data were available only

between 1996–2016. An estimated probability was successfully calculated using dummy infant mortality. We used the water variable dummy for improved and unimproved water sources for every country, and average precipitation data from the CHIRPS (quasi-global rainfall data).

C. Model. We utilized the ordinary least squares regression model for the analysis. We estimated the following equation:

$$Y_{cnmti} = \beta_0 + \beta_1 PM_{cnt}^{2.5} + \beta_2 X_{icnmt} + \mu_c + \delta_t + \delta_{tc} + \delta_{nm} + \varepsilon_{icnmt} \quad [1]$$

where Y_{cnmti} represents the infant mortality dummy, i is the index of the child, c is the index of the village, t is the index of birth year, n is the index of country, and m is an index of birth month. $PM_{cnt}^{2.5}$ is the PM2.5 at birth or conception, which is our main explanatory variable of interest. In some specification, we put both PM2.5 in the birth and conception year. In our final specification, we included both PM2.5 at conception and birth. Note that as we have annual information on PM2.5, we do not have information on PM2.5 during the birth month or the conception. X_{icnmt} are the individual covariates (e.g., child sex, household qualities), whereas μ_c and δ_t are the village and birth year fixed effects, respectively. The δ_{nm} controls the country birth month fixed effects. The country month of birth fixed effects control the country's specific seasonal variations in infant mortality. The δ_{tc} is the country year fixed effect that controls the country-specific time trend of mortality owing to changes in the health system or national-level income.

We also used nighttime light data to control the effect of income as a country-level economic indicator. Birth month was controlled for each infant born and birth month by country-level fixed effects was also added. This variable accounts for the time-invariant unobservable factors, which could be correlated to mortality and PM2.5 within the cluster and country levels. Thus, if a particular month of birth has a higher or lower mortality, it will be accounted for by the fixed effects. Additionally, we partial out the interview month by country fixed effects to control unobserved factors that impact infant mortality and

1. WHO (2017) The cost of a polluted environment: 1.7 million child deaths a year, says who (www.who.int/mediacentre/news/releases/2017/pollution-childdeath/en/). Accessed January 15th 2021.
2. Heft-Neal S, Burney J, Bendavid E, Burke M (2018) Robust relationship between air quality and infant mortality in africa. *Nature* 559(7713):254–258.
3. Hammer MS, et al. (2020) Global estimates and long-term trends of fine particulate matter concentrations (1998–2018). *Environmental Science & Technology* 54(13):7879–7890.
4. Van Donkelaar A, et al. (2016) Global estimates of fine particulate matter using a combined geophysical-statistical method with information from satellites, models, and monitors. *Environmental science & technology* 50(7):3762–3772.
5. Chen YT, et al. (2021) Association between short-term exposure to air pollution and sudden infant death syndrome. *Chemosphere* 271:129515.
6. Anwar A, Ullah I, Younis M, Flahault A (2021) Impact of air pollution (pm2.5) on child mortality: Evidence from sixteen asian countries. *International Journal of Environmental Research and Public Health* 18(12):6375.
7. Lelieveld J, Haines A, Pozzer A (2018) Age-dependent health risk from ambient air pollution: a modelling and data analysis of childhood mortality in middle-income and low-income countries. *The lancet Planetary health* 2(7):e292–e300.