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# Early-life exposure to ambient fine particulate air pollution and infant mortality: pooled evidence from 43 low- and middle-income countries

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## Abstract

**Background:** Many low- and middle-income countries are experiencing high and increasing exposure to ambient fine particulate air pollution ($PM_{2.5}$). The effect of $PM_{2.5}$ on infant and child mortality is usually modelled using concentration response curves extrapolated from studies conducted in settings with low ambient air pollution, which may not capture its full effect.

**Methods:** We pool data on more than half a million births from 69 nationally representative Demographic and Health Surveys that were conducted in 43 low- and middle-income countries between 1998 and 2014, and we calculate early-life exposure (exposure *in utero* and post partum) to ambient $PM_{2.5}$ using high-resolution calibrated satellite data matched to the child's place of residence. We estimate the association between the log of early-life $PM_{2.5}$ exposure, both overall and separated by type, and the odds of neonatal and infant mortality, adjusting for child-level, parent-level and household-level characteristics.

**Results:** We find little evidence that early-life exposure to overall $PM_{2.5}$ is associated with higher odds of mortality relative to low exposure to $PM_{2.5}$. However, about half of $PM_{2.5}$ is naturally occurring dust and sea-salt whereas half is from other sources, comprising mainly carbon-based compounds, which are mostly due to human activity. We find a very strong association between exposure to carbonaceous $PM_{2.5}$ and infant mortality, particularly neonatal mortality, i.e. mortality in the first 28 days after birth. We estimate that, at the mean level of exposure in the sample to carbonaceous $PM_{2.5}$—10.9 $\mu g/m^3$—the odds of neonatal mortality are over 50% higher than in the absence of pollution.

**Conclusion:** Our results suggest that the current World Health Organization guideline of

limiting the overall ambient $PM_{2.5}$ level to less than 10 $\mu g/m^3$ should be augmented with a lower limit for harmful carbonaceous $PM_{2.5}$.

**Key words:** Ambient air pollution, fine particulate matter, pregnancy outcome, child mortality, infant mortality, neonatal mortality, Demographic and Health Survey (DHS), Geographic Information System (GIS)

**Key Messages**

- A high level of early-life exposure to overall ambient fine particulate matter ($PM_{2.5}$) is only weakly associated with higher odds of neonatal mortality in children in low- and middle-income countries.
- There is a strong association between exposure to carbonaceous $PM_{2.5}$ and mortality in the neonatal period.
- The association between early-life $PM_{2.5}$ exposure in the form of dust and sea-salt, which make up over half of all fine particulates, and mortality is weak; however, other types of fine particulates, which are mainly due to human activity, are strongly associated with mortality, even at low levels of exposure.
- Reducing ambient carbonaceous $PM_{2.5}$ could contribute substantially to achieving the Sustainable Development Goal of lowering neonatal mortality to below 12 per 1000 children by 2030.
- The World Health Organization guideline for overall ambient $PM_{2.5}$ to be below 10 $\mu g/m^3$ should be augmented with a guideline for ambient $PM_{2.5}$ excluding dust and sea-salt at a lower level.

## Introduction

Over 2.5 million children die annually within the first 28 days of birth, with three out of four of these neonatal deaths occurring in Southern Asia and sub-Saharan Africa.[1] At the same time, many low- and middle-income countries are becoming increasingly exposed to adverse environmental stresses, particularly ambient fine particulate air pollution ($PM_{2.5}$),[2] and estimates from the World Health Organization (WHO) find that over 4.2 million deaths worldwide can be attributed to ambient air pollution.[3] Given the evidence of the relationship between exposure to ambient air pollution and under-5 mortality,[4–10] high and rising ambient air pollution is likely to be a key determinant for the continuing high rate of neonatal and child mortality in low- and middle-income countries.[11]

The dominant approach in the literature is to model global mortality due to ambient air pollution, both in children under age 5 as well as in adults, using concentration response curves.[12–16] These curves are estimated from studies in high-income countries. Due to the scarcity of studies in low-income settings where ambient air pollution is very high, integrated concentration response curves are constructed that use results from studies of active smoking and second-hand smoke to estimate the effect of high levels of $PM_{2.5}$. This modelling approach, however, is open to question, since not only is ambient air pollution in low- and middle-income countries generally higher than in high-income countries, but it also has different sources and toxicity, and hence its effect may be different from that of tobacco.[17–20]

Two studies have directly estimated the relationship between early-life exposure to ambient air pollution and under-5 mortality at the local level in urban middle-income settings: one in Mexico City and one in São Paulo, Brazil.[21,22] Both these studies find an association between intrauterine mortality and air pollution; moreover, the studies find that mortality was most strongly associated with exposure to ambient air pollution within a few days before death, with the strongest association observed for an increase in the average concentration 3–5 days prior to death. In addition, the average ambient air-pollution level has been found to be related to national child-mortality rates in Africa.[23]

We improve on this evidence by compiling a global sample of individual-level child data from a wider range of low- and middle-income settings, from which we are able to match child-mortality data to the local ambient $PM_{2.5}$ pollution level when the children were *in utero* and in their first year of birth. After we submitted this for review, a study with a similar approach to ours was published[24] finding a robust relationship between overall $PM_{2.5}$ and infant mortality in Africa. In comparison to that study, our sample covers countries worldwide—for which data are available—and not only in Africa. In addition, we focus on results for $PM_{2.5}$ pollution by type, separating naturally occurring dust and sea-salt from other types of $PM_{2.5}$, which

we find to be more harmful. Also, we focus more on disaggregating the timing of death and examine the relationship of $PM_{2.5}$ exposure with death in the first month as well as the first year after birth.

Although we focus on child mortality in this study, we recognize that air pollution can also be linked to other health outcomes, such as respiratory infection, low birthweight and child stunting.[8,14,25–27] Three recent systematic reviews and pooled cohort studies found that maternal exposure to ambient $PM_{2.5}$ is strongly, and likely causally, associated with an increase in risk in pre-term birth and low birthweight,[28–30] whereas a third systematic review found associations between maternal exposure and small-for-gestational-age births,[31] although all reviews reviewed studies of samples drawn from high-income countries. Several systematic reviews and meta-analyses have identified the significant linkages between air pollution and mortality in adults, both in high-income settings[32] as well as in low- and middle-income settings.[33] The Nashville Air Pollution study was possibly the first to suggest that chronic exposure to ambient air pollution was related to neonatal death[34] and similar findings have been reported in several other studies in developed countries,[6,8,16,35–40] although there is little evidence that examines the relationship between exposure to ambient air pollution and child mortality in low- and middle-income settings. Given the evidence from the literature to date, it is likely that the link between air pollution and perinatal outcomes (reduced birthweight, pre-term birth, etc.) is more than an association and tilts heavily towards causality.

## Methods

In this study, we pool data on children born in the previous 5 years from 69 nationally representative surveys that were conducted in 43 low- and middle-income countries from 1998 to 2014. We combine the pooled DHS dataset with high-resolution spatial data on ambient $PM_{2.5}$ to analyse the relationship between exposure to ambient air pollution and child mortality. We focus on $PM_{2.5}$ because its association with child-health outcomes has been found to be more pronounced than other sizes of particulates.[36] However, the composition and sources of the particulates may matter in addition to their sizes[41,42] and we distinguish dust and sea-salt, which make up over half of total exposure, from other types of particulates.

### Study population

We obtain data on children born in the last 5 years from the Demographic and Health Surveys (DHSs), which are nationally representative, cross-sectional household surveys that cover a range of health topics.[43] The DHS employs a two-stage cluster sampling design, stratifying by region and urban/rural residence and interviewing about 20–30 women aged 15–49 per primary sampling unit (a DHS cluster), each of which generally corresponds to a census enumeration area and is randomly selected within each stratum.[44] For each woman of reproductive age (aged 15–49) in sampled households, detailed information is recorded on each of her births over the previous 5 years, including whether the child has died and, if so, the age of death. In many DHS surveys, the location of each cluster of sampled DHS households (typically between 20 and 30 households) is recorded in the dataset at the centroid of the sampled households, although noise is added to the reported coordinates in order to protect respondent privacy. Specifically, urban clusters are randomly displaced up to a distance of 2 km from the true cluster location, whereas rural clusters are randomly displaced up to 5 km[45]—we account for this geographic perturbation when calculating our exposure as per the DHS recommended methodology.[46] We collected data from all 104 DHS surveys conducted between 1998 and 2014 that included global positioning system (GPS) data of DHS cluster locations. Of these surveys, 69 surveys are used for the main analysis based on availability of data on outcomes, exposures and covariates (see Supplementary Table 2 and Supplementary Figure 1, available as Supplementary data at *IJE* online). After excluding observations with missing data, our resulting sample consists of 534 476 children born in 34 450 clusters across 43 countries (see Supplementary Table 3, available as Supplementary data at *IJE* online).

### Exposure

The key explanatory variable in this study is exposure to $PM_{2.5}$ measured in micro grams per cubic meter ($\mu g/m^3$). Estimates are produced by the Atmospheric Composition Analysis Group[19,47] and are based on triangulation of three data sources. Aerosol Optical Depth is estimated from satellite data using both passive data and active laser scattering. These estimates of fine particulate matter are bias-corrected and calibrated to available ground-based monitoring data.[19,47] Additionally, they have a model of land-use and pollution sources, particle composition, dispersion in the atmosphere and optical properties that is used to further adjust the estimates. This source modelling is also used to determine the composition of the $PM_{2.5}$ particles. The data provide annual average $PM_{2.5}$ overall, and by type, for the period 1998–2016, at a resolution of $0.01 \times 0.01^{\circ}$ (approximately $1 \times 1$ km). Similar data have been used by the Global Burden of Diseases studies to model attributable disability-adjusted life years to ambient air pollution.[14,48]

Sources of fine particulate matter include both natural sources, mostly desert dust and sea-salt, and anthropogenic sources, such as emissions from industries, transportation, residential energy use, electricity generation, biomass burning and agriculture.[18,49] These sources produce different types of particulates, which in turn may have heterogeneous health effects. We therefore report results that disentangle exposures due to naturally occurring dust and sea-salt from other particulate exposures that are mainly organic carbon-based and man-made.

The data on $PM_{2.5}$ are matched temporally and spatially with each birth in our dataset. For infant mortality, we estimate the exposure of the child *in utero* pre-birth and the first year post birth. Given the lack of available data on gestational age in the DHS, we assume that each birth was carried to a full 9-month term. This exposure therefore covers 21 months. This period will overlap with 2 or 3 years of our annual average $PM_{2.5}$ data. We weight each year of observed data by the number of months of exposure of the child in that year divided by 12. Our calculation of early-life $PM_{2.5}$ exposure assumes that the level of $PM_{2.5}$ is constant and uniform across months within a calendar year. For example, for a child who was born in May 2016, her level of early-life $PM_{2.5}$ exposure for infant mortality would be calculated as the weighted average of:

1. her *in utero* exposure to $PM_{2.5}$, which is the sum of four-twelfths of the $PM_{2.5}$ level from 2015 (the child's first 4 months *in utero* from September 2015 to December 2015) and five-twelfths of the $PM_{2.5}$ level from 2016 (the child's last 5 months *in utero* from January 2016 to May 2016); and
2. her post-partum level of exposure to $PM_{2.5}$; 1 year after birth exposure to be the sum of the remaining seven-twelfths of the $PM_{2.5}$ level for 2016, which would capture her level of exposure from June 2016 to December 2016, and the first five-twelfths of the $PM_{2.5}$ level for 2017, which would capture her level of exposure from January 2017 to May 2017.

For neonatal mortality, we calculate the average exposure in the same way, but use data only from 1 month post birth. For post-neonatal infant mortality, we use the same exposure as for infant mortality.

We obtain the geographically specific $PM_{2.5}$ level for each child in our sample by matching the GPS coordinate data for each DHS cluster with the annual average ambient $PM_{2.5}$ concentrations for that cluster. We construct an average $PM_{2.5}$ exposure over the relevant displacement radius around the reported DHS cluster location (a 2-km average exposure radius for urban clusters and a 5-km average exposure radius for rural clusters). The averaging of the exposure over the displacement radius corrects for the spatial displacement of the DHS cluster coordinates[27] and is the methodology that is recommended by the DHS.[46] This procedure to construct the average $PM_{2.5}$ exposure is conducted using QGIS software (version 2.18.24; Open Source Geospatial Foundation Project). The Zonal Statistics Plugin of QGIS, which we use to average the $PM_{2.5}$ concentration level over the displacement radius, includes only grid squares whose midpoint falls within the displacement radius in this computation. On average, an urban cluster included approximately 10 $PM_{2.5}$ grid squares whereas a rural cluster included approximately 65 grid squares.

## Outcomes

We measure three outcomes—infant mortality, deaths that occur in the first year after birth and two sub-periods, neonatal morality that occurs in the first 28 days post birth and post-neonatal infant mortality. Recent global evidence finds that 76% of all deaths among children under age 5 occur within the first year of life and that 46.2% of all deaths among children under age 5 (and 61% of all deaths among children under age 1) occur within the first month of life.[1] The neonatal and post-neonatal infant mortality are mutually exclusive categories. The sample for infant mortality comprises all births more than 1 year before the survey so that we can observe whether infant mortality has occurred. The sample for neonatal mortality is all births at least 28 days before the survey so we can observe whether neonatal mortality has occurred. The sample for post-neonatal infant mortality consists of children who were born at least 1 year before the survey but survived to at least 28 days of age. A difficulty with examining under-5 mortality in our sample is that we do not observe whether a child survives to age 5, since all the children are aged less than 5 in the survey.

## Statistical analysis

We estimate the relationship between ambient early-life $PM_{2.5}$ exposure and child mortality using multivariate logistic regressions. We provide results for the overall level of $PM_{2.5}$ and separate regressions where we have two exposure variables: $PM_{2.5}$ with and without dust and sea-salt.

All regressions control for child-level, parental-level and household-level characteristics.[50] The child-level variables are whether the child was first born, the birth order of the child, the interval from the previous birth, whether the child was a multiple birth and the sex of the child. Parental-level characteristics include the age of the mother, the education level of the mother, whether the mother used

tobacco[51,52] and the education level of the mother's partner. Household-level characteristics include the place of residence (rural or urban), the type of cooking fuel used in the household (solid cooking fuel has been linked to indoor air pollution),[53] the type of toilet facility accessible to the household (flush-toilet facility or not), the source of drinking water available to the household (piped drinking water or not) and the wealth quintile of the household.[54–58] These covariates were selected based on existing evidence on the key determinants of infant mortality in low- and middle-income settings.[59–61] In addition, all our regressions include subnational region fixed effects and survey dummies. Finally, we include a country-specific trend in birth year[62,63] to capture improvements in child health over time.

Estimates from our regressions are presented as odds ratios (ORs) with standard errors clustered at the DHS cluster level to account for a sampling methodology and allow for correlations between outcomes for children within a DHS cluster.

Recent studies have calibrated the relationship between the ambient $PM_{2.5}$ level and child mortality by using either a logarithmic concentration response curve or an integrated exposure response curve.[36,64] For our main model, we estimate the association between early-life $PM_{2.5}$ exposure and mortality using logarithmic concentration response curves. We test this functional form assumption by comparing it with a linear response curve and a spline with three knots. In addition, we run a regression of neonatal mortality on categorical $PM_{2.5}$ exposure by dividing the sample into eight equal bins of $PM_{2.5}$ level, both overall as well as by source.

We also conduct several robustness checks of our main specification. An important issue is that mothers and children may have moved between conception and the time of the survey, which would render our exposure variables invalid. Information on such migration is not recorded in all surveys. We therefore give results restricting the sample to children for whom we know the mother has not migrated. This restricts the sample size by dropping many surveys that do not have this information. We also give results that drop country-specific trends in the birth year and subnational region fixed effects.

As an additional analysis, we estimate the country-specific relationships between early-life exposure to $PM_{2.5}$ and neonatal mortality for each country in our sample by separating out exposure by source and using the same fully adjusted logarithmic response curve specification as our main model. We then conduct a meta-analysis that relates the country-specific estimates for a one-unit increase in the logarithm of the $PM_{2.5}$ level to the pooled global estimate and we present these estimates using a Forest plot. In our meta-analysis, the relative contribution (weight) of each country-specific estimate to the pooled estimate is calculated and the 95% confidence interval (CI) from each country-specific analysis is estimated. We include random effects in accordance with the DerSimonian and Laird method,[65] and we conduct a test of whether the overall pooled estimate is equal to the null as well as a test for heterogeneity, i.e. whether the country-specific estimates are the same. This heterogeneity is quantified using the I-squared measure.[66]

All analyses are conducted using STATA software (version STATA/SE 14.1; StataCorp LP, College Station, Texas, USA).

## Results

Table 1 presents descriptive statistics of the key outcome variables in our analysis. For neonatal mortality, our primary outcome of interest, we find that 2.8% of children in the sample of 529 974 children who were potentially exposed to mortality in the first month of life died in the neonatal period. Similarly, 2.8% of children of a sample of 413 397 potentially exposed children died in the post-neonatal infant period (between 1 month and 1 year post partum). Finally, we find that 5.6% of children in our global sample died within the first year of life.

Figure 1 presents the geographical distribution of annual average ambient $PM_{2.5}$ concentrations in 1998 and 2014 and shows that the highest concentrations in low- and middle-income countries occur in desert regions, such as the Sahara, the Arabian Gobi and the Thar deserts, due to dust. Figure 2 shows the distribution of $PM_{2.5}$ concentrations excluding dust and sea-salt in 1998 and 2014. Based on the figure, we observe high concentrations in the Eastern USA, parts of the Western USA (the Californian central valley and the South Coast Air Basin, in particular), Europe and in South and Eastern Asia, due to human industrial and transportation activities, as well as in the Amazon and sub-Saharan Africa due to biomass burning.

**Table 1.** Descriptive statistics: outcomes

| | Number of observations | Number of cases | Proportion |
|---|---|---|---|
| Neonatal death | 529 874 | 15 042 | 0.028 |
| Post-neonatal infant death | 413 397 | 11 730 | 0.028 |
| Infant death | 425 440 | 23 773 | 0.056 |

The number of observations differ for the three outcomes because the sample for neonatal mortality consists of children who were born at least 1 month before the survey date, whereas the samples for post-neonatal infant mortality and infant mortality consist of children who were born at least 1 year before the survey date.

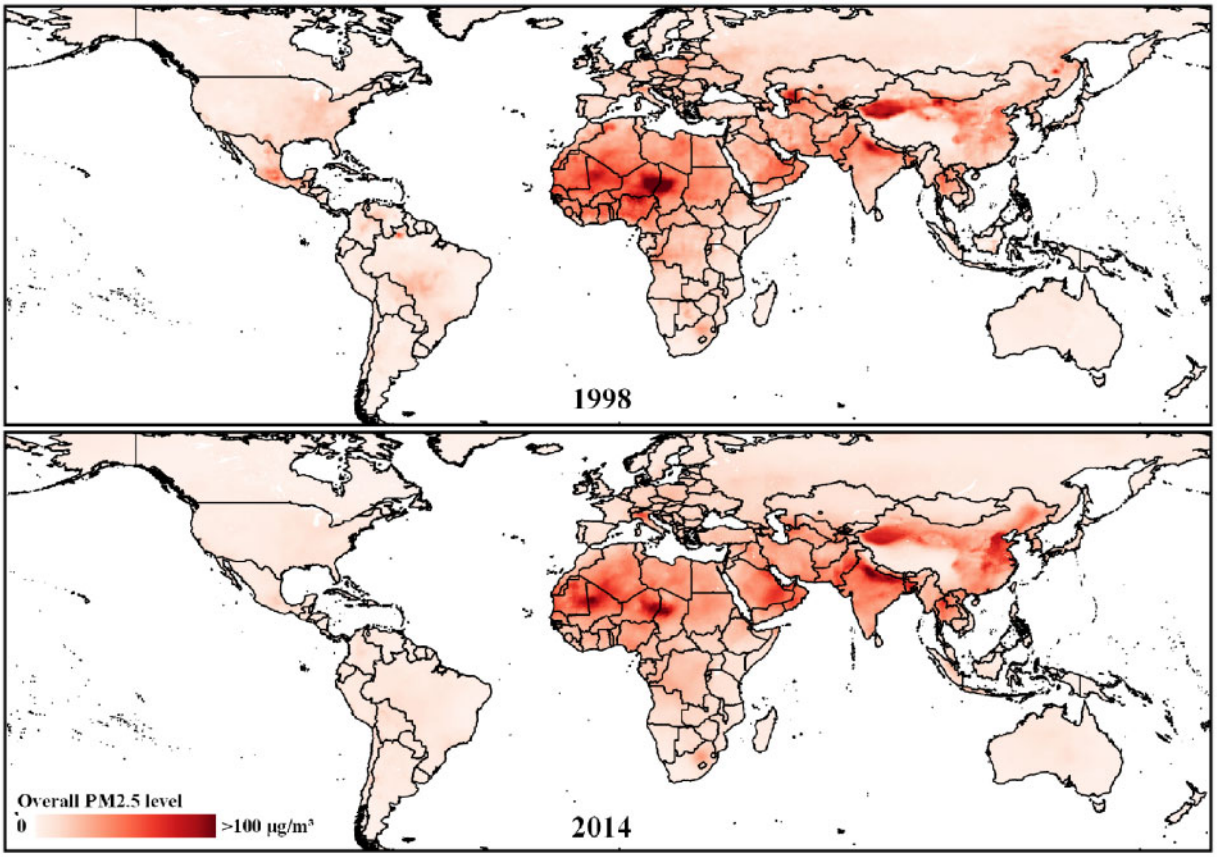


**Figure 1.** Geographic distribution of overall annual ambient $PM_{2.5}$ concentration in 1998 (top) and 2014 (below). Observations above 100 $\mu g/m^3$ in the data have been capped at 100 $\mu g/m^3$ in the figure for visualization.

Table 2 presents descriptive statistics of early-life $PM_{2.5}$ exposure for the neonatal sample, both in absolute levels as well as in logged levels, the latter of which are used for our main logarithmic concentration response analysis. Descriptive statistics for exposures for the post-neonatal infant and infant samples are presented in Supplementary Tables 4 and 5, available as Supplementary data at *IJE* online. The mean exposure to overall $PM_{2.5}$ in the sample is 24.43 $\mu g/m^3$ and a majority of children in our sample are exposed to $PM_{2.5}$ levels that exceed the WHO guideline of 10 $\mu g/m^3$ (Table 2). The mean exposure to $PM_{2.5}$ without dust and sea-salt was 10.88 $\mu g/m^3$, whereas the mean exposure to $PM_{2.5}$ from dust and sea-salt was 13.55 $\mu g/m^3$.

Figure 3 presents box plots of early-life exposure to $PM_{2.5}$ without dust and sea-salt in our sample by country. We find that Nepal suffers from the worst pollution in our sample, due to an inflow of pollution from India on the prevailing south-westerly winds and the bowl-shaped topography of the Kathmandu valley, which can lead to pollutants being trapped in the area.[67,68] Some sub-Saharan African countries, such as the Democratic Republic of Congo, also report a high level of ambient air pollution from non-dust and sea-salt sources, which may be due to high levels of biomass burning.[47]

Descriptive statistics for the selected covariates for our neonatal-mortality sample are presented in Table 3. We find that 82% of households of children in our neonatal sample use solid cooking fuel, while 16% of households for the sample have access to a flush toilet and 31% of households have access to piped water. Almost 30% of children in our neonatal sample are from urban households, whereas 72% of children are born to mothers who have a primary education or less. Roughly half of the sample is female, while 44% of children in the sample are born following a short birth interval (within 3 years of the previous birth). Descriptive statistics for covariates for the post-neonatal infant and infant samples are presented in Supplementary Tables 4 and 5, available as Supplementary data at *IJE* online.

Our main analysis estimates the relationship between exposure to ambient $PM_{2.5}$ and mortality using logarithmic concentration responses. We find the association between early-life exposure to overall $PM_{2.5}$ and neonatal, post-neonatal infant and infant mortality to be inconclusive (Table 4, columns 1–3). On the other hand, the estimated associations between our selected covariates and mortality are in the expected direction. Being a female child, having a mother or a mother's partner with higher

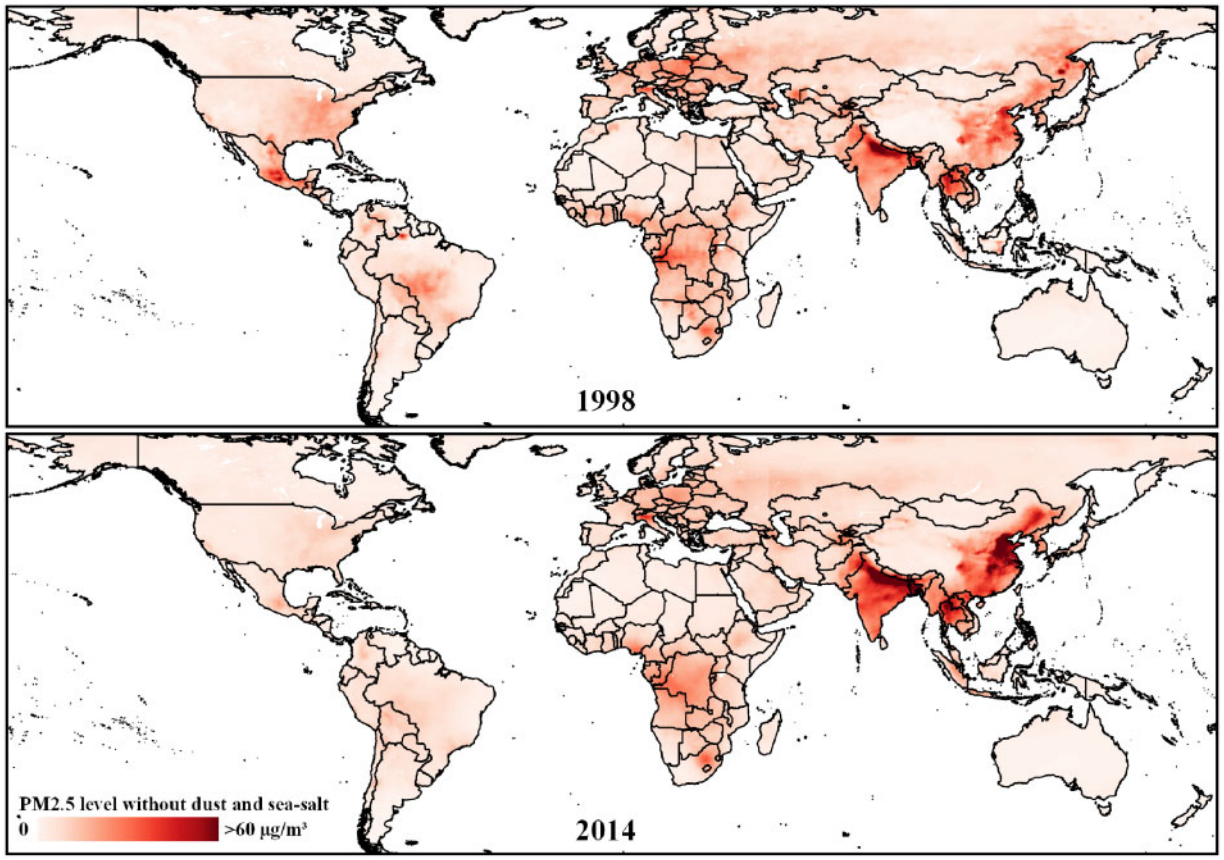


**Figure 2.** Geographic distribution of annual ambient $PM_{2.5}$ concentration without dust and sea-salt in 1998 (top) and 2014 (below). Observations above 60 $\mu g/m^3$ in the data have been capped at 60 $\mu g/m^3$ in the figure for visualization.

**Table 2.** Descriptive statistics: exposures for the neonatal-mortality sample

| Variable, statistic | Mean | SD |
|---|---|---|
| Early-life all source $PM_{2.5}$ level | 24.43 | 14.56 |
| Early-life $PM_{2.5}$ level without dust and sea-salt | 10.88 | 7.47 |
| Early-life dust and sea-salt level | 13.55 | 14.80 |
| Log (1 + early-life all source $PM_{2.5}$ level) | 3.05 | 0.65 |
| Log (1 + early-life $PM_{2.5}$ level without dust and sea-salt) | 2.29 | 0.63 |
| Log (1 + early-life dust and sea-salt level) | 2.05 | 1.20 |
| Number of observations | | 529 874 |

The sample for neonatal mortality is different from the post-neonatal infant-mortality and infant-mortality samples. Descriptive statistics for exposure for the post-neonatal infant-mortality sample and the infant-mortality sample are shown in an online appendix, available as Supplementary data at *IJE* online.

educational attainment and having access to a flush-toilet facility are associated with lower odds of mortality, whereas shorter birth intervals, being a multiple birth and maternal use of tobacco are associated with higher odds of child mortality. The estimated coefficients on maternal age depict a U-shaped association with mortality, with ages in the interval 30–34 being associated with the lowest odds. Use of solid cooking fuel is not associated with mortality, possibly due to the correlation between the use of solid cooking fuel and ambient $PM_{2.5}$ in low- and middle-income countries (see Supplementary Tables 6–8, available as Supplementary data at *IJE* online).[69]

When disaggregating $PM_{2.5}$ exposure by source type, i.e. from dust and sea-salt vs from other, mainly anthropogenic, sources, as shown in Table 5, we find that the odds of neonatal mortality strongly increase with increase in exposure to early-life $PM_{2.5}$ without dust and sea-salt (OR: 1.22; 95% CI: 1.11, 1.35). In particular, our estimated OR of 1.22 on the log $PM_{2.5}$ exposure variable implies that a doubling in the level of $PM_{2.5}$ without dust and sea-salt is associated with a 14.9% increase in the odds of neonatal mortality. While we do not find any relationship between ambient $PM_{2.5}$ without dust and sea-salt and post-neonatal infant mortality, we also find a strong association between exposure to early-life $PM_{2.5}$ without dust and sea-salt and infant mortality, which includes neonatal deaths (OR: 1.13; 95% CI: 1.04, 1.23); here, a doubling of the level of $PM_{2.5}$ without dust and sea-salt is associated with a 8.8% increase in the odds of mortality before age 1. In contrast,

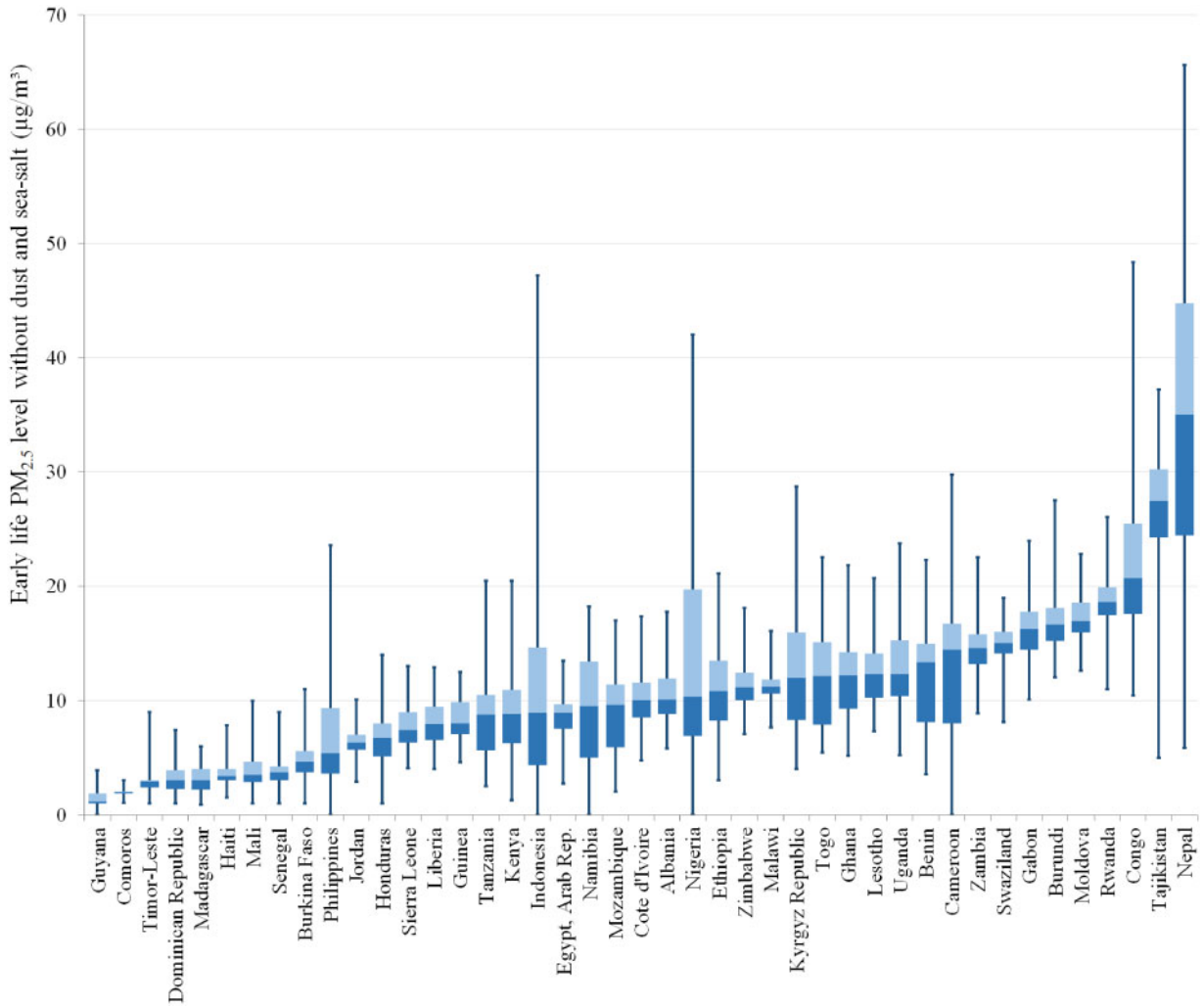


**Figure 3.** Box plot of early-life $PM_{2.5}$ level without dust and sea-salt by country for the neonatal-mortality sample (in $\mu g/m^3$). The data across countries are not strictly comparable, as different countries may have been surveyed in different years.

we do not find strong associations between exposure to $PM_{2.5}$ from dust and sea-salt and either overall or age-specific mortality.

Based on the model results in column 1 of Tables 4 and 5, we plot the ORs of neonatal mortality together with their 95% CIs at different levels of early-life exposure to $PM_{2.5}$, both overall as well as with and without dust and sea-salt (Figures 4 and 5). These figures confirm our findings from the tables, where we find strong associations between exposure to $PM_{2.5}$ without dust and sea-salt and neonatal mortality but only a weak association between neonatal mortality and either early-life exposure to overall $PM_{2.5}$ or exposure to $PM_{2.5}$ from only dust and sea-salt.

We also examine the robustness of our results to different specifications of the functional form of the exposure effect on mortality. Instead of a log of $PM_{2.5}$ exposure, we estimate models with a linear response and with a spline with three knots. Findings from these analyses are presented in Supplementary Tables 13 and 14, available as Supplementary data at *IJE* online. The corresponding predicted probabilities of neonatal mortality that are derived from these estimates across levels of exposure to $PM_{2.5}$ without dust and sea-salt are plotted in Figure 6; these are based on the same models as Figure 5 but with different functional forms. We also show the 95% CI for the spline with three knots. The predicted probabilities of neonatal mortality under our preferred logarithmic specification all lie within the 95% CI for the spline with three knots. The linear model, however, is outside this CI for a part of the range.

We conduct several robustness checks to confirm our main findings; tabular results from our robustness checks are presented in the Supplementary Materials section, available as Supplementary data at *IJE* online. First, we limit the sample to children for whom we are certain that their place of birth matches the place of interview, which is not recorded in all DHS surveys (column 1 of Supplementary Tables 10–12, available as Supplementary

**Table 3.** Descriptive statistics: covariates for the neonatal-mortality sample

| Variable, statistic | Mean | SD | Number of cases |
|---|---|---|---|
| Birth order (number) | 3.64 | 2.40 | |
| Household uses solid cooking fuel | | | |
| No | 0.18 | | 93 382 |
| Yes | 0.82 | | 436 492 |
| Mother uses tobacco | | | |
| No | 0.96 | | 511 123 |
| Yes | 0.04 | | 18 751 |
| Short birth interval | | | |
| No | 0.57 | | 299 899 |
| Birth interval <18 months | 0.06 | | 30 432 |
| Birth interval 18–35 months | 0.38 | | 199 543 |
| Multiple birth | | | |
| No | 0.97 | | 513 376 |
| Yes | 0.03 | | 16 498 |
| Female | | | |
| No | 0.51 | | 269 179 |
| Yes | 0.49 | | 260 695 |
| First child | | | |
| No | 0.79 | | 419 662 |
| Yes | 0.21 | | 110 212 |
| Age band of mother (in years) | | | |
| 15–19 | 0.05 | | 25 253 |
| 20–24 | 0.22 | | 116 138 |
| 25–29 | 0.28 | | 149 224 |
| 30–34 | 0.21 | | 112 207 |
| 35–39 | 0.15 | | 77 946 |
| 40–44 | 0.07 | | 37 002 |
| 45–49 | 0.02 | | 12 104 |
| Education level of mother | | | |
| None | 0.38 | | 203 756 |
| Primary | 0.34 | | 181 335 |
| Secondary | 0.23 | | 120 507 |
| Higher | 0.05 | | 24 276 |
| Education level of mother's partner | | | |
| None | 0.32 | | 169 323 |
| Primary | 0.32 | | 168 513 |
| Secondary | 0.29 | | 154 810 |
| Higher | 0.07 | | 37 228 |
| Household has access to flush toilet | | | |
| No | 0.84 | | 442 777 |
| Yes | 0.16 | | 87 097 |
| Household has access to piped water | | | |
| No | 0.69 | | 368 011 |
| Yes | 0.31 | | 161 863 |
| Urban residence | | | |
| No | 0.71 | | 375 082 |
| Yes | 0.29 | | 154 792 |
| Wealth quintile of household | | | |
| Poorest | 0.26 | | 136 738 |
| Poor | 0.22 | | 117 026 |
| Middle | 0.20 | | 106 898 |
| Rich | 0.18 | | 92 894 |
| Richest | 0.14 | | 76 318 |
| Number of observations | | | 529 874 |

The sample for neonatal mortality is different from the post-neonatal infant-mortality and infant-mortality samples. Descriptive statistics for the covariates for the post-neonatal infant-mortality sample and the infant-mortality sample are shown in an online appendix, available as Supplementary data at *IJE* online. For continuous variables, the mean and standard deviation are presented. For discrete (categorical, binary) variables, the mean (proportion of cases) as well as the number of cases for that category are presented.

data at *IJE* online). We find that our results for neonatal mortality are robust. In running models that exclude country-specific trends in the birth year (column 2 of Supplementary Tables 10–12, available as Supplementary data at *IJE* online) and subnational region fixed effects (column 3 of Supplementary Tables 10–12, available as Supplementary data at *IJE* online), we similarly find our results for neonatal mortality to be robust to these sensitivity checks.

Finally, we estimate the relationship between exposure without dust and sea-salt and neonatal mortality for each country in our sample using the main, fully adjusted log specification and then conduct a meta-analysis to compare the country-specific results against the summary estimate of the meta-analysis. The results from the country-specific regressions and the meta-analysis are presented in a Forest plot (Figure 7). Findings from this analysis suggest that the estimates of the association from individual countries vary modestly. This is confirmed by the I-squared statistic of 23.3% ($p = 0.09$), which suggests modest but not substantial heterogeneity across the country-specific coefficients. Given the large CIs around the countries, the results from the meta-analysis suggest that individual country-level studies using DHS data may not have sufficient power to be able to detect a relationship due to the small sample size within each country dataset.

## Discussion

In this study, we combine child-level health data from 43 low- and middle-income countries with high-resolution spatial data on ambient fine particulate matter ($PM_{2.5}$) to analyse the relationship between exposure to ambient air pollution and child mortality. We find early-life exposure to overall $PM_{2.5}$ does not have a strong association with infant mortality. When we disaggregate $PM_{2.5}$ exposure by source, however, we find a strong association between

**Table 4.** The association of log of early-life overall $PM_{2.5}$ level on neonatal, post-neonatal infant and infant mortality

| | (1) Neonatal death | (2) Post-neonatal infant death | (3) Infant death |
|---|---|---|---|
| Log(1 + neonatal early-life overall $PM_{2.5}$ level) | 1.08 [0.95, 1.23] | – | – |
| Log(1 + infant early-life overall $PM_{2.5}$ level) | – | 0.89 [0.76, 1.05] | 0.99 [0.89, 1.12] |
| Household uses solid cooking fuel (1 = yes) | 0.99 [0.91, 1.08] | 1.01 [0.90, 1.13] | 1.02 [0.94, 1.10] |
| Mother uses tobacco (1 = yes) | 1.21*** [1.10, 1.32] | 1.25*** [1.14, 1.38] | 1.24*** [1.16, 1.34] |
| Birth interval: <18 months | 3.38*** [3.17, 3.61] | 2.75*** [2.56, 2.96] | 3.24*** [3.07, 3.41] |
| Birth interval: 18–35 months | 1.40*** [1.34, 1.46] | 1.51*** [1.44, 1.58] | 1.52*** [1.47, 1.58] |
| Multiple birth: (1 = yes) | 6.82*** [6.42, 7.24] | 3.20*** [2.95, 3.48] | 5.19*** [4.92, 5.48] |
| Female (1 = yes) | 0.75*** [0.73, 0.78] | 0.92*** [0.89, 0.95] | 0.83*** [0.81, 0.85] |
| First child (1 = yes) | 2.35*** [2.20, 2.50] | 1.49*** [1.39, 1.60] | 1.94*** [1.85, 2.04] |
| Age of mother: 15–19 years | 1.17*** [1.09, 1.27] | 1.34*** [1.21, 1.48] | 1.29*** [1.20, 1.38] |
| Age of mother: 25–29 years | 0.86*** [0.82, 0.91] | 0.92** [0.87, 0.98] | 0.88*** [0.84, 0.92] |
| Age of mother: 30–34 years | 0.91** [0.85, 0.98] | 0.85*** [0.79, 0.91] | 0.87*** [0.82, 0.92] |
| Age of mother: 35–39 years | 1.04 [0.96, 1.13] | 0.88** [0.80, 0.96] | 0.93* [0.87, 0.99] |
| Age of mother: 40–44 years | 1.20*** [1.08, 1.33] | 0.83** [0.74, 0.93] | 0.98 [0.91, 1.07] |
| Age of mother: 45–49 years | 1.53*** [1.34, 1.75] | 0.89 [0.77, 1.04] | 1.14* [1.02, 1.27] |
| Education level of mother: primary | 1.01 [0.96, 1.07] | 0.96 [0.90, 1.01] | 0.99 [0.95, 1.03] |
| Education level of mother: secondary | 0.95 [0.89, 1.02] | 0.86*** [0.79, 0.93] | 0.89*** [0.84, 0.95] |
| Education level of mother: higher | 0.88 [0.76, 1.01] | 0.61*** [0.50, 0.74] | 0.74*** [0.65, 0.83] |
| Education level of mother's partner: primary | 0.99 [0.94, 1.05] | 0.93* [0.88, 0.99] | 0.96* [0.92, 1.00] |
| Education level of mother's partner: secondary | 0.92** [0.87, 0.98] | 0.90** [0.84, 0.96] | 0.90*** [0.85, 0.94] |
| Education level of mother's partner: higher | 0.80*** [0.72, 0.88] | 0.83** [0.73, 0.94] | 0.80*** [0.73, 0.87] |
| Household has access to flush toilet (1 = yes) | 0.92 [0.85, 1.00] | 0.85** [0.77, 0.94] | 0.89** [0.84, 0.96] |
| Household has access to piped water (1 = yes) | 0.98 [0.94, 1.03] | 0.97 [0.91, 1.03] | 0.97 [0.93, 1.01] |
| Urban residence (1 = yes) | 0.98 [0.93, 1.04] | 1.03 [0.97, 1.10] | 1.02 [0.97, 1.06] |
| Wealth quintile: poor | 1.00 [0.95, 1.06] | 0.98 [0.93, 1.04] | 0.98 [0.94, 1.02] |
| Wealth quintile: middle | 1.01 [0.96, 1.07] | 0.95 [0.90, 1.01] | 0.97 [0.93, 1.02] |
| Wealth quintile: rich | 1.02 [0.96, 1.09] | 0.90** [0.84, 0.97] | 0.95* [0.90, 1.00] |
| Wealth quintile: richest | 0.96 [0.88, 1.05] | 0.78*** [0.71, 0.86] | 0.86*** [0.80, 0.93] |
| Observations | 528 889 | 411 164 | 425 100 |

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

The unit of observation is the child. Odds ratios are presented with 95% confidence intervals in the square brackets. All regressions include country-specific time trends with survey and subnational region fixed effects, and standard errors are clustered at the primary sampling unit (DHS cluster) level.

exposure excluding dust and sea-salt and infant mortality, with almost all the burden falling on mortality in the neonatal period. Our findings indicate that exposure to particulates due to human activity may be particularly harmful to child health and development. Our results suggest that the mechanism responsible for the association between ambient air-pollution exposure and neonatal mortality may depend on the composition and toxicity of particulate matter.

Two other studies are similar to our study in that they examine the association of ambient $PM_{2.5}$ on infant or child mortality using evidence from Africa.[23,24] The first focuses on the association of national average level of exposure with national average under-5 mortality rate. We improve on this by using more detailed spatial matching of exposure and infant mortality. The study closest to ours uses the same data and a very similar method, matching individual infant deaths from the DHS to cluster-level $PM_{2.5}$ exposure. However, several key differences exist. First, the sample for that study is limited to countries in Africa, whereas we include all countries for which GPS data are available in the DHS and spatial matching can be conducted. Second, they focus on the relationship between infant mortality with overall $PM_{2.5}$ exposure, whereas we focus on the disaggregated relationship by type of $PM_{2.5}$. We find a much stronger association for $PM_{2.5}$ without dust and sea-salt with mortality than for $PM_{2.5}$ due to dust and sea-salt. Third, whereas they focus on the association of $PM_{2.5}$ exposure with infant mortality, we also disaggregate the time of death between the neonatal and post-neonatal periods. We find that, in our sample, the association of infant mortality with ambient $PM_{2.5}$ exposure is driven primarily by deaths during the neonatal period rather than during the post-neonatal period. Whereas they estimate that an increase of 10 $\mu g/m^3$ in overall $PM_{2.5}$ level is associated with a 9% (95% CI: 4, 14%) increase in

**Table 5.** The association of log of early-life $PM_{2.5}$ level, by source, on neonatal, post-neonatal infant, and infant mortality

| | (1) Neonatal death | (2) Post-neonatal infant death | (3) Infant death |
|---|---|---|---|
| Log (1 + neonatal early-life $PM_{2.5}$ level without dust and sea-salt) | 1.22*** [1.11, 1.35] | – | – |
| Log (1 + neonatal early-life dust and sea-salt level) | 0.98 [0.89, 1.08] | – | – |
| Log (1 + infant early-life $PM_{2.5}$ level without dust and sea-salt) | – | 1.07 [0.96, 1.20] | 1.13** [1.04, 1.23] |
| Log (1 + infant early-life dust and sea-salt level) | – | 0.87* [0.76, 0.99] | 0.96 [0.87, 1.05] |
| Household uses solid cooking fuel | 0.99 [0.91, 1.08] | 1.01 [0.90, 1.13] | 1.02 [0.94, 1.10] |
| Mother uses tobacco | 1.21*** [1.10, 1.33] | 1.26*** [1.14, 1.38] | 1.25*** [1.16, 1.34] |
| Birth interval, < 18 months | 3.39*** [3.18, 3.61] | 2.75*** [2.56, 2.96] | 3.24*** [3.07, 3.42] |
| Birth interval, 18–35 months | 1.40*** [1.34, 1.47] | 1.51*** [1.44, 1.58] | 1.52*** [1.47, 1.58] |
| Multiple birth | 6.81*** [6.41, 7.24] | 3.20*** [2.95, 3.48] | 5.19*** [4.92, 5.48] |
| Female | 0.75*** [0.73, 0.78] | 0.92*** [0.89, 0.95] | 0.83*** [0.81, 0.85] |
| First child | 2.35*** [2.21, 2.50] | 1.49*** [1.39, 1.60] | 1.94*** [1.85, 2.04] |
| Age of mother, 15–19 years | 1.17*** [1.09, 1.27] | 1.34*** [1.21, 1.48] | 1.29*** [1.20, 1.38] |
| Age of mother, 25–29 years | 0.86*** [0.82, 0.91] | 0.92** [0.87, 0.98] | 0.88*** [0.84, 0.92] |
| Age of mother, 30–34 years | 0.91** [0.85, 0.98] | 0.85*** [0.79, 0.91] | 0.87*** [0.82, 0.92] |
| Age of mother, 35–39 years | 1.04 [0.96, 1.13] | 0.88** [0.80, 0.96] | 0.93* [0.87, 0.99] |
| Age of mother, 40–44 years | 1.20*** [1.08, 1.32] | 0.83** [0.74, 0.93] | 0.98 [0.90, 1.07] |
| Age of mother, 45–49 years | 1.53*** [1.33, 1.75] | 0.89 [0.77, 1.04] | 1.14* [1.02, 1.27] |
| Education level of mother, primary | 1.01 [0.96, 1.06] | 0.95 [0.90, 1.01] | 0.98 [0.94, 1.02] |
| Education level of mother, secondary | 0.95 [0.88, 1.02] | 0.85*** [0.79, 0.92] | 0.89*** [0.84, 0.94] |
| Education level of mother, higher | 0.88 [0.76, 1.01] | 0.61*** [0.50, 0.74] | 0.73*** [0.65, 0.83] |
| Education level of mother's partner, primary | 0.99 [0.94, 1.04] | 0.93* [0.88, 0.98] | 0.95* [0.91, 0.99] |
| Education level of mother's partner, secondary | 0.92** [0.86, 0.97] | 0.89*** [0.83, 0.95] | 0.89*** [0.85, 0.94] |
| Education level of mother's partner, higher | 0.79*** [0.71, 0.88] | 0.83** [0.73, 0.93] | 0.79*** [0.73, 0.87] |
| Household has access to flush toilet | 0.92 [0.85, 1.00] | 0.85** [0.77, 0.94] | 0.90** [0.84, 0.96] |
| Household has access to piped water | 0.99 [0.94, 1.04] | 0.97 [0.92, 1.03] | 0.97 [0.93, 1.01] |
| Urban residence | 0.98 [0.93, 1.04] | 1.03 [0.97, 1.10] | 1.02 [0.97, 1.06] |
| Wealth quintile, Poor | 1.00 [0.95, 1.06] | 0.98 [0.93, 1.04] | 0.98 [0.94, 1.02] |
| Wealth quintile, Middle | 1.01 [0.95, 1.07] | 0.95 [0.89, 1.01] | 0.97 [0.93, 1.01] |
| Wealth quintile, Rich | 1.02 [0.96, 1.09] | 0.90** [0.84, 0.96] | 0.95* [0.90, 1.00] |
| Wealth quintile, Richest | 0.95 [0.87, 1.04] | 0.78*** [0.70, 0.86] | 0.86*** [0.80, 0.92] |
| Observations | 528 889 | 411 164 | 425 100 |

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

The unit of observation is the child. Odds ratios are presented with 95% confidence intervals in the square brackets. All regressions include country-specific time trends with survey and subnational region fixed effects, and standard errors are clustered at the primary sampling unit (DHS cluster) level.

infant mortality, we find almost no association between overall $PM_{2.5}$ level and infant mortality (OR: 1.00, 95% CI: 0.99, 1.00) for the linear specification (Supplementary Table 9, column 3, available as Supplementary data at *IJE* online). However, we use somewhat different covariates from that study and control for subnational region fixed effects, survey fixed effects and country-specific birth-year trend whereas they control for cluster fixed effects, birth-year fixed effects and country-month effects.

There are several important limitations to this study and its findings. Our study does not identify the biological mechanism through which exposure to $PM_{2.5}$ may affect child mortality. Exposure to pollution after birth is likely to lead to inflammation and oxidative stress. However, there may also be an *in utero* effect of maternal exposure to air pollution due to particles that enter the mother's bloodstream and interfere with uterine growth during pregnancy due to decreased exchange of oxygen and nutrients across the placenta.[70]

The issue of the key period of exposure, *in utero* or shortly after birth, might be addressed if we had greater temporal specificity of exposure, rather than our annual average exposure levels. Since our annual $PM_{2.5}$ concentration data are not measured at a sufficiently high frequency (e.g. daily or monthly) for us to identify the extent to which the association between air pollution and mortality is driven by *in utero* exposure or by post-partum exposure, we have therefore defined early-life exposure to ambient PM2.5 to cover the *in utero* and first-month post-partum periods for neonatal mortality and the *in utero* and first-year post-partum periods for infant mortality. We are unable to specifically disentangle the *in utero* impact of exposure to $PM_{2.5}$ from the post-partum impact.

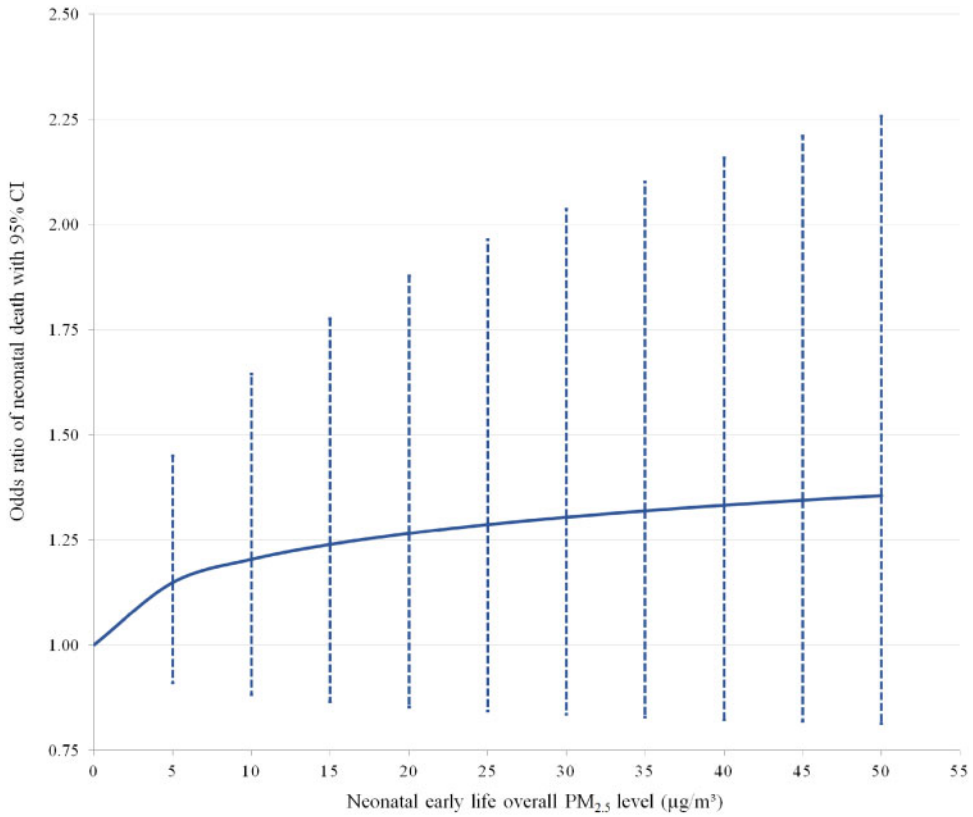


**Figure 4.** Odds ratios and corresponding 95% confidence intervals of neonatal death for early-life overall $PM_{2.5}$ level based on a logarithmic concentration response curve. The graph was created based on the regression result in Table 4, column 1.

Our exposure assessment is subject to misclassification as we lack information on the exact DHS cluster location and also our averaging of the $PM_{2.5}$ concentration level over the displacement radius of the DHS cluster excludes some grid squares in case of a partial overlap. However, our data exhibit high spatial homogeneity in the $PM_{2.5}$ concentration level over small distances and the point estimate of the $PM_{2.5}$ concentration level at the reported DHS cluster location has more than 99% correlation with the average $PM_{2.5}$ concentration level over the DHS cluster displacement radius. Our data on $PM_{2.5}$ exposure by type are based on the modelling of sources of pollution and how it disperses rather than direct observation of the type of pollution. It may therefore be less reliable than the overall $PM_{2.5}$ data.

Also important to the discussion of the relationship between exposure to $PM_{2.5}$ and mortality is the role of gestational age and pre-term birth. Due to limitations in our dataset, we are unable to measure a child's gestational age and, therefore, assume that each child in our sample is carried to term, which is reflected in our calculation of a child's level of *in utero* exposure to $PM_{2.5}$. More generally, however, it may be that gestational age and pre-term birth mediate the relationship between exposure to ambient air pollution and child-mortality outcomes. Given the literature on (i) the relationship between air pollution and gestational age and (ii) the relationship between gestational age and child mortality, it would then seem that controlling for gestational age, as a likely mediator, would lead to bias in our analysis.

Our key explanatory variable, early-life exposure to ambient $PM_{2.5}$, is subject to measurement error due to the paucity of ground-based air-quality monitoring data in low- and middle-income countries. Estimates that are based on satellite data are calibrated to match these ground-based measures, but this may not work well in regions that lack dense monitoring networks. In addition, diurnal and seasonal variability in the $PM_{2.5}$ concentrations may also influence child-health outcomes, but we do not have this information in our dataset.

Furthermore, we are unable to control for the more detailed composition of ambient $PM_{2.5}$ or the level of other air pollutants, which may bias our estimates of the mortality effects of $PM_{2.5}$.[71] More specifically, we are unable to account for the level of ultrafine particles from traffic pollution and from other pollutants such as $O_3$ and $NO_2$, which together represent a range of pollutants for which we do not have global data and which are also likely to be correlated with $PM_{2.5}$. As a result, our estimates may be

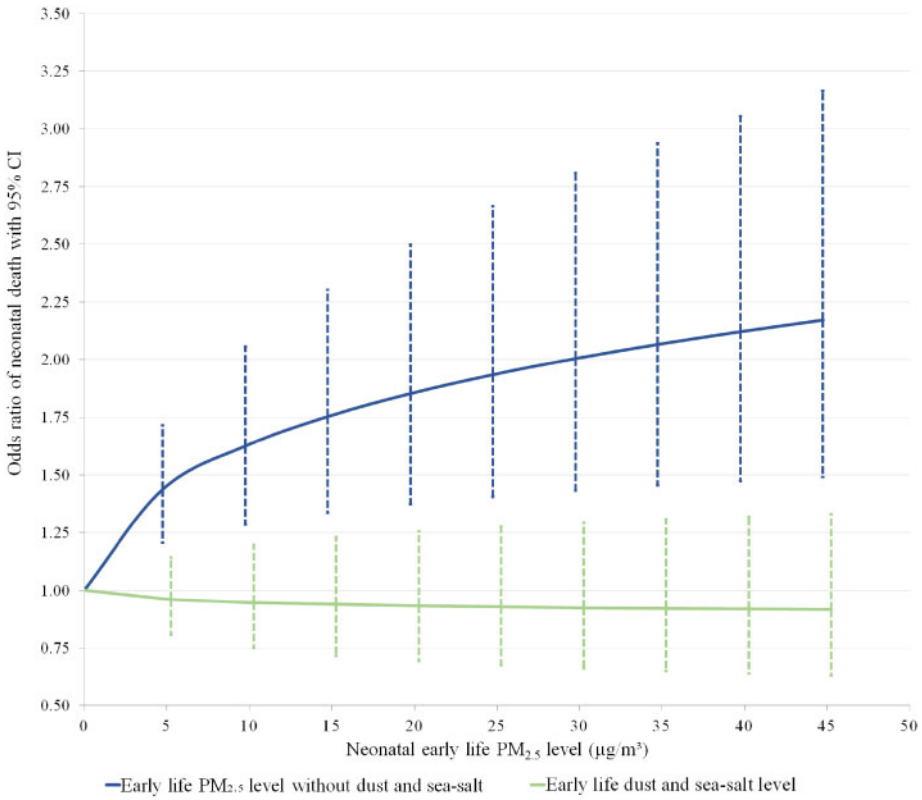


**Figure 5.** Odds ratios and corresponding 95% confidence intervals of neonatal death for exposure to early-life $PM_{2.5}$ level without dust and sea-salt and exposure to early-life dust and sea-salt based on a logarithmic concentration response curve. The graph was created based on the regression result in Table 5, column 1.

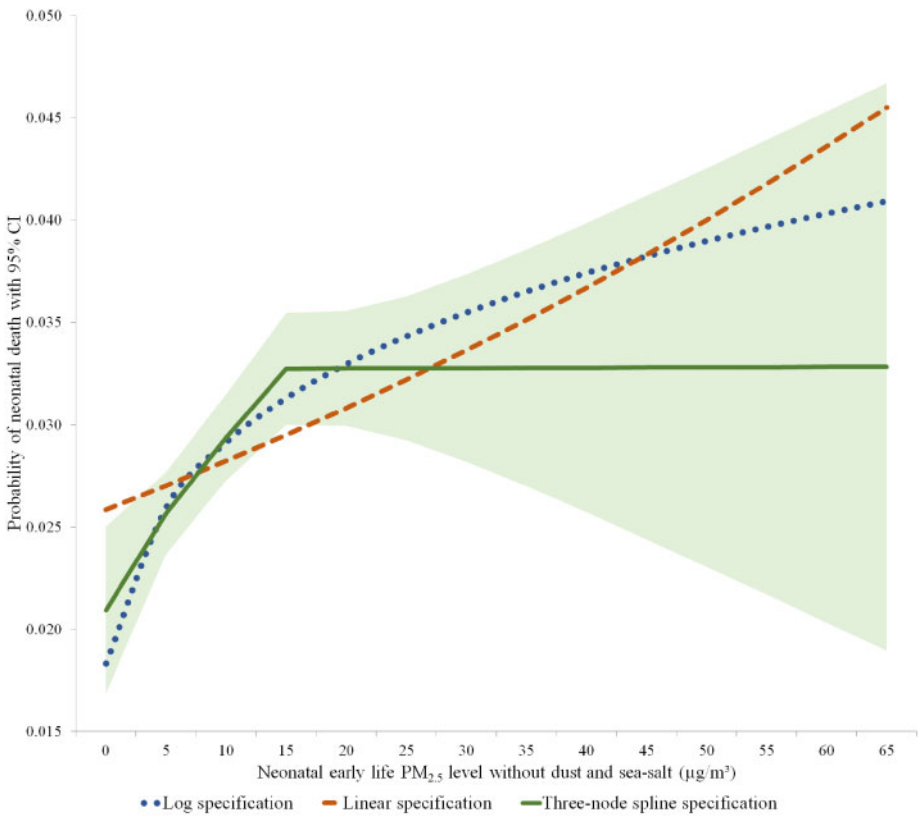


**Figure 6.** Predicted probability of neonatal death across alternative model specifications.

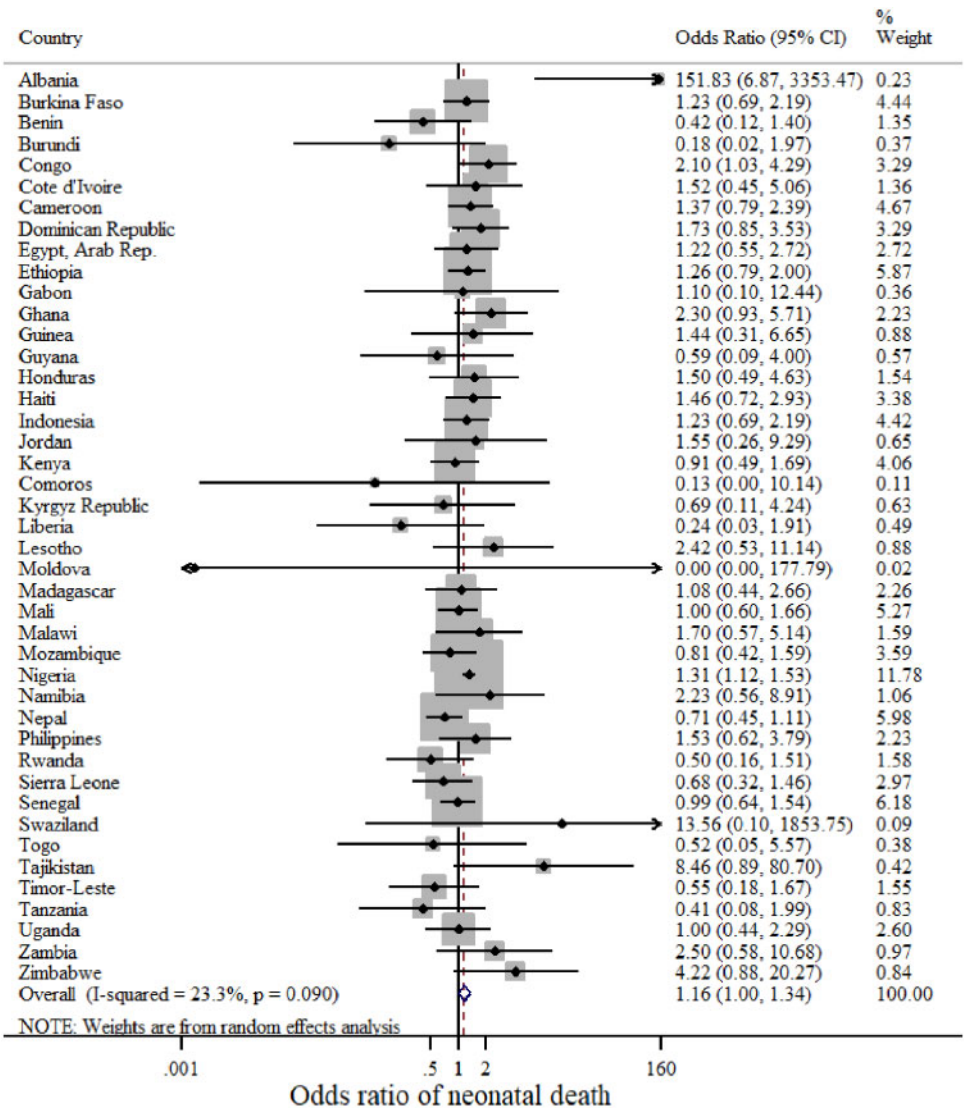


**Figure 7.** Country-specific analysis of the association of early-life $PM_{2.5}$ level without dust and sea-salt with neonatal death.

capturing a more general association of pollution with mortality rather than a specific association from $PM_{2.5}$.

Our global sample pools together data on all births over the 5 years preceding the survey date for women of reproductive age (ages 15–49) who lived in the sampled households. Since these data on births are reported by mothers, however, we may be missing data on children in the household whose mothers have died or who were not present at the time of the survey. Finally, our findings may suffer from residual confounding from omitted variables that are correlated with $PM_{2.5}$ exposure as well as child mortality.

## Conclusion

Children in our sample experienced an average $PM_{2.5}$ level nearly two and a half times the WHO-recommended guideline of 10 $\mu g/m^3$. About half of this exposure was due to dust and sea-salt whereas half was from other, mainly anthropogenic, sources. Our results indicate that children with higher exposure to ambient $PM_{2.5}$ without dust and sea-salt face substantially higher odds of neonatal mortality, even at levels well below the WHO-recommended guideline of 10 $\mu g/m^3$. Policies that aim to reduce ambient air pollution in low- and middle-income countries could contribute significantly to reducing neonatal mortality from the level of 28 per 1000 births observed in our sample and meeting the Sustainable Development Goal of lowering neonatal mortality below 12 per 1000 children by 2030.

## Supplementary data

Supplementary data are available at *IJE* online.

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**Conflict of interest:** We have read and understood the *International Journal of Epidemiology*'s policy on declaration of interests and declare that we have no competing interests.

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