# In-Utero Exposure to Ambient Fine Particulate Air Pollution and Child 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 ambient fine particulate air pollution ($PM_{2.5}$). The effect of $PM_{2.5}$ on mortality is usually modelled using concentration response curves extrapolated from studies conducted in settings with low ambient air pollution. We directly estimate the association between child mortality and exposure to $PM_{2.5}$, both overall and by $PM_{2.5}$ source.

**Methods:** We pool data of over 500 000 children 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 in-utero exposure to ambient $PM_{2.5}$ using high resolution satellite data that is matched to the child's place of residence. We estimate the association between in-utero $PM_{2.5}$ exposure and the odds of child mortality, adjusting for child-level, parent-level, and household-level characteristics.

**Results:** We find that in-utero exposure to overall $PM_{2.5}$ above 12.3 µg/m³ is associated with a higher odds of child mortality, particularly neonatal mortality, relative to low in-utero exposure to overall $PM_{2.5}$. Exposure to dust and sea-salt has little effect, while exposure to other (mainly anthropogenic, carbonaceous) particulates is associated with increased odds of neonatal mortality even at levels as low as 3.4 µg/m, with exposure above the median level raising the odds of neonatal mortality by over one third.

**Conclusion:** While our results are consistent with the current World Health Organization guideline of limiting the overall ambient $PM_{2.5}$ level at less than 10 µg/m³, they suggest the need for a much lower limit for harmful, carbonaceous $PM_{2.5}$.

**Keywords:** 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 in-utero exposure to ambient fine particulate matter ($PM_{2.5}$) is associated with higher odds of child mortality in low- and middle-income countries.
- The association between in-utero exposure and mortality is strongest in the neonatal period.
- The association between $PM_{2.5}$ exposure in the form of dust and sea-salt, which make up over half of all fine particulates, and child mortality is weak; however, other types of fine particulates, which are mainly due to human activity, may have a large impact on mortality even at a low level.
- Reducing ambient $PM_{2.5}$ could contribute substantially to achieving the Sustainable Development Goal of lowering neonatal mortality 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 to be around 4 $\mu g/m^3$.

## 1. 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). Given the evidence of the relationship between exposure to ambient air pollution and child mortality (2-8), high and rising ambient air pollution may be a key factor for the continuing high rate of neonatal mortality in low- and middle-income countries (9).

The dominant approach in the literature is to model mortality due to ambient air pollution using concentration response curves that are estimated from studies in high-income countries (10-12). This approach, however, is open to question since ambient air pollution in low- and middle-income countries is generally higher than in high-income countries and has dissimilar sources and toxicity (13-16).

Two studies have directly estimated the effect of exposure to ambient air pollution at the local level in middle-income settings, one in Mexico City and one in São Paulo, Brazil (17, 18). Moreover, the average national ambient air pollution level has been found to be related to national child mortality rates in Africa (19). We improve on this evidence by using a large international sample of individual-level child data, which allows us to match child mortality data to the local ambient $PM_{2.5}$ pollution level when the children were in-utero.

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 birth weight, and child stunting (6, 11, 20-22). The Nashville Air Pollution study was possibly the first to suggest that

chronic exposure to ambient air pollution was related to neonatal death (23). Similar findings have been reported in many studies in developed countries (4, 6, 24-29).

The biological mechanism for the effect of in-utero exposure to air pollution on child health is thought to be through Polycyclic Aromatic Hydrocarbons (PAH) that are found in particulate matter. PAH particles enter the mother's bloodstream and accumulate in the nucleus of cells, resulting in anti-estrogenic activity that interferes with uterine growth during pregnancy as well as DNA damage that results in a decreased exchange of oxygen and nutrients with the placenta (30). If the mechanism depends on the effect of PAH on health, then it is likely that particulates from different sources may affect child health differently.

In this study, we pool data on children aged 0 to 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 fine particulate matter ($PM_{2.5}$) to analyse the relationship between in-utero exposure to ambient air pollution and child mortality. We focus on $PM_{2.5}$ because its effect on child health has been found to be more pronounced than other sizes of particulates (25). However, the composition and sources of the particulates may matter in addition to their sizes (31, 32) and we distinguish dust and sea-salt, which make up over half of total exposure, from other types of particulates.

## 2. Methods

**Study population.** We obtain data on children from the Demographic and Health Surveys (DHS), which are nationally representative household surveys (33). The DHS employs a two-

stage randomized cluster sampling design (34). Information is recorded on all births in the previous five years, including whether the child died and, if so, the age of death. In many DHS surveys, the location of each cluster of sampled DHS households is recorded in the dataset, although a small amount of noise is added to the reported coordinates in order to protect household privacy (35). 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, 69 surveys are used for the main analysis based on availability of data on exposures and covariates (see Supplemental Material: Table S2 and Figure S1). After excluding observations with missing data, our resulting sample consists of 534 476 children born in 34 450 clusters across 43 countries (see Supplemental Material: Table S3).

**Outcomes.** We estimate the association between $PM_{2.5}$ exposure and the probability of mortality for all children in the sample. We find that 72 per 1000 children born in the last five years had died by the time of the survey. We also examine the timing of the effect by conducting disaggregated analyses on neonatal mortality, post-neonatal infant mortality, and post-infant child mortality. The age-specific mortality rates are reported in Table 1.

[Insert Table 1 here]

**Exposure.** The key explanatory variable in this study is the total level of ambient $PM_{2.5}$ (measured in $\mu g/m^3$) that the child was exposed to in-utero. We also estimated the impact of post-birth exposure on air pollution but do not find any significant relationship. We use data on annual average ambient $PM_{2.5}$ concentrations, estimated through a triangulation of multiple

satellite information sources, simulation results, and available ground-based monitoring data; in particular, the satellite data has been bias-corrected to match the available ground-based monitoring data (15, 36). The data covers the period 1998 to 2014, at a resolution of 0.01° x 0.01° (approximately, 1 km x 1 km). Similar data have been used by the Global Burden of Diseases (GBD) studies to model attributable disability-adjusted life years (DALYs) to ambient air pollution (11, 37). The geographical distribution of annual average ambient $PM_{2.5}$ concentrations in 1998 and 2014 shows that the highest concentrations occur in desert regions, such as the Sahara, the Arabian Gobi, and the Thar deserts, due to dust (Figure 1).

Sources of fine particulate matter include 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 (14, 38). These sources produce different types of particulates, which 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 contain a high proportion of polycyclic aromatic hydrocarbons (PAH). Figure 2 shows the distribution of $PM_{2.5}$ concentrations excluding dust and sea-salt in 1998 and 2014. We observe high concentrations in the Eastern United States, 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.

[Insert Figures 1 and 2 here]

We obtain the geographically specific $PM_{2.5}$ level for each child in our sample by matching the GPS information in the DHS with the annual average ambient $PM_{2.5}$ concentrations in a small spatial zone surrounding the cluster location during the time the child was in-utero. This procedure is conducted using QGIS software (version 2.14.21-Essen; Open Source Geospatial Foundation Project). The creation of the spatial zone corrects for the noise that is added to the DHS GPS location data within the exposure variable (22). We calculate the in-utero $PM_{2.5}$ level as the $PM_{2.5}$ level in the nine months before a child's birth; for births whose intervals spanned two calendar years, we use a weighted average of the annual $PM_{2.5}$ levels over these two years.

The dust and sea-salt level that the child is exposed to in-utero is calculated by subtracting the in-utero $PM_{2.5}$ level without dust and sea-salt from the in-utero overall $PM_{2.5}$ level. Due to measurement error in these two variables, in-utero dust and sea-salt levels were found to be below zero for some children in our final sample; these exposure levels were set to zero. Later, we confirm that this change does not affect our main findings significantly (see Supplemental Material: Tables S4-7).

The mean in-utero exposure to overall $PM_{2.5}$ in the sample is 24.44 $\mu g/m^3$, and nearly all children in our sample are exposed to $PM_{2.5}$ levels that exceed the World Health Organization (WHO) guideline of 10 $\mu g/m^3$ (Table 2). Further, the mean in-utero exposure to $PM_{2.5}$ without dust and sea-salt was 10.75 $\mu g/m^3$. Figure 3 shows box plots of exposure of this type in our sample by country. 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 (39, 40). Some Sub-Saharan

African countries, such as the Democratic Republic of Congo, report a high level of ambient air pollution due to biomass burning (36).

[Insert Table 2 here]

[Insert Figure 3 here]

Recent studies have calibrated the relationship between ambient $PM_{2.5}$ level and under-5 mortality by either using a logarithmic concentration response curve or an integrated exposure response curve (25, 41). For our main model, where we pool data across all surveys in our sample, we do not impose any functional form; instead, we estimate the association between exposure and mortality over eight quantiles of exposure to allow the data to determine the dose response function.

Our regressions control for child-, parental-, and household-level characteristics (42). 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. For post-infant child mortality, we also include the age (or potential age, if dead) of the child to control for differential durations of exposure to mortality risk. Parental characteristics include the age of the mother, the education level of the mother, whether the mother used tobacco (43, 44), and the education level of the mother's partner. Household 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 (45), 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 (46-50). Descriptive statistics for these covariates are presented in Table 3.

[Insert Table 3 here]

**Statistical analysis.** We estimate the relationship between ambient $PM_{2.5}$ and child mortality using multivariate logistic regression. Estimates are presented as odds ratios with standard errors clustered at the DHS cluster level to account for a sampling methodology that allows for correlations between outcomes for children within a cluster. In addition, our regressions include sub-national region fixed effects and survey dummies. Finally, we include a trend in the country-specific birth year (51, 52). All analyses are conducted using STATA software (version STATA/SE 14.1; StataCorp LP, College Station, Texas, USA).

## 3. Results

We first estimate the relationship between in-utero exposure to overall ambient $PM_{2.5}$ and child mortality. Compared to children in the reference group, children who are exposed to higher levels of $PM_{2.5}$ have higher odds of mortality (Table 4, column 4; Figure 4). While the association between air pollution and child mortality appears to increase up to an exposure level of around 20 $\mu g/m^3$, it seems to flatten at higher levels of exposure. On the other hand, the association between exposure and age-specific measures of child mortality – neonatal, post-neonatal infant, and post-infant child mortality – is less clear, although high levels of exposure are generally associated with increased neonatal mortality (Table 4, columns 1-3).

[Insert Table 4 here]

[Insert Figure 4 here]

The estimated effects of covariates and on child mortality are consistent with the literature. Being a female child, having a mother or a mother's partner with higher educational attainment, and having access to a flush toilet facility are associated with lower odds, while shorter birth intervals, multiple births, and maternal use of tobacco are associated with higher odds of child mortality. The age of the mother has a U-shaped association with child mortality, with ages in the interval 30-34 being associated with the lowest odds. The estimate of the effect of use of solid cooking fuel is not statistically significant, possibly due to the high correlation between the use of solid cooking fuel and ambient $PM_{2.5}$ in low- and middle-income countries (53).

In Table 5, we report results that disaggregate in-utero $PM_{2.5}$ exposure based on its source, i.e. due to dust and sea-salt and from other, mainly anthropogenic, sources. We do not find strong associations between exposure to dust and sea-salt on overall or age-specific child mortality. However, we observe that the odds of neonatal mortality increase rapidly with increase in in-utero exposure to $PM_{2.5}$ without dust and sea-salt (Figure 5). These results may explain the puzzling decline in child mortality at elevated levels of in-utero exposure to overall $PM_{2.5}$ that are seen in Figure 4 – very high exposures in the sample are usually due to high levels of dust and sea-salt, which may have negligible effects on child death.

[Insert Table 5 here]

Based on the model in column 1 of Table 5, we predict neonatal mortality at different levels of in-utero exposure to $PM_{2.5}$ without dust and sea-salt (Figure 6). We see that the predicted probability of neonatal mortality is relatively stable at 31 to 32 neonatal deaths per 1000 live births for in-utero $PM_{2.5}$ level above 10 µg/m³ but decreases sharply to 28 deaths and 23 deaths per 1000 live births for in-utero $PM_{2.5}$ level of 5.2-7.1 µg/m³ and < 3.4 µg/m³, respectively.

[Insert Figure 6 here]

We conduct several robustness checks to confirm the main findings (see Supplemental Material: Tables S4-7). First, we limit the sample to children for whom we are certain that the place of birth matches the place of interview (this is not recorded in all surveys) and, second, we impute dummies for missing covariates rather than dropping observations. We find that the estimates in each of these analyses are comparable to our main results.

We also examine the association of in-utero $PM_{2.5}$ exposure using a logarithmic response curve (see Supplemental Material: Table S8). The odds ratios for the logarithmic specification for total exposure and by source at different levels of exposure are close to the estimates that we find under the non-parametric specification (Figure 7).

[Insert Figure 7 here]

Finally, we estimate the relationship between exposure and neonatal mortality for each country in our data set, separating out exposure by source and using the log specification. The results are

presented in Figure 8. For country-level results, we report the association with the logarithmic response curve since the small sample sizes make estimate a flexible functional form difficult. While the overall estimate from a meta-analysis is similar to the pooled analysis, the estimates for individual countries vary. These results suggest that individual country studies using DHS data may not be able to detect a relationship due to small sample sizes.

## 4. Discussion

In this study, we find that in-utero exposure to overall $PM_{2.5}$ concentration above the WHO guideline of 10 μg/m³ is associated with higher odds of child mortality. When we disaggregate $PM_{2.5}$ exposure by source, we find a much larger association due to exposure excluding dust and sea-salt, with nearly all the burden from ambient air pollution falling on mortality in the neonatal period. The response curve also appears to be nonlinear, sharply increasing even at low levels of exposure and then flattening out at higher levels. Thus, our findings indicate that exposure to particulates due to human activity may be particularly harmful to child health even a very low level of exposure.

Based on our estimates, reducing children's exposure to $PM_{2.5}$ without dust and sea-salt from the sample mean to a level of 3.4 μg/m³ or less would reduce the neonatal mortality rate from 28 to 23 per 1000, approximately. Our results strongly suggest that the mechanism responsible for the effect of ambient air pollution exposure in-utero on neonatal mortality may depend on the composition and toxicity of particulate matter.

There are several limitations to this study and its findings. Our key explanatory variable, in-utero 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 do not control for the more detailed composition of ambient $PM_{2.5}$ or the level of other pollutants, which may bias our estimates (54). Finally, our findings may suffer from residual confounding from omitted variables that are correlated with $PM_{2.5}$ exposure as well as child mortality.

## 5. Conclusion

Our results indicate that children with higher in-utero exposure to ambient $PM_{2.5}$ without dust and sea-salt face significantly higher odds of neonatal mortality. In our sample, a decrease in exposure to ambient $PM_{2.5}$ without dust and sea-salt from the sample mean of about 11 $\mu g/m^3$ to about 2.5 $\mu g/m^3$ is associated with almost a 25% decrease in the predicted neonatal mortality. Given that over 2.5 million children died within the first month of life in 2016 alone, and that the global population-weighted annual average $PM_{2.5}$ level was nearly three times as high at approximately 30 $\mu g/m^3$, policies that aim to reduce ambient air pollution in low- and middle-income countries could contribute significantly to preventing neonatal mortality.

## 6. Acknowledgements

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## 7. Ethics

Ethical approval for the evaluation was granted by the Harvard TH Chan School of Public Health Institutional Review Board (IRB), Protocol No. IRB16-1011.

## 8. Author contributions

NG surveyed the literature, conducted the statistical analysis, and drafted the text; MK contributed substantially to data collection, statistical analysis, and editing the manuscript; DC supervised the study, contributed substantially to its conceptual development, and edited the manuscript. All named authors were responsible for the overall conceptualization, analysis, writing, and finalization of the paper.

## 9. Competing interests

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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## Figures and Tables

**Table 1: Descriptive statistics, outcomes**

| | Mean | Number of cases | Number of observations |
|---|---|---|---|
| Neonatal death (1 = yes) | 0.028 | 15 039 | 529 806 |
| Post-neonatal infant death (1 = yes) | 0.028 | 11 730 | 413 397 |
| Post-infant child death (1 = yes) | 0.025 | 9954 | 401 667 |
| Child death (1 = yes) | 0.072 | 38 645 | 534 476 |

**Table 2: Descriptive statistics, exposures**

| Variable, Statistic | Mean | SD | Number of Cases |
|---|---|---|---|
| In-utero overall $PM_{2.5}$ level (µg/m³) | 24.436 | 14.585 | |
| Log (In-utero overall $PM_{2.5}$ level – 7.3) if $PM_{2.5}$ level > 8.3, 0 otherwise | 2.364 | 1.181 | |
| In-utero $PM_{2.5}$ level without dust and sea-salt (µg/m³) | 10.749 | 7.807 | |
| Log (In-utero $PM_{2.5}$ level without dust and sea-salt – 2.4) if $PM_{2.5}$ level > 3.4, 0 otherwise | 1.692 | 1.025 | |
| In-utero dust and sea-salt level (µg/m³) | 13.920 | 15.137 | |
| Log (In-utero dust and sea-salt level – $4.8e^{-07}$ + 1) if dust and sea-salt level > $4.8e^{-07}$, 0 otherwise | 2.016 | 1.290 | |
| *In-utero overall $PM_{2.5}$ level* | | | |
| < 8.3 µg/m³ | 5.444 | | 64 776 |
| 8.3-12.3 µg/m³ | 10.793 | | 64 085 |
| 12.3-16.0 µg/m³ | 14.066 | | 60 483 |
| 16.0-20.7 µg/m³ | 18.327 | | 69 340 |
| 20.7-27.9 µg/m³ | 24.119 | | 67 639 |
| 27.9-34.5 µg/m³ | 31.396 | | 72 079 |
| 34.5-42.3 µg/m³ | 38.003 | | 75 970 |
| > 42.3 µg/m³ | 51.793 | | 60 104 |
| *In-utero $PM_{2.5}$ level without dust and sea-salt* | | | |
| < 3.4 µg/m³ | 2.530 | | 76 843 |
| 3.4-5.2 µg/m³ | 4.332 | | 72 951 |
| 5.2-7.1 µg/m³ | 6.207 | | 66 294 |
| 7.1-9.9 µg/m³ | 8.356 | | 65 623 |
| 9.9-12.4 µg/m³ | 11.176 | | 70 725 |
| 12.4-15.7 µg/m³ | 13.990 | | 68 417 |
| 15.7-21.0 µg/m³ | 17.887 | | 67 804 |
| > 21.0 µg/m³ | 28.691 | | 45 819 |
| *In-utero dust and sea-salt level* | | | |
| < $4.8e^{-07}$ µg/m³ | 0.000 | | 46 317 |
| $4.8e^{-07}$-1.0 µg/m³ | 0.645 | | 72 190 |
| 1.0-2.1 µg/m³ | 1.573 | | 61 223 |
| 2.1-4.5 µg/m³ | 3.106 | | 66 775 |
| 4.5-13.2 µg/m³ | 8.852 | | 61 654 |
| 13.2-21.4 µg/m³ | 17.483 | | 70 101 |
| 21.4-31.3 µg/m³ | 26.396 | | 75 268 |
| > 31.3 µg/m³ | 41.154 | | 80 948 |
| **Number of observations** | | | **534 476** |

**Table 3: Descriptive statistics, covariates**

| Variable, Statistic | Mean | SD | Number of Cases |
|---|---|---|---|
| **Child-level covariate** | | | |
| First child (1 = yes) | 0.208 | | 111 020 |
| Birth order (number) | 3.641 | 2.403 | |
| Multiple birth (1 = yes) | 0.031 | | 16 636 |
| Child sex (1 = female) | 0.492 | | 262 908 |
| Birth interval, < 18 months (1 = yes) | 0.057 | | 30 607 |
| Birth interval, 18-35 months (1 = yes) | 0.377 | | 201 266 |
| Birth interval, > 35 months (1 = yes) | 0.566 | | 302 603 |
| Time from birth to survey date (months) | 29.164 | 17.184 | |
| **Mother-level covariate** | | | |
| Age of mother, 15-19 years (1 = yes) | 0.048 | | 25 746 |
| Age of mother, 20-24 years (1 = yes) | 0.220 | | 117 371 |
| Age of mother, 25-29 years (1 = yes) | 0.282 | | 150 470 |
| Age of mother, 30-34 years (1 = yes) | 0.212 | | 113 077 |
| Age of mother, 35-39 years (1 = yes) | 0.147 | | 78 485 |
| Age of mother, 40-44 years (1 = yes) | 0.070 | | 37 181 |
| Age of mother, 45-49 years (1 = yes) | 0.023 | | 12 146 |
| Education level of mother, none (1 = yes) | 0.385 | | 205 676 |
| Education level of mother, primary (1 = yes) | 0.342 | | 182 849 |
| Education level of mother, secondary (1 = yes) | 0.227 | | 121 478 |
| Education level of mother, higher (1 = yes) | 0.046 | | 24 473 |
| Education level of mother's partner, none (1 = yes) | 0.320 | | 170 947 |
| Education level of mother's partner, primary (1 = yes) | 0.318 | | 169 886 |
| Education level of mother's partner, secondary (1 = yes) | 0.292 | | 156 110 |
| Education level of mother's partner, higher (1 = yes) | 0.070 | | 37 533 |
| Mother uses tobacco (1 = yes) | 0.035 | | 18 859 |
| **Household-level covariate** | | | |
| Household uses solid cooking fuel (1 = yes) | 0.824 | | 440 375 |
| Household has access to flush toilet (1 = yes) | 0.164 | | 87 780 |
| Household has access to piped drinking water (1 = yes) | 0.305 | | 163 151 |
| Urban residence (1 = yes) | 0.292 | | 156 049 |
| Wealth quintile of household, poorest (1 = yes) | 0.258 | | 137 996 |
| Wealth quintile of household, poor (1 = yes) | 0.221 | | 118 072 |
| Wealth quintile of household, middle (1 = yes) | 0.202 | | 107 870 |
| Wealth quintile of household, rich (1 = yes) | 0.175 | | 93 632 |
| Wealth quintile of household, richest (1 = yes) | 0.144 | | 76 906 |
| **Number of observations** | | | **534 476** |

**Table 4: The effect of in-utero all source $PM_{2.5}$ level on child mortality**

| Variable, Outcome | (1) Neonatal death | (2) Post-neonatal infant death | (3) Post-infant child death | (4) Child death |
|---|---|---|---|---|
| *In-utero overall $PM_{2.5}$ level (Reference group: 0.0-8.3 µg/m³)* | | | | |
| 8.3-12.3 µg/m³ | 1.158 [0.971,1.381] | 1.046 [0.846,1.294] | 1.048 [0.791,1.388] | 1.106 [0.978,1.252] |
| 12.3-16.0 µg/m³ | 1.095 [0.901,1.330] | 1.135 [0.906,1.423] | 1.302* [0.969,1.751] | 1.174** [1.027,1.342] |
| 16.0-20.7 µg/m³ | 1.097 [0.882,1.364] | 1.193 [0.928,1.533] | 1.523** [1.103,2.104] | 1.262*** [1.087,1.466] |
| 20.7-27.9 µg/m³ | 1.181 [0.934,1.495] | 1.196 [0.911,1.569] | 1.410* [0.998,1.993] | 1.277*** [1.086,1.501] |
| 27.9-34.5 µg/m³ | 1.362** [1.057,1.755] | 1.172 [0.875,1.569] | 1.189 [0.825,1.714] | 1.280*** [1.077,1.522] |
| 34.5-42.3 µg/m³ | 1.277* [0.984,1.658] | 1.155 [0.855,1.561] | 1.068 [0.734,1.554] | 1.211** [1.013,1.447] |
| > 42.3 µg/m³ | 1.287* [0.984,1.683] | 1.149 [0.843,1.566] | 1.118 [0.764,1.635] | 1.216** [1.012,1.460] |
| Household uses solid cooking fuel | 0.991 [0.907,1.082] | 1.004 [0.897,1.124] | 0.956 [0.836,1.093] | 0.995 [0.935,1.059] |
| Mother uses tobacco | 1.198*** [1.090,1.316] | 1.248*** [1.132,1.375] | 1.140** [1.011,1.286] | 1.207*** [1.136,1.282] |
| Time from birth to survey (months) | - | - | 1.025*** [1.019,1.031] | 1.012*** [1.009,1.015] |
| Birth interval, < 18 months | 3.384*** [3.174,3.608] | 2.756*** [2.562,2.964] | 1.949*** [1.795,2.116] | 2.825*** [2.704,2.950] |
| Birth interval, 18-35 months | 1.398*** [1.336,1.463] | 1.509*** [1.438,1.584] | 1.387*** [1.317,1.461] | 1.427*** [1.387,1.468] |
| Multiple birth | 6.820*** [6.421,7.244] | 3.204*** [2.949,3.481] | 2.037*** [1.835,2.262] | 4.422*** [4.221,4.632] |
| Female | 0.750*** [0.725,0.776] | 0.919*** [0.886,0.954] | 0.932*** [0.895,0.971] | 0.847*** [0.830,0.866] |
| First child | 2.348*** [2.206,2.499] | 1.490*** [1.388,1.598] | 1.345*** [1.244,1.454] | 1.756*** [1.687,1.827] |
| Birth order | 1.031*** [1.018,1.045] | 1.035*** [1.020,1.049] | 1.046*** [1.031,1.061] | 1.036*** [1.027,1.045] |
| Age of mother, 15-19 years | 1.174*** [1.087,1.267] | 1.337*** [1.210,1.476] | 1.073 [0.947,1.216] | 1.193*** [1.131,1.259] |
| Age of mother, 25-29 years | 0.862*** [0.816,0.911] | 0.923*** [0.869,0.979] | 0.890*** [0.833,0.949] | 0.900*** [0.870,0.932] |
| Age of mother, 30-34 years | 0.915*** [0.855,0.979] | 0.850*** [0.790,0.915] | 0.861*** [0.796,0.931] | 0.884*** [0.847,0.922] |
| Age of mother, 35-39 years | 1.040 [0.959,1.128] | 0.878*** [0.803,0.959] | 0.833*** [0.757,0.917] | 0.938** [0.890,0.988] |
| Age of mother, 40-44 years | 1.199*** [1.082,1.328] | 0.836*** [0.746,0.936] | 0.758*** [0.672,0.855] | 0.960 [0.899,1.026] |
| Age of mother, 45-49 years | 1.534*** [1.338,1.758] | 0.896 [0.769,1.044] | 0.867* [0.741,1.013] | 1.136*** [1.040,1.241] |
| Education level of mother, primary | 1.015 [0.963,1.070] | 0.956 [0.903,1.011] | 0.946* [0.890,1.005] | 0.979 [0.948,1.012] |
| Education level of mother, secondary | 0.951 [0.886,1.020] | 0.857*** [0.793,0.927] | 0.723*** [0.661,0.791] | 0.867*** [0.828,0.907] |

| Variable, Outcome | (1) Neonatal death | (2) Post-neonatal infant death | (3) Post-infant child death | (4) Child death |
|---|---|---|---|---|
| Education level of mother, higher | 0.879* | 0.610*** | 0.389*** | 0.697*** |
| | [0.764,1.012] | [0.503,0.740] | [0.294,0.516] | [0.630,0.772] |
| Education level of mother's partner, primary | 0.991 | 0.928** | 0.936** | 0.953*** |
| | [0.939,1.045] | [0.877,0.983] | [0.880,0.996] | [0.922,0.986] |
| Education level of mother's partner, secondary | 0.919*** | 0.892*** | 0.867*** | 0.887*** |
| | [0.863,0.977] | [0.834,0.954] | [0.804,0.935] | [0.852,0.923] |
| Education level of mother's partner, higher | 0.794*** | 0.825*** | 0.789*** | 0.787*** |
| | [0.716,0.881] | [0.729,0.934] | [0.684,0.909] | [0.734,0.845] |
| Household has access to flush toilet | 0.923* | 0.848*** | 0.825*** | 0.893*** |
| | [0.851,1.001] | [0.765,0.941] | [0.724,0.939] | [0.843,0.946] |
| Household has access to piped water | 0.981 | 0.969 | 0.954 | 0.970* |
| | [0.934,1.031] | [0.914,1.026] | [0.893,1.019] | [0.938,1.003] |
| Urban residence | 0.979 | 1.020 | 0.910*** | 0.969* |
| | [0.927,1.034] | [0.958,1.086] | [0.848,0.975] | [0.933,1.006] |
| Wealth quintile of household, poor | 1.005 | 0.985 | 1.005 | 0.996 |
| | [0.954,1.060] | [0.933,1.041] | [0.947,1.065] | [0.964,1.029] |
| Wealth quintile of household, middle | 1.012 | 0.952 | 0.927** | 0.966* |
| | [0.957,1.071] | [0.897,1.012] | [0.869,0.988] | [0.933,1.001] |
| Wealth quintile of household, rich | 1.027 | 0.904*** | 0.904*** | 0.947*** |
| | [0.963,1.095] | [0.844,0.968] | [0.838,0.976] | [0.909,0.987] |
| Wealth quintile of household, richest | 0.964 | 0.782*** | 0.710*** | 0.834*** |
| | [0.883,1.052] | [0.707,0.864] | [0.632,0.798] | [0.787,0.884] |
| N | 528 821 | 411 164 | 391 636 | 534 265 |

All regressions include survey and subnational region fixed effects. The coefficients are the odds ratio of outcome in comparison to the reference category (for categorical variables) or for a unit increase in exposure or covariate (for continuous variables). The 95% confidence intervals are presented in brackets. * p<0.10, ** p<0.05, *** p<0.01.

**Table 5: The effect of in-utero $PM_{2.5}$ level by source on child mortality**

| Variable, Outcome | (1) Neonatal death | (2) Post-neonatal infant death | (3) Post-infant child death | (4) Child death |
|---|---|---|---|---|
| *In-utero $PM_{2.5}$ level without dust and sea-salt (Reference group: 0-3.4 µg/m³)* | | | | |
| 3.4-5.2 µg/m³ | 1.107** | 1.058 | 1.097* | 1.097*** |
| | [1.006,1.219] | [0.955,1.173] | [0.984,1.224] | [1.030,1.168] |
| 5.2-7.1 µg/m³ | 1.212*** | 1.047 | 1.172** | 1.137*** |
| | [1.071,1.370] | [0.914,1.198] | [1.025,1.339] | [1.048,1.232] |
| 7.1-9.9 µg/m³ | 1.319*** | 1.052 | 1.059 | 1.135*** |
| | [1.152,1.510] | [0.903,1.227] | [0.916,1.225] | [1.038,1.241] |
| 9.9-12.4 µg/m³ | 1.349*** | 1.048 | 0.995 | 1.101* |
| | [1.159,1.570] | [0.883,1.244] | [0.842,1.176] | [0.997,1.217] |
| 12.4-15.7 µg/m³ | 1.393*** | 1.119 | 0.981 | 1.132** |
| | [1.184,1.640] | [0.933,1.341] | [0.819,1.175] | [1.018,1.259] |
| 15.7-21.0 µg/m³ | 1.417*** | 1.066 | 1.020 | 1.146** |
| | [1.186,1.693] | [0.877,1.295] | [0.838,1.240] | [1.022,1.285] |
| > 21.0 µg/m³ | 1.406*** | 0.965 | 0.912 | 1.069 |
| | [1.142,1.731] | [0.770,1.209] | [0.715,1.163] | [0.934,1.223] |
| *In-utero dust and sea-salt level (Reference group: 0-4.8e$^{-07}$)* | | | | |
| 4.8e$^{-07}$-1.0 µg/m³ | 0.986 | 1.077 | 0.956 | 0.961 |
| | [0.879,1.104] | [0.959,1.210] | [0.833,1.096] | [0.896,1.030] |
| 1.0-2.1 µg/m³ | 1.113* | 0.988 | 0.960 | 0.970 |
| | [0.982,1.262] | [0.866,1.128] | [0.821,1.122] | [0.896,1.051] |
| 2.1-4.5 µg/m³ | 1.025 | 1.049 | 0.880 | 0.959 |
| | [0.895,1.173] | [0.912,1.207] | [0.745,1.039] | [0.880,1.045] |
| 4.5-13.2 µg/m³ | 1.091 | 1.002 | 0.988 | 0.985 |
| | [0.923,1.290] | [0.834,1.204] | [0.789,1.238] | [0.880,1.103] |
| 13.2-21.4 µg/m³ | 1.159 | 1.005 | 1.042 | 1.014 |
| | [0.944,1.424] | [0.796,1.269] | [0.789,1.376] | [0.882,1.167] |
| 21.4-31.3 µg/m³ | 1.192 | 0.993 | 0.922 | 0.982 |
| | [0.951,1.493] | [0.765,1.287] | [0.682,1.247] | [0.843,1.145] |
| > 31.3 µg/m³ | 1.110 | 1.007 | 0.913 | 0.952 |
| | [0.871,1.413] | [0.764,1.327] | [0.665,1.253] | [0.808,1.122] |
| Household uses solid cooking fuel | 0.990 | 1.004 | 0.956 | 0.995 |
| | [0.907,1.081] | [0.897,1.125] | [0.836,1.094] | [0.935,1.059] |
| Mother uses tobacco | 1.199*** | 1.244*** | 1.134** | 1.205*** |
| | [1.091,1.317] | [1.129,1.372] | [1.006,1.279] | [1.134,1.279] |
| Time from birth to survey | - | - | 1.025*** | 1.012*** |
| | | | [1.018,1.031] | [1.009,1.015] |
| Birth interval, < 18 months | 3.389*** | 2.754*** | 1.947*** | 2.825*** |
| | [3.179,3.614] | [2.560,2.962] | [1.793,2.114] | [2.705,2.950] |
| Birth interval, 18-35 months | 1.400*** | 1.509*** | 1.386*** | 1.427*** |
| | [1.338,1.465] | [1.438,1.584] | [1.316,1.459] | [1.387,1.468] |
| Multiple birth | 6.815*** | 3.203*** | 2.036*** | 4.420*** |
| | [6.416,7.238] | [2.948,3.480] | [1.833,2.260] | [4.220,4.631] |
| Female | 0.750*** | 0.919*** | 0.932*** | 0.848*** |
| | [0.725,0.776] | [0.886,0.954] | [0.895,0.971] | [0.830,0.866] |
| First child | 2.350*** | 1.489*** | 1.344*** | 1.756*** |
| | [2.208,2.502] | [1.388,1.598] | [1.243,1.453] | [1.687,1.828] |
| Birth order | 1.031*** | 1.035*** | 1.046*** | 1.036*** |
| | [1.018,1.045] | [1.020,1.050] | [1.031,1.062] | [1.027,1.045] |

| Variable, Outcome | (1) Neonatal death | (2) Post-neonatal infant death | (3) Post-infant child death | (4) Child death |
|---|---|---|---|---|
| Age of mother, 15-19 years | 1.174*** | 1.337*** | 1.073 | 1.194*** |
| | [1.088,1.268] | [1.211,1.476] | [0.947,1.216] | [1.131,1.259] |
| Age of mother, 25-29 years | 0.861*** | 0.923*** | 0.890*** | 0.900*** |
| | [0.815,0.910] | [0.869,0.979] | [0.834,0.950] | [0.870,0.932] |
| Age of mother, 30-34 years | 0.914*** | 0.850*** | 0.861*** | 0.884*** |
| | [0.854,0.978] | [0.790,0.915] | [0.796,0.931] | [0.847,0.922] |
| Age of mother, 35-39 years | 1.039 | 0.878*** | 0.833*** | 0.938** |
| | [0.958,1.127] | [0.803,0.959] | [0.757,0.917] | [0.890,0.988] |
| Age of mother, 40-44 years | 1.196*** | 0.835*** | 0.757*** | 0.960 |
| | [1.080,1.325] | [0.746,0.936] | [0.672,0.854] | [0.898,1.025] |
| Age of mother, 45-49 years | 1.531*** | 0.897 | 0.868* | 1.136*** |
| | [1.336,1.754] | [0.770,1.045] | [0.742,1.014] | [1.040,1.241] |
| Education level of mother, primary | 1.012 | 0.957 | 0.947* | 0.979 |
| | [0.960,1.066] | [0.904,1.012] | [0.892,1.007] | [0.947,1.012] |
| Education level of mother, secondary | 0.948 | 0.858*** | 0.722*** | 0.866*** |
| | [0.884,1.017] | [0.793,0.928] | [0.659,0.790] | [0.827,0.906] |
| Education level of mother, higher | 0.876* | 0.611*** | 0.389*** | 0.697*** |
| | [0.761,1.007] | [0.503,0.741] | [0.294,0.516] | [0.629,0.772] |
| Education level of mother's partner, primary | 0.984 | 0.929** | 0.939** | 0.952*** |
| | [0.933,1.038] | [0.877,0.984] | [0.882,0.999] | [0.921,0.985] |
| Education level of mother's partner, secondary | 0.913*** | 0.892*** | 0.868*** | 0.886*** |
| | [0.858,0.971] | [0.834,0.954] | [0.805,0.936] | [0.851,0.922] |
| Education level of mother's partner, higher | 0.788*** | 0.825*** | 0.788*** | 0.786*** |
| | [0.711,0.875] | [0.729,0.933] | [0.684,0.909] | [0.733,0.843] |
| Household has access to flush toilet | 0.926* | 0.849*** | 0.823*** | 0.893*** |
| | [0.853,1.005] | [0.765,0.941] | [0.723,0.937] | [0.843,0.946] |
| Household has access to piped water | 0.983 | 0.969 | 0.954 | 0.971* |
| | [0.936,1.033] | [0.914,1.027] | [0.893,1.020] | [0.939,1.004] |
| Urban residence | 0.980 | 1.023 | 0.911*** | 0.971 |
| | [0.928,1.036] | [0.961,1.089] | [0.850,0.977] | [0.936,1.008] |
| Wealth quintile of household, poor | 1.004 | 0.986 | 1.005 | 0.996 |
| | [0.952,1.058] | [0.934,1.042] | [0.948,1.066] | [0.964,1.029] |
| Wealth quintile of household, middle | 1.011 | 0.953 | 0.928** | 0.966* |
| | [0.956,1.069] | [0.897,1.012] | [0.870,0.989] | [0.933,1.001] |
| Wealth quintile of household, rich | 1.026 | 0.905*** | 0.906** | 0.947*** |
| | [0.962,1.093] | [0.845,0.969] | [0.840,0.978] | [0.910,0.987] |
| Wealth quintile of household, richest | 0.960 | 0.782*** | 0.712*** | 0.833*** |
| | [0.880,1.048] | [0.707,0.864] | [0.634,0.800] | [0.786,0.884] |
| N | 528 821 | 411 164 | 391 636 | 534 265 |

All regressions include survey and subnational region fixed effects. The coefficients are the odds ratio of outcome in comparison to the reference category (for categorical variables) or for a unit increase in exposure or covariate (for continuous variables). The 95% confidence intervals are presented in brackets. * p<0.10, ** p<0.05, *** p<0.01.

**Figure 1: The geographic distribution of annual ambient $PM_{2.5}$ concentration from all sources in 1998 (top) and 2014 (below)**

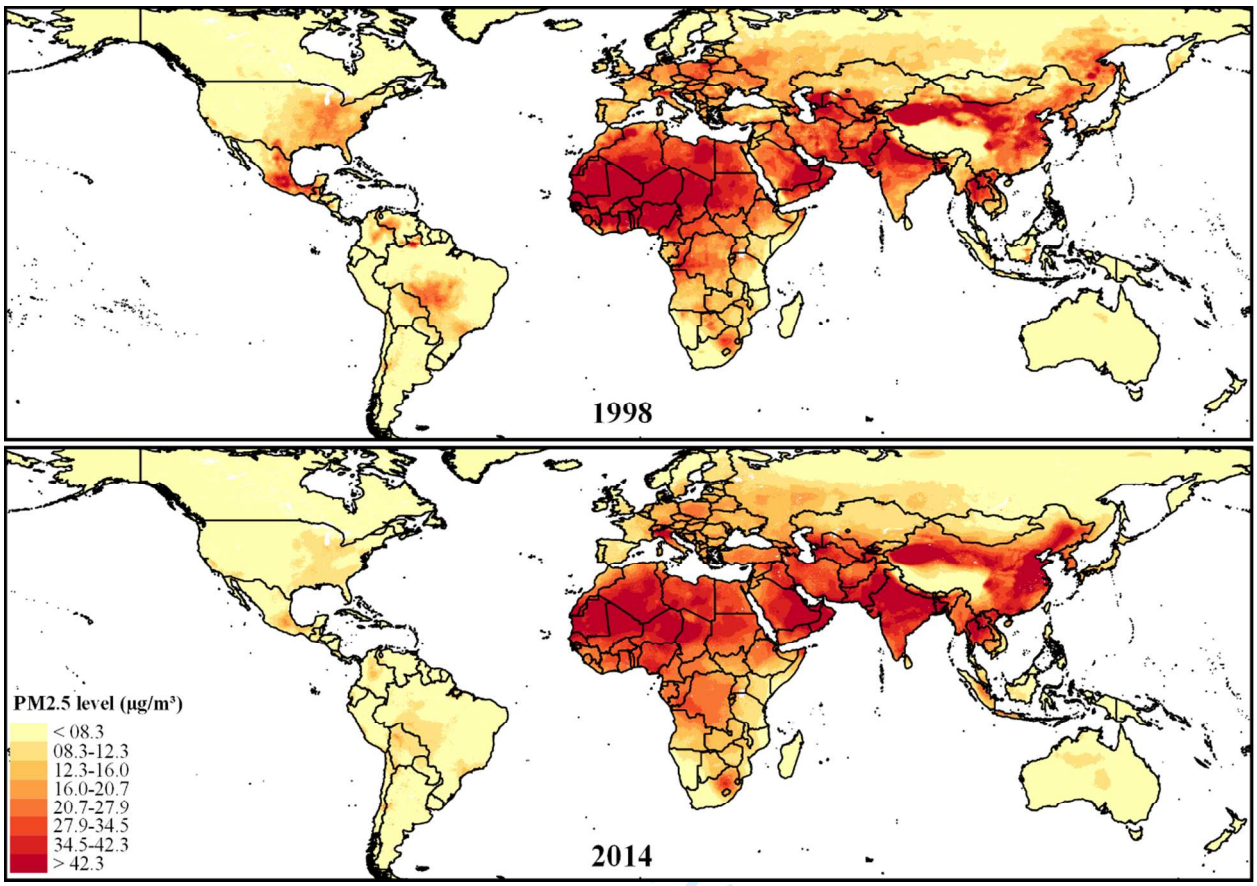

**Figure 2: The geographic distribution of annual ambient $PM_{2.5}$ concentration without dust and sea-salt in 1998 (top) and 2014 (below)**

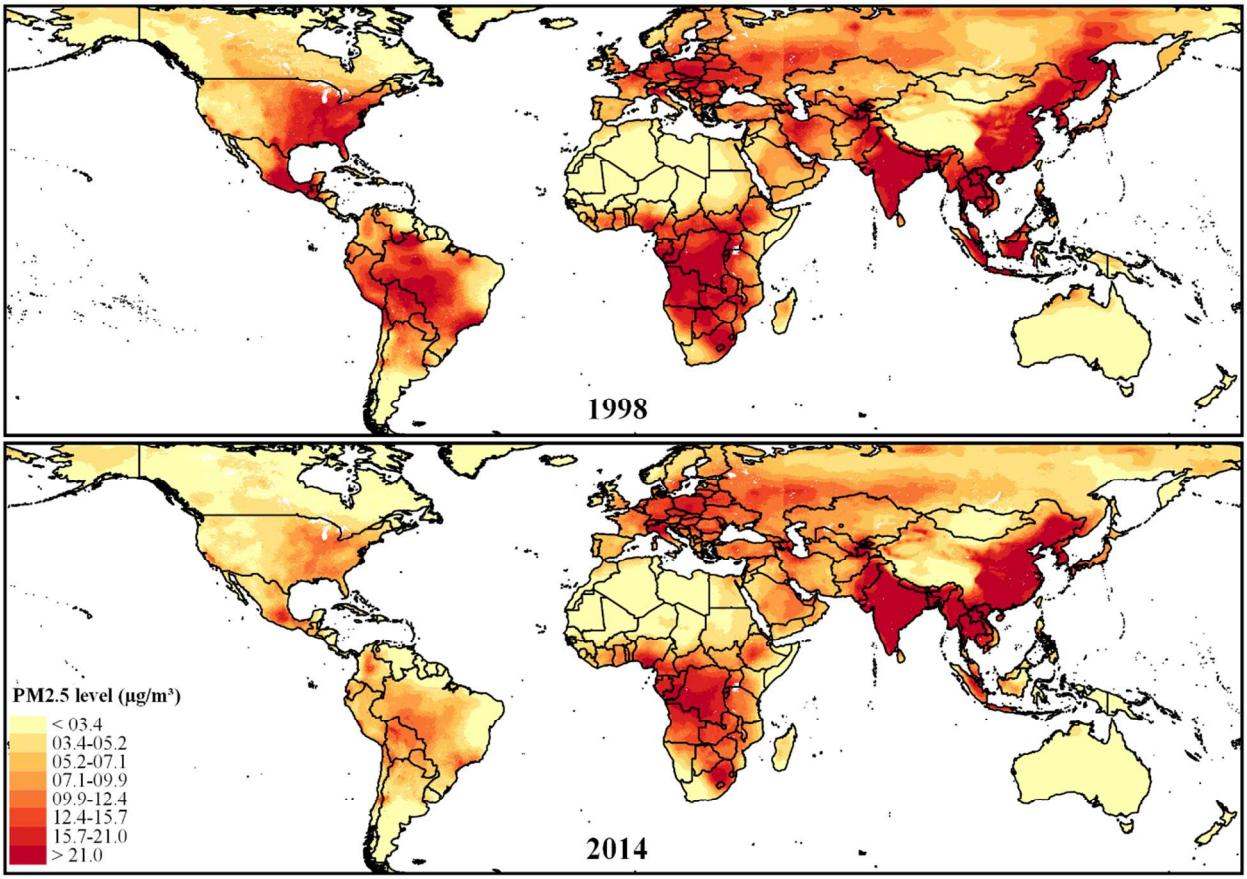

**Figure 3: Box plot of in-utero $PM_{2.5}$ level without dust and sea-salt by country (µg/m³).** Note that the data across countries are not strictly comparable as different countries may have been surveyed in different years.

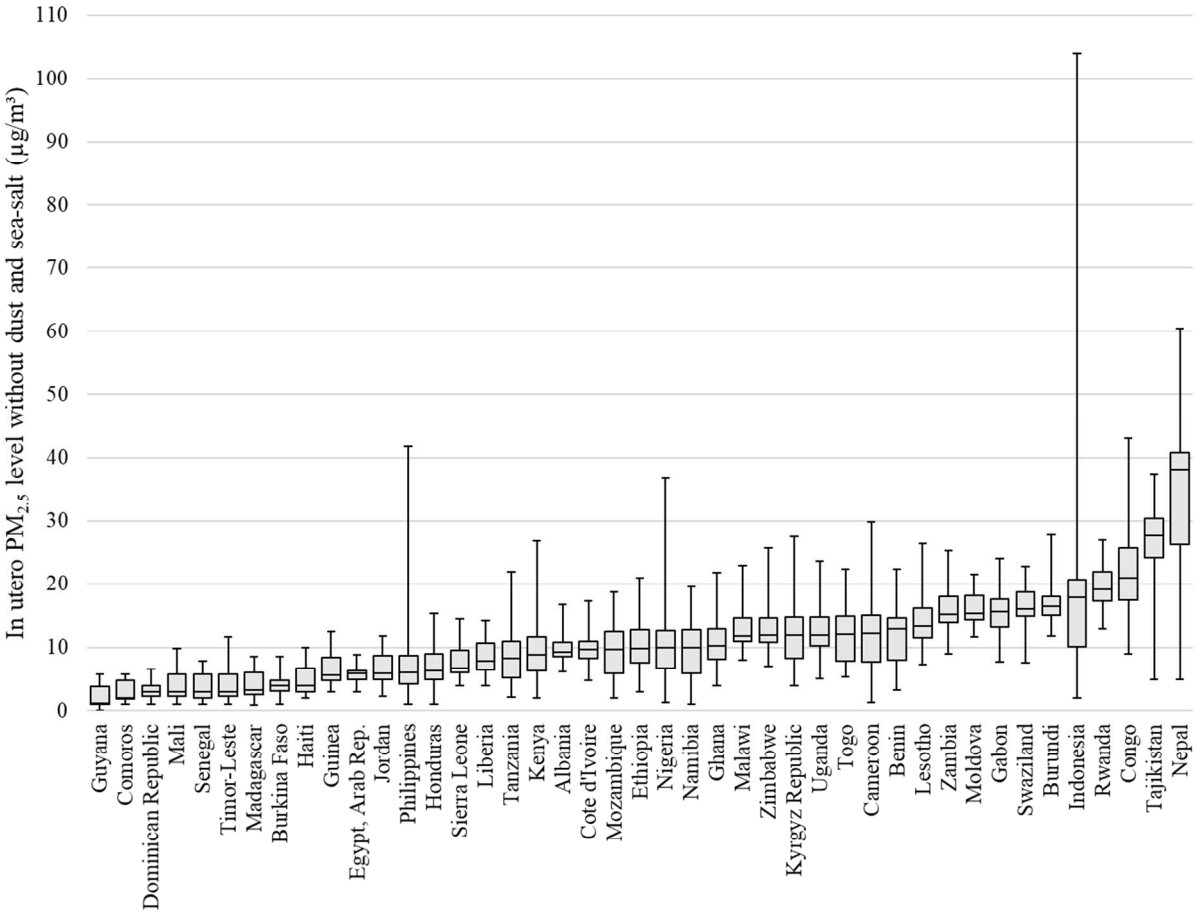

**Figure 4: The odds ratio of child death for in-utero overall $PM_{2.5}$ level.** The values of the odds ratios have been plotted at the mean values of the exposure categories in the sample.

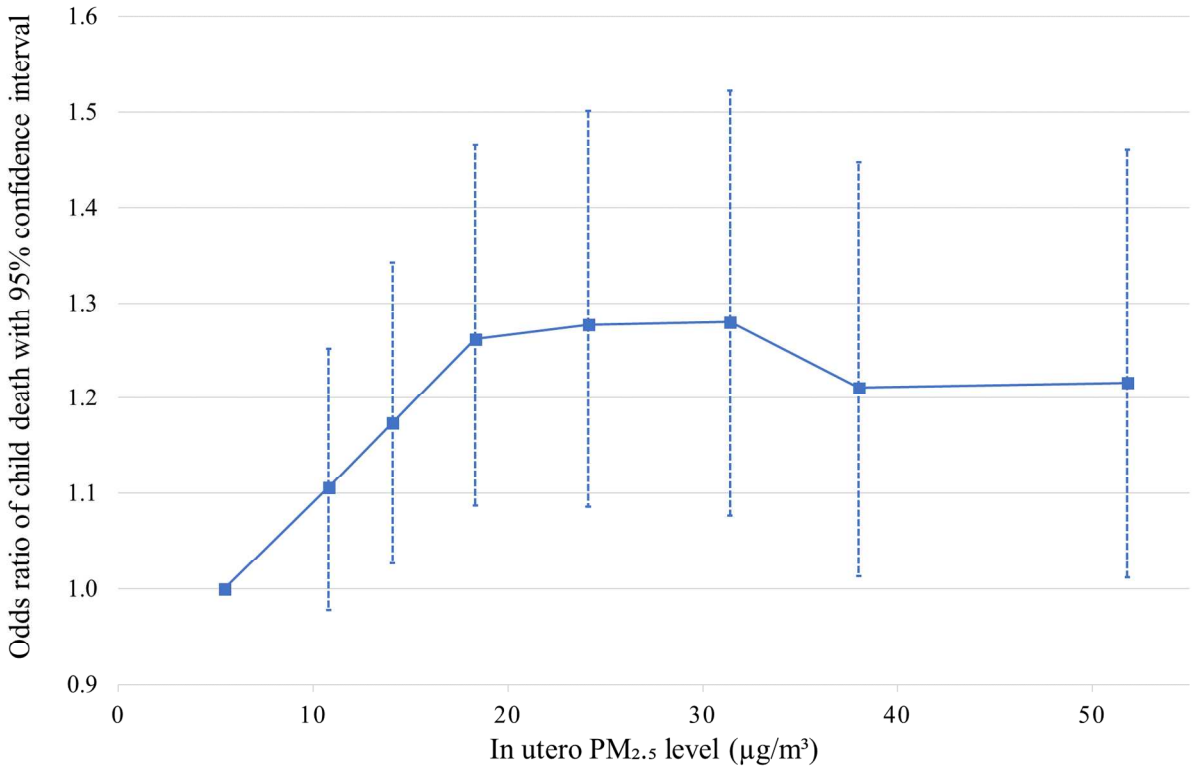

**Figure 5: The odds ratio of neonatal death for in-utero $PM_{2.5}$ level by source.** The values of the odds ratios have been plotted at the mean values of the exposure categories in the sample.

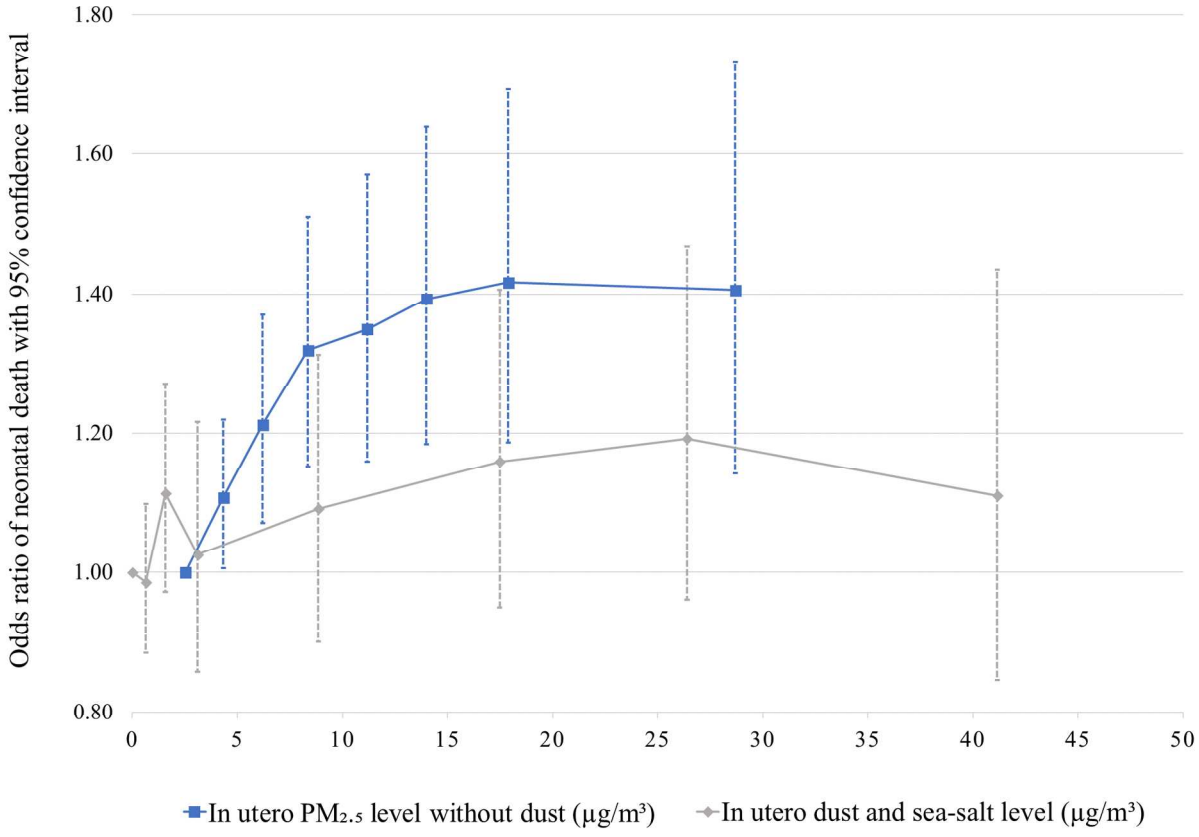

**Figure 6: Adjusted predicted mean neonatal mortality with 95% confidence interval (at observed values of covariates).** The values of the odds ratios have been plotted at the mean values of the exposure categories in the sample.

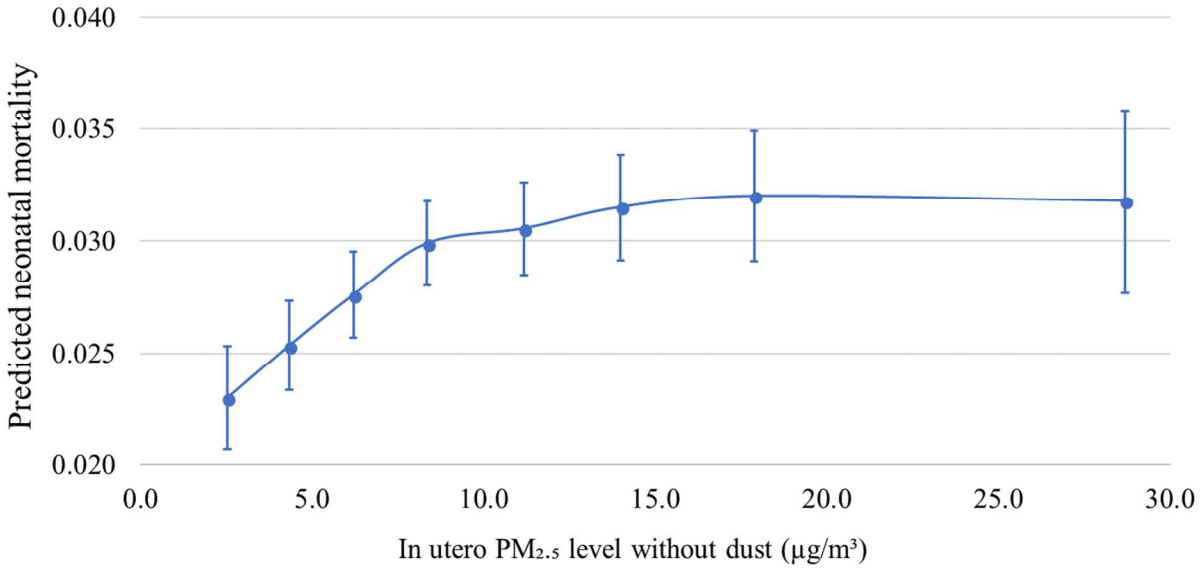

**Figure 7: The odds ratio of neonatal death for in-utero $PM_{2.5}$ level, overall as well as by source, based on logarithmic concentration response curves.** The values of the odds ratios have been plotted at the mean values of the exposure categories in the sample.

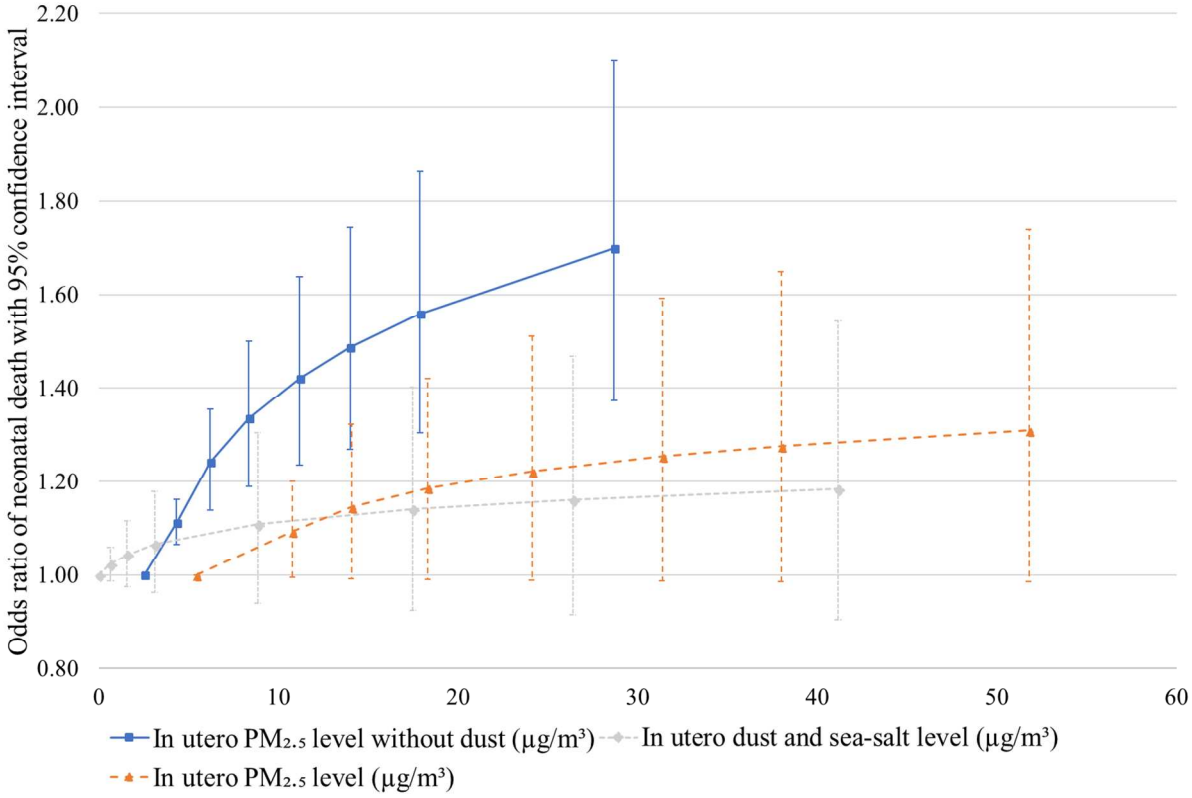

**Figure 8: Country-specific analysis of the effect of in-utero $PM_{2.5}$ level without dust and sea-salt on neonatal death.** Guyana and Comoros are excluded from the forest plot as they do not have observations with in-utero $PM_{2.5}$ level without dust and sea-salt exceeding 3.4 µg/m³. Moldova is excluded from the forest plot as its odds ratio (3389.56) and 95% confidence interval (2.51, 4 569 115) were too large to depict on the graph.