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Air pollution and under-5 child mortality: linking satellite and IPUMS-DHS data across 41 countries in South Asia and Sub-Saharan Africa

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Abstract

Background Despite progress, under-five mortality remains high, especially in Sub-Saharan Africa and South Asia, where around 13,400 children die daily. Environmental pollutants, including PM2.5 from outdoor air and household air pollution, significantly contribute to these preventable deaths.

Methods This cross-country study combined satellite data with 113 surveys from the IPUMS-DHS dataset (1998–2019) to examine under-five child mortality in 41 developing countries. The integration of Global Annual Particulate Matter with a diameter of 2.5 micrometres or less (PM2.5) Grids from Socioeconomic Data and Applications Center (SEDAC) and geospatial data from the DHS Program enabled a focused analysis of the association between indoor and outdoor air pollution, particularly PM2.5, and child mortality rates using both logistic and multilevel logistic regression models, as well as estimating Population Attributable Fractions (PAF) to quantify the mortality burden attributable to these pollutants.

Results Outdoor air pollution, measured by a one standard deviation increase in PM2.5, significantly increased the risk of child mortality (Odds Ratio [OR]: 1.14; 95% Confidence Interval [CI]: 1.10–1.18; $p < 0.001$). Moderate and high household air pollution exposure also heightened this risk, with increases of 37% (OR: 1.37; 95% CI: 1.24–1.53; $p < 0.001$) and 40% (OR: 1.40; 95% CI: 1.26–1.56; $p < 0.001$), respectively, compared to no exposure. Multilevel models (Models 5a and 10a) produced similar estimates to standard logistic regression, indicating robust associations. Additionally, Population Attributable Fraction analysis revealed that approximately 11.9% of under-five mortality could be prevented by reducing ambient PM2.5 exposure and 12.0% by mitigating household air pollution. The interaction between indoor and outdoor pollution revealed complex dynamics, with moderate and high household exposure associated with a reduction in mortality risk when combined with PM2.5. Geographical disparities were observed, with stronger correlations between outdoor air pollution and child mortality in Africa compared to Asia, and more pronounced impacts in low-income countries. However, household air pollution had stronger association with child mortality in Africa and lower- and middle-income countries.

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Conclusions Our findings could serve as a guide for policy development aimed at reducing under-five mortality, ultimately contributing to the attainment of the Sustainable Development Goal (SDGs).

Keywords Under-5 child mortality, Outdoor air pollution, Indoor air pollution, Solid cooking fuels, Developing countries

Background

While the world has made remarkable strides in reducing child mortality over the past three decades, the under-five mortality rate remains alarmingly high. Despite the global reduction in under-five mortality by 60 per cent, from 93 deaths per 1,000 live births in 1990 to 37 in 2022, progress has slowed significantly in recent years [1, 2]. In 2022 alone, approximately 13,400 children under the age of five died every day, highlighting the persistent and urgent challenge of child survival. The burden of these largely preventable deaths is disproportionately felt in regions like Sub-Saharan Africa and South Asia, where poverty, infectious diseases, and malnutrition continue to claim young lives at an intolerable rate [1]. Sub-Saharan Africa, in particular, faces a staggering disparity, with a child's risk of dying before their fifth birthday being 15 times higher than in high-income countries [1, 3].

Under-five child mortality is influenced by various factors, including infectious diseases, neonatal complications, and socio-economic disparities. For instance, in Northern Ethiopia, major causes of under-five mortality include bacterial sepsis, prematurity, intestinal infection disease, acute lower respiratory infections, and birth asphyxia, with significant disparities observed between rural and urban residents [4]. Similarly, in South Africa, leading causes of death among children under five are pneumonia, gastroenteritis, prematurity, and injuries, indicating the need for comprehensive health interventions across service delivery platforms [5]. Furthermore, the global burden of child mortality is exacerbated by factors such as maternal education, access to healthcare, and socio-economic status, with significant variations observed across regions and countries [6].

Among the major factors contributing to child mortality, air pollution stands out as a significant and preventable cause. Exposure to fine particulate matter (PM2.5) has been consistently linked to an increased risk of child mortality, particularly in children under the age of five. Studies such as studies [7] and [8] have demonstrated that even small increases in PM2.5 concentrations can significantly increase the likelihood of mortality in this vulnerable age group.

The causal mechanisms through which PM2.5 leads to under-5 child mortality operate through both prenatal and postnatal pathways. During pregnancy, exposure to PM2.5 can impair fetal development, leading to adverse outcomes such as low birth weight (LBW) and preterm birth (PTB). These conditions are critical risk factors for neonatal mortality, as infants born with LBW or prematurely are more susceptible to health complications that can be fatal [9]. Study [9] illustrate this by showing that a 10 $\mu g/m^3$ increase in PM2.5 is associated with significant reductions in birth weight and gestational age, both of which are directly linked to higher neonatal and infant mortality rates.

Postnatal exposure to PM2.5 further exacerbates the risk of mortality through its impact on respiratory health. Infants and young children are particularly vulnerable to respiratory infections and other complications resulting from inhaling fine particulate matter. Study [10] found that long-term exposure to PM2.5 is significantly associated with postneonatal mortality, particularly from respiratory causes. The harmful effects of PM2.5 are not uniform; the specific composition of PM2.5 plays a critical role in determining its impact. Study [11] found that carbonaceous PM2.5, primarily from human activities, is particularly detrimental, increasing the odds of neonatal mortality by over 50%.

Extending the discussion on the association between PM2.5 exposure and under-5 child mortality, it is important to consider the role of various environmental pollutants that often coexist with PM2.5, such as polycyclic aromatic hydrocarbons (PAHs) and other toxic compounds. Study by [12] have demonstrated that PAHs, commonly found alongside PM2.5, contribute significantly to health risks, including carcinogenic outcomes. The environmental persistence and bioaccumulation of these compounds can exacerbate the adverse effects of PM2.5, particularly in vulnerable populations like children. Additionally, the interaction of PM2.5 with other environmental contaminants, such as those found in water and soil, as explored by study [13] and [14], illustrates how pollutants can re-enter the air through secondary emissions, complicating efforts to mitigate PM2.5 exposure and its effects. Moreover, the presence of pesticides like malathion, as highlighted by study [15], can further interact with PM2.5, leading to compounded health risks.

Numerous studies have established a clear link between indoor air pollution caused by the use of solid cooking fuels and increased child mortality. For instance, study [16] found that solid fuel use significantly increases child deaths, particularly in the post-neonatal period, with more pronounced effects observed among girls. They reported that the use of solid fuels for cooking raises the risk of under-five mortality by 4.9% in India. Study [17]

also identified a strong association between solid fuel use and post-neonatal and child mortality in Nigeria, attributing a significant percentage of deaths in these age groups to the use of solid fuels. These findings are supported by study [18], who found that household air pollution from solid fuels is linked to neonatal and infant mortality, as well as other adverse health outcomes in Bangladesh.

The primary causal mechanism through which indoor air pollution increases the risk of child mortality is the heightened susceptibility to acute lower respiratory infections (ALRIs), such as pneumonia [19]. The combustion of solid fuels releases a high concentration of particulate matter and toxic pollutants, which impair the respiratory system and increase the likelihood of severe respiratory infections. Study [19] demonstrated that the risk of ALRIs in children significantly increases with longer exposure to solid fuel smoke, with odds ratios rising as the duration of exposure grows. Additionally, the lack of proper ventilation in households using solid fuels exacerbates the exposure, leading to higher concentrations of indoor pollutants and a corresponding increase in the risk of ALRI-related mortality [20]. This direct impact on respiratory health is a key pathway through which indoor air pollution contributes to the high rates of child mortality observed in these settings.

Another critical mechanism is the effect of indoor air pollution on adverse birth outcomes, which in turn increase the risk of neonatal and infant mortality. Exposure to toxic fumes from solid fuels during pregnancy has been linked to low birth weight (LBW), preterm birth, and other complications that increase the vulnerability of newborns to mortality. Study [18] found that cooking with solid fuels inside the house was associated with higher risks of LBW and neonatal mortality in Bangladesh. The inhalation of harmful pollutants by pregnant women can lead to intrauterine growth restrictions, contributing to poor birth outcomes that predispose infants to higher mortality risks.

The link between indoor air pollution (IAP) and child mortality is further explained by examining the presence of harmful pollutants in indoor environments where solid fuels are commonly used for cooking. The study [21] identifies the presence of hazardous BTEX compounds (benzene, toluene, ethylbenzene, and xylene) in indoor air, which are particularly prevalent in environments with poor ventilation and high solid fuel use. These compounds pose significant health risks, including increased respiratory illnesses that can elevate the risk of mortality in children. Moreover, the study [14] underscores the importance of addressing environmental contaminants, as these pollutants can contribute to the cumulative exposure that exacerbates respiratory conditions.

The interaction between indoor and outdoor air pollution also significantly increases the risk of child mortality, particularly in developing regions where both sources of pollution are prevalent. Studies have shown that indoor air pollution (IAP) from the use of solid fuels for cooking and heating is a major contributor to respiratory infections, which are a leading cause of child mortality. For example, the study [22] highlights that exposure to unprocessed solid fuels substantially increases the risk of pneumonia in children under five, a critical age group for respiratory-related mortality. This risk is compounded when indoor pollutants interact with outdoor air pollution, such as particulate matter (PM) from traffic emissions or industrial activities. Study [23] report that outdoor air pollution, particularly fine particulate matter (PM2.5), is associated with significant mortality from cardiopulmonary diseases, including in children.

The synergistic effect of indoor and outdoor air pollution is evident in settings where poor ventilation exacerbates the infiltration of outdoor pollutants into indoor environments. The study [24] demonstrate that a significant proportion of mortality associated with outdoor PM is attributable to indoor exposure to particles of outdoor origin. This indicates that even if outdoor air pollution is the primary source, its impact is magnified by poor indoor air quality. Furthermore, the combined exposure to both indoor and outdoor pollutants has been shown to result in higher rates of acute respiratory infections and other health complications in children [25].

This study offers a comprehensive analysis of the relationship between outdoor air pollution, as measured by PM2.5 levels, indoor air pollution, and their synergetic effects, and child mortality across 41 countries in sub-Saharan Africa and South Asia. While a similar previous work [26] focused solely on India using data from a single survey, our research spans 113 surveys conducted over 21 years, from 1998 to 2019. Additionally, our study investigates the potential synergistic effects of both indoor and outdoor air pollution on child mortality, aiming to reveal whether combined exposures have a more pronounced impact than individual ones. By encompassing a broad geographic scope and examining both outdoor and indoor air pollution, our work seeks to uncover patterns and long-term effects that provide valuable insights into the impact of air pollution on child health in these regions.

Methodology

The Integrated Public Use Microdata Series-Demographic and Health Surveys (IPUMS-DHS) dataset includes diverse observations collected from 41 countries across 113 surveys. Data spanning 21 years, from 1998 to 2019, were used in this study. The number of surveys representing each country varies, ranging from a minimum

of one survey (e.g., Togo) to a maximum of seven surveys (e.g., Senegal). This rich dataset provides insights into demographic and health surveys over two decades across diverse countries.

The outcome variable was under-five mortality rate, indicating mortality of children before five years of age. Children who died before age five were coded as 1, while children alive and under five at their mother's interview were coded as 0.

Outdoor air pollution was measured using annual mean PM2.5 concentrations obtained from the Socioeconomic Data and Applications Center (SEDAC) database.[1] The SEDAC dataset provides global annual PM2.5 grids from 1998 to 2018 at a 0.01-degree resolution, derived from satellite remote sensing data. Specifically, the Global Annual PM2.5 Grids from MODIS, MISR, and SeaWiFS consist of annual concentrations of fine particulate matter (PM2.5) in micrograms per cubic meter. This dataset combines aerosol optical depth (AOD) retrievals from multiple satellite algorithms, including MODIS, MISR, and SeaWiFS, and uses the GEOS-Chem model to relate these measures to near-surface PM2.5 concentrations. Adjustments for residual bias are made using geographically weighted regression (GWR) with global WHO ground-based data. The SEDAC GeoTIFF datasets were linked to displaced GPS coordinates of IPUMS-DHS survey clusters, anonymised by 0–2 km (urban) and 0–5 km (rural), connecting PM2.5 concentrations to survey cluster locations.

We standardised the PM2.5 variable to improve interpretability and address biases from regional variations in pollution exposure, ensuring our analysis reflects relative changes rather than absolute pollution levels. The unit of analysis is now a one standard deviation increase in PM2.5 concentration per cubic meter.

To align PM2.5 exposure data with the year preceding each child death, SEDAC PM2.5 data from one year before the mortality date were linked to each IPUMS-DHS cluster where a child death occurred. For censored cases, SEDAC PM2.5 data from the survey year were connected to IPUMS-DHS clusters. This chronological alignment preserved data integrity while allowing robust exploration of the research question.

Household air pollution (HAP) was constructed based on cooking fuel type and kitchen location from IPUMS-DHS data. The variable representing the type of cooking fuel was constructed by categorising the fuel sources into two distinct groups: solid cooking fuel and clean cooking fuel. Solid cooking fuels include sources like wood, charcoal, coal, and agricultural residues, while clean cooking fuels comprise electricity, LPG, biogas, and other petroleum-based or improved smokeless cook stoves. Missing data were excluded. Households using clean fuels were classified as 'No exposure.' For solid fuel, 'Moderate exposure' indicated a separate kitchen, while 'High exposure' meant no separate kitchen. This classified potential exposure to household air pollutants from cooking. A limitation is that HAP exposure level at the interview may not match the preceding 5 years. However, living standards often improve over time, suggesting if high exposure was reported at interview, past exposure was at least as high, if not worse. This presumption is supported by previous studies successfully using DHS data on household air pollution to assess its association with child mortality [27].

[1] https://sedac.ciesin.columbia.edu/data/set/sdei-global-annual-gwr-pm2-5-modis-misr-seawifs-aod-v4-gl-03/data-download.

Covariates

The analysis incorporated several covariates to capture multifaceted influences. In our analysis, we incorporated a comprehensive set of covariates, broadly categorized into three types: child-related, parent-related, and household-related factors. Child-related variables included parameters such as the child's size at birth and gender. We also considered parent-related factors like parental education and maternal employment status. On the household level, we looked at variables such as the total number of children born into the family and the household's wealth status.

Following similar literature [28], the covariates included 'Child Birth Size' (categorized as small, average, or large), 'Sex of Child' (classified as female or male), 'ANC Visits' (bifurcated into no visits, 1–4 visits, or 5+visits), 'Maternal Employment' (dichotomized as not working or working), and 'Mother's Current Age' (divided into less than 21, 18–34, or over 34 years). 'Woman's Current Marital Status' was differentiated as either married or not currently married, while 'Mother's Education' and 'Father's Education' were both classified into no education, primary, secondary, or higher education. 'Number of Children Ever Born' was grouped into three categories: 1–2, 3–4, or more than 4. Lastly, the 'Household Wealth Index' was stratified into five levels: poorest, poorer, middle, richer, or richest. Each of these covariates, with their respective levels, allowed a nuanced exploration of various factors potentially affecting child mortality.

Statistical analysis

Our estimation strategy comprises several steps. Initially, we identified variables that could potentially confound the relationship between air pollution and child mortality. Given that our outcome variable, child mortality, is binary, we utilized a logistic regression model. We incorporated spatial and temporal factors into our model for added context. Temporal factors were represented via an indicator variable spanning across the decades from 1998

to 2010 and 2011–2019. Likewise, we included a continent indicator variable to account for heterogeneity at the continental level. Decade-based temporal grouping smooths out short-term fluctuations and captures long-term trends, providing a more stable basis for analysis. Continent-level aggregation reduces the complexity of cross-country heterogeneity, highlighting regional patterns and facilitating comparisons across broader geographical areas.

To ensure the robustness of our findings, we conducted several checks, primarily by analyzing different subsets of the data. We broke down our data by continent and income groups, focusing specifically on low-income and lower-to-upper middle-income countries. It is important to note that our sample does not contain any high-income countries but encompasses countries primarily from South Asia and Africa. We further disaggregated our data from the beginning of the sample period up to 2010 and then created another category from 2011 to 2019. The inclusion of continent-level data was aimed at investigating how different continents might influence the relationship between air pollution and child mortality. Similarly, examining different income levels might reveal insights into the strategies various countries employ to mitigate the effects of air pollution.

As for the temporal variable, it is hypothesized that pollution has increased over time, which could raise the risk of child mortality. However, technological advancements over the same period could offset the pollution effect, possibly reducing the child mortality risk. Thus, to determine the net effect of air pollution on child mortality, we dissected our data into different continents, income groups, and decades. The covariates were included interactively into our model, with each set of factors incorporated into one model iteration at a time. This approach aimed to detect any shifts in the relationship between air pollution and child mortality and to ascertain the direction of these changes. By systematically integrating different sets of variables, we sought to thoroughly examine the intricate link between air pollution, various demographic factors, and child mortality.

Standard logistic regression models, while widely utilized in cross-country cross-sectional surveys [29], assume independence of observations, which may lead to biased estimates and underestimated standard errors in the presence of clustering and spatial-temporal correlations [30]. To ensure robustness in our analysis of under-five mortality across diverse settings, we complemented the initial weighted logistic regression with a multilevel logistic regression. This multilevel approach incorporates random intercepts at the Primary Sampling Unit (PSU) level, effectively accounting for intra-cluster correlations and unobserved heterogeneity within PSUs [31]. By including comprehensive covariates that capture geographical variability, we control for higher-level spatial and temporal variations within the fixed component of the model. Consequently, modeling random intercepts solely at the PSU level sufficiently addresses residual clustering and spatial-temporal correlations without overcomplicating the model structure [32, 33].

Finally, drawing upon the established literature [34, 35], a risk assessment was undertaken utilizing Population Attributable Fraction (PAF) analysis to quantify the proportion of under-five mortality attributable to ambient and household air pollution across 41 countries. PAF estimates the fraction of deaths that could be prevented by eliminating exposure to these pollutants. The method [36], we used Stata' *punaf* command after estimating logistic regression models to estimate proportion of under-five mortality attributable to air pollution. For ambient air pollution, the analysis expresses exposure in standard deviation units, with a value of 1 representing a one standard deviation increase from the mean PM2.5 levels. For household air pollution, the baseline scenario (Scenario 0) represents "no exposure," and both moderate and high exposure levels are interpreted relative to this baseline, quantifying their impact on mortality risk.

Results

Figure 1 gives the under-five child mortality rates per 1000 live births. We observe several intriguing patterns. The highest reported child mortality rate was in Mali in 2001, followed closely by Burkina Faso in 1998. In stark contrast, the lowest rate in Jordan in 2007 was recorded as 16. Most countries show a trend of decreasing child mortality rates over time, with Bangladesh presenting a strong downward trend. In contrast, Cameroon's mortality rate increased from 79 in 1998 to 95 in 2004 before dropping to 57 in 2018.

Figure 2 provides data on PM2.5 $\mu g/m^3$ (fine particulate matter with a diameter of 2.5 micrometres or less per cubic meter, which can penetrate deep into the lungs and even enter the bloodstream) and reveals several interesting patterns. The highest levels of PM2.5 were found in Bangladesh in 2014 and Nigeria in 2018. Conversely, Ethiopia in 2000 showed the lowest PM2.5 level. In some countries, like Bangladesh, there has been a noticeable upward trend, with the value rising from 48.2 in 2000 to 69.1 in 2014. Though Fig. 2 gives the estimates in $\mu g/m^3$ terms, we standardised the PM2.5 variable for regression analysis to improve interpretability and address biases from regional variations in pollution exposure, ensuring our analysis reflects relative changes rather than absolute pollution levels.

Figure 3 shows many countries have dramatically reduced the percentage of high household air pollution exposure over time, while others maintained or increased it. Bangladesh saw a significant reduction from 34.5% in

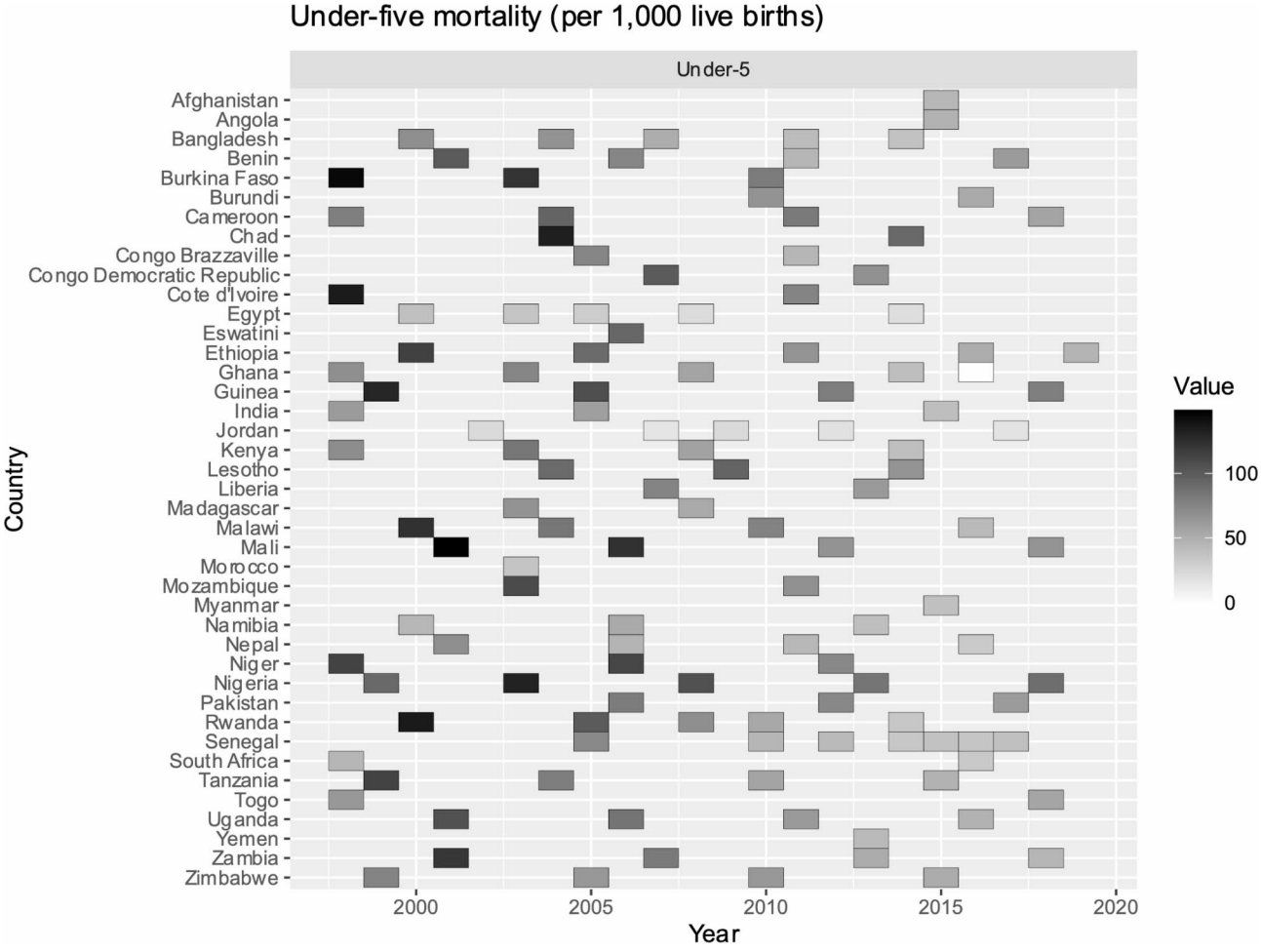

Fig. 1 Under-five child mortality (per 1,000 live births)

2004 to 8.5% in 2014. A stark contrast can be observed in Ethiopia, where the high exposure level skyrocketed from 0% in 2000 and 2005 to 83.9% in 2011. Notably, some countries consistently maintain 100% no exposure, potentially indicating successful interventions.

However, other factors such as geographical location, economic development, cultural practices, and urbanization can also influence these figures. Therefore, while the data provides valuable insights, it is essential to consider these context-specific factors when interpreting the results.

The pairwise correlation between L1 (lag of PM2.5 μg/m3 in a given geographical area) and the three categories of household air pollution exposure reveals interesting associations. L1 shows a negative correlation of -0.3137 with no exposure, suggesting that areas with higher PM2.5 lags tend to have less likelihood of no exposure to household air pollution. Conversely, L1 is positively correlated with high exposure at 0.2316, indicating that higher PM2.5 lags are associated with increased likelihood of high exposure to household air pollution. The correlation with moderate exposure is relatively weak at 0.0581.

Regression analysis

Across all models (Model 1–5 in Table 1), we consistently found that a one standard deviation increase in PM2.5 significantly elevates the risk of child mortality. The strength of this association remained relatively stable across the models, with some minor variations as additional covariates were introduced (Odds Ratio [OR]: 1.10; 95% Confidence Interval [CI]: 1.09–1.12; p-value < 0.001).

Regarding household air pollution (Model 6–10 in Table 1), we found that compared to no exposure, moderate exposure significantly heightened the risk of child mortality, with an approximately 91% increased risk (OR: 1.91; 95% CI: 1.82–2.00; p-value < 0.001). High exposure was associated with an even higher risk, increasing child mortality risk by around 102% (OR: 2.02; 95% CI: 1.93–2.10; p-value < 0.001). However, as more covariates were added, the association between moderate exposure and child mortality attenuated, remaining statistically

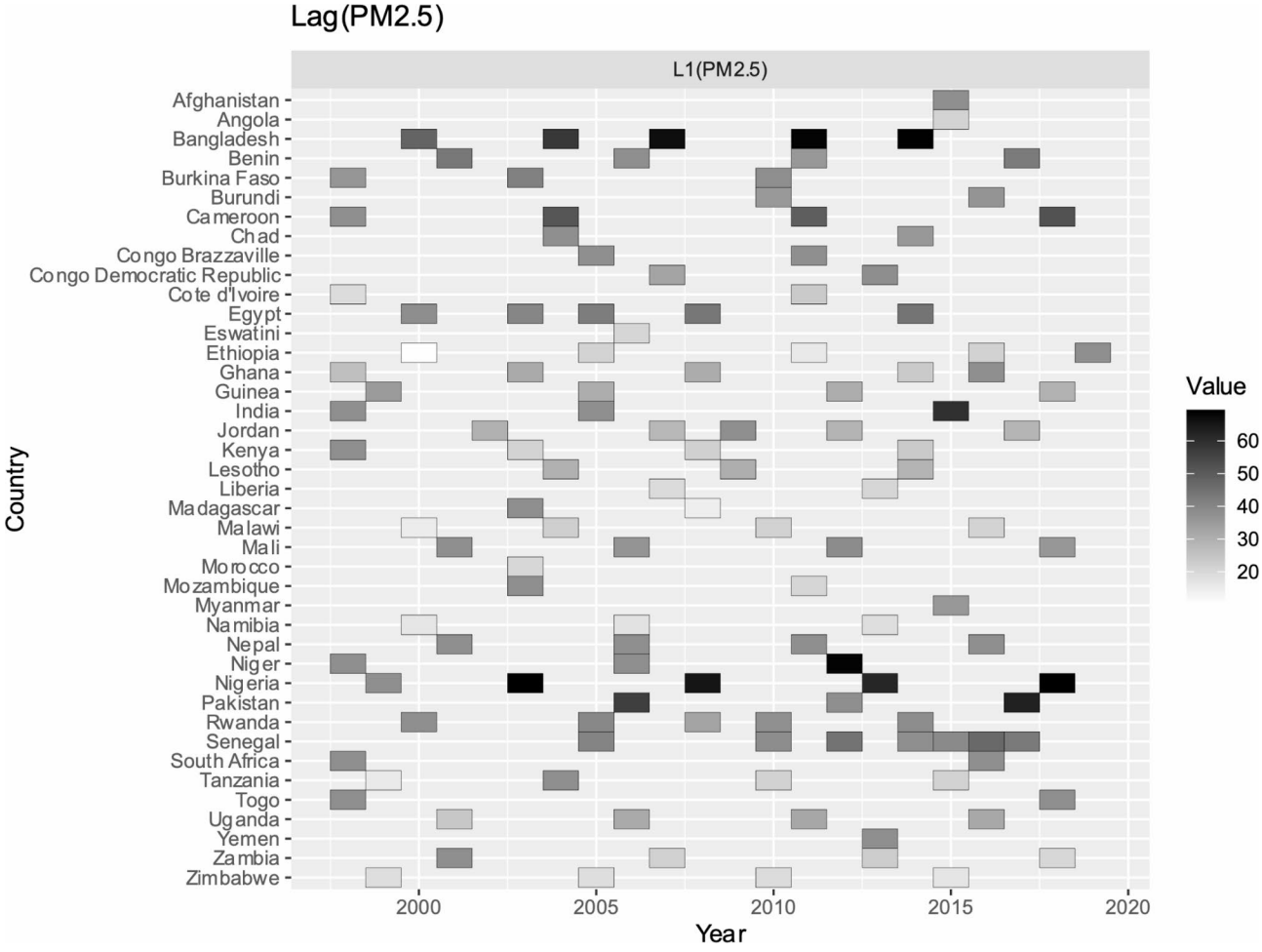

Fig. 2 Outdoor air pollution proxied by Lag ($PM_{2.5\ \mu g/m3}$)

significant though reduced in magnitude (OR: 1.21; 95% CI: 1.10–1.32; p-value < 0.001). Similarly, for high exposure, although the association weakened, it remained statistically significant (OR: 1.13; 95% CI: 1.03–1.24; p-value < 0.01), indicating a 13% increased risk of child mortality.

To ensure the robustness of our findings and address potential clustering and spatial-temporal correlations inherent in the survey data, we complemented our standard logistic regression models (Models 5 and 10) with multilevel logistic regression models (Models 5a and 10a in Table 1). These multilevel models incorporated random intercepts for Primary Sampling Units (PSUs), effectively accounting for unobserved heterogeneity and intra-cluster correlations within PSUs. The results from Models 5a and 10a closely mirrored those of their logistic regression counterparts, indicating minimal changes in the estimated associations. Specifically, Model 5a yielded an odds ratio (OR) of 1.15 (CI: 1.13–1.17; p < 0.001) for a one standard deviation increase in PM2.5, compared to Model 5's OR of 1.16 (95% CI: 1.14–1.18; p < 0.001). Regarding household air pollution, Model 10a reported an OR of 1.18 (95% CI: 1.10–1.27; p < 0.001) for high exposure, closely aligning with Model 10's OR of 1.18 (95% CI: 1.03–1.24; p < 0.001). These slight variations in coefficients between the logistic and multilevel models underscore the stability of our estimates.

Synergetic relation of indoor and outdoor air pollution

Our analysis (Table 2) reveals significant impacts of both outdoor and indoor air pollution on under-5 child mortality. A one standard deviation increase in PM2.5 consistently and significantly raises the mortality risk across all models (OR: 1.14; 95% CI: 1.10–1.18; p-value < 0.001). This effect remained robust, showing only minor variations as additional covariates were introduced.

For household air pollution, children in moderately exposed households experienced a significantly higher mortality risk, initially 91% greater than those in non-exposed households (OR: 1.91; 95% CI: 1.82–2.01; p-value < 0.001). While this association attenuated as more covariates were incorporated,

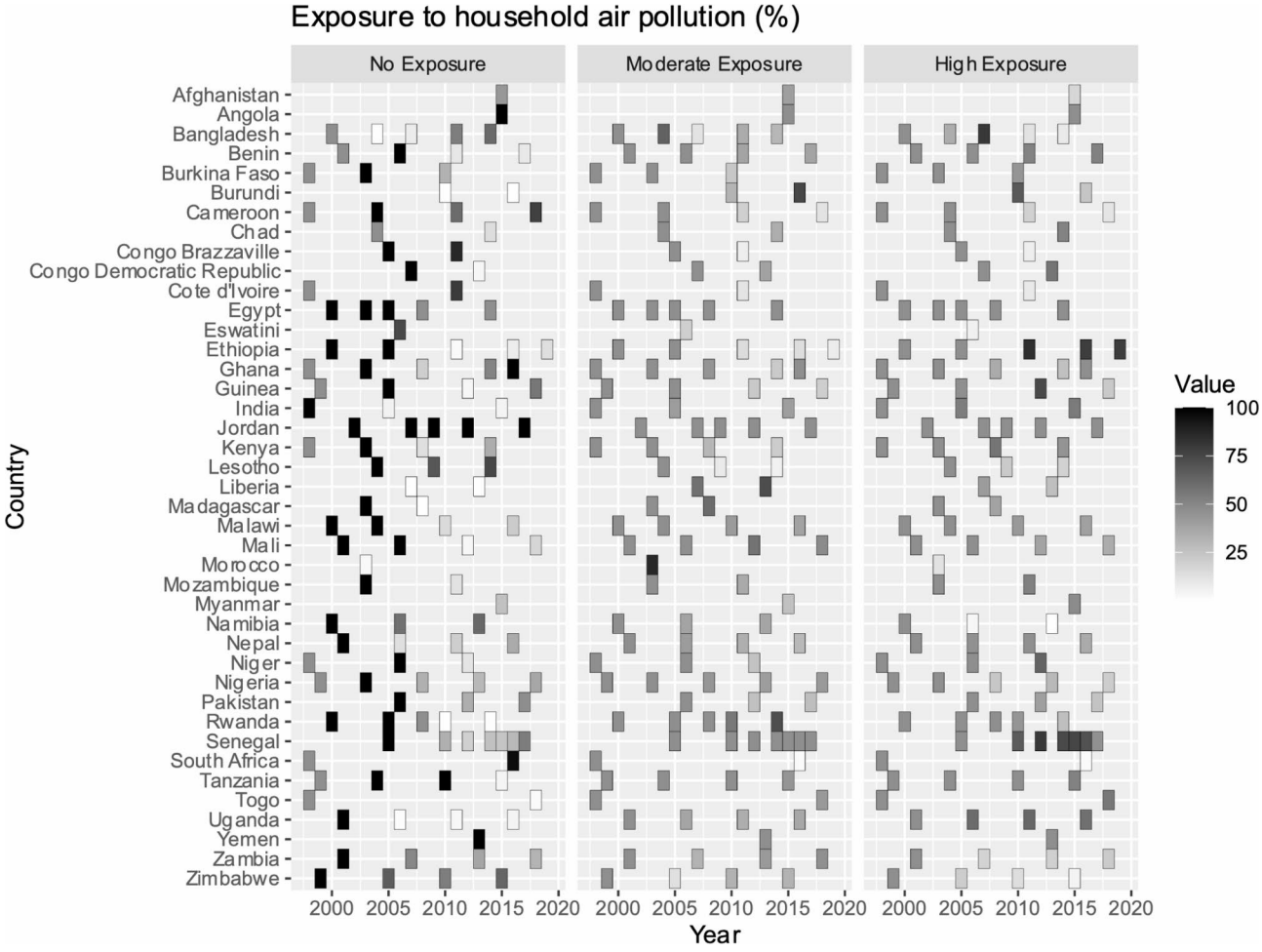

Fig. 3 Levels of exposure to household air pollution

it remained statistically significant in the final model, with a 37% increased risk (OR: 1.37; 95% CI: 1.24–1.53; p-value < 0.001).

Children in highly exposed households faced an even more substantial risk, initially showing a 120% increase in mortality risk (OR: 2.20; 95% CI: 2.10–2.32; p-value < 0.001). Though this risk decreased somewhat after adjusting for covariates, it persisted at 40% higher than in non-exposed households in the fully adjusted model (OR: 1.40; 95% CI: 1.26–1.56; p-value < 0.001).

The interaction effects between household air pollution and PM2.5 further highlight the complex dynamics. The interaction between no exposure to household air pollution and PM2.5 serves as the baseline (OR: 1.00; 95% CI: 1.00–1.00). For children in moderately exposed households, the interaction between indoor air pollution and PM2.5 was associated with a reduction in the odds of child mortality, with a 14% lower risk in the fully adjusted model (OR: 0.86; 95% CI: 0.78–0.95; p-value < 0.01). Similarly, in households with high exposure, the interaction with PM2.5 resulted in a 19% decrease in mortality risk (OR: 0.81; 95% CI: 0.73–0.89; p-value < 0.001).

To account for potential clustering and spatial-temporal correlations inherent in the survey data, we complemented our standard logistic regression models (Models 1–5) with a multilevel logistic regression model (Model 6). This multilevel model incorporated random intercepts for Primary Sampling Units (PSUs), effectively capturing unobserved heterogeneity and intra-cluster correlations within PSUs. As shown in Table 2, the estimates from Model 6 were largely consistent with those from the standard logistic regression models, indicating that the multilevel approach effectively accounts for residual clustering and spatial-temporal correlations without substantially altering the primary associations.

Disaggregated analysis

Spatial variability

Our analysis reveals significant regional and economic disparities in the association between air pollution and under-5 child mortality (Table 3). A one standard

Table 1 Association of PM2.5 and household air-pollution with under five child mortality

Under-5 child mortality	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(5a)**	**(6)**	**(7)**	**(8)**	**(9)**	**(10)**	**(10a)**
	OR	**OR**	**OR**	**OR**	**OR**	**OR**	**OR**	**OR**	**OR**	**OR**	**OR**	**OR**
$Z(PM2.5)_{t-1}$	1.10***	1.05***	1.15***	1.15***	1.16***	1.15***						
	[1.09,1.12]	[1.04,1.07]	[1.13,1.17]	[1.13,1.17]	[1.14,1.18]	[1.13,1.17]						
HH air pollution (Ref. No exposure)												
Moderate exposure							1.91***	1.54***	1.23***	1.20***	1.21***	1.22***
							[1.82,2.00]	[1.45,1.63]	[1.13,1.34]	[1.10,1.31]	[1.10,1.32]	[1.14,1.31]
High exposure							2.02***	1.56***	1.14**	1.12**	1.13**	1.18***
							[1.93,2.10]	[1.47,1.66]	[1.05,1.24]	[1.03,1.23]	[1.03,1.24]	[1.10,1.27]
Child birth size (Ref. Small)												
Average		0.59***	0.67***	0.67***	0.67***	0.67***		0.60***	0.66***	0.66***	0.65***	0.63***
		[0.57,0.62]	[0.64,0.70]	[0.64,0.70]	[0.64,0.70]	[0.64,0.70]		[0.56,0.63]	[0.61,0.71]	[0.61,0.71]	[0.61,0.70]	[0.59,0.66]
Large		0.81***	0.81***	0.81***	0.81***	0.79***		0.71***	0.72***	0.71***	0.72***	0.70***
		[0.77,0.84]	[0.77,0.85]	[0.77,0.85]	[0.77,0.85]	[0.76,0.83]		[0.66,0.76]	[0.66,0.78]	[0.66,0.77]	[0.66,0.78]	[0.66,0.75]
Sex of child (Ref. Female)												
Male		1.13***	1.20***	1.20***	1.20***	1.18***		1.06**	1.11***	1.12***	1.11***	1.12***
		[1.10,1.17]	[1.16,1.24]	[1.16,1.24]	[1.16,1.24]	[1.14,1.21]		[1.02,1.11]	[1.05,1.18]	[1.05,1.18]	[1.05,1.18]	[1.07,1.18]
ANC visits (Ref. No visits)												
1–4		0.76***	0.81***	0.82***	0.82***	0.79***		0.68***	0.66***	0.67***	0.68***	0.66***
		[0.73,0.79]	[0.77,0.85]	[0.78,0.86]	[0.78,0.87]	[0.76,0.83]		[0.64,0.71]	[0.62,0.71]	[0.63,0.72]	[0.63,0.73]	[0.62,0.70]
5+		0.61***	0.83***	0.84***	0.85***	0.80***		0.53***	0.65***	0.67***	0.68***	0.63***
		[0.58,0.64]	[0.78,0.88]	[0.80,0.89]	[0.80,0.90]	[0.76,0.84]		[0.50,0.57]	[0.60,0.71]	[0.61,0.73]	[0.62,0.74]	[0.59,0.68]
Maternal employment (Ref. Not working)												
Working			1.28***	1.27***	1.26***	1.26***			1.26***	1.25***	1.27***	1.27***
			[1.23,1.33]	[1.22,1.32]	[1.21,1.31]	[1.22,1.30]			[1.18,1.34]	[1.17,1.34]	[1.18,1.36]	[1.21,1.34]
Mother's current age (Ref. <21)												
18–34			0.67***	0.65***	0.66***	0.67***			0.71***	0.66***	0.66***	0.66***
			[0.63,0.70]	[0.61,0.69]	[0.62,0.70]	[0.63,0.70]			[0.65,0.78]	[0.60,0.73]	[0.59,0.72]	[0.61,0.71]
> 34			0.90***	0.82***	0.83***	0.85***			0.95	0.77***	0.77***	0.77***
			[0.85,0.96]	[0.76,0.88]	[0.76,0.89]	[0.79,0.90]			[0.86,1.05]	[0.68,0.88]	[0.68,0.88]	[0.70,0.86]
Woman's current marital status (Ref. Married)												
Not currently married			1.73***	1.74***	1.72***	1.70***			2.01***	2.05***	2.06***	1.93***
			[1.61,1.85]	[1.62,1.86]	[1.61,1.85]	[1.61,1.80]			[1.78,2.26]	[1.82,2.31]	[1.83,2.32]	[1.75,2.14]
Mother's education (Ref. No Education)												
Primary			1.04	1.04+	1.05+	1.02			0.93+	0.95	0.96	0.98
			[0.99,1.09]	[0.99,1.10]	[0.99,1.10]	[0.98,1.06]			[0.86,1.01]	[0.87,1.03]	[0.88,1.04]	[0.92,1.05]
Secondary			0.73***	0.75***	0.76***	0.78***			0.69***	0.72***	0.73***	0.79***
			[0.68,0.78]	[0.70,0.81]	[0.71,0.81]	[0.74,0.83]			[0.62,0.76]	[0.65,0.79]	[0.66,0.80]	[0.73,0.85]
Higher			0.50***	0.53***	0.54***	0.58***			0.50***	0.55***	0.56***	0.60***
			[0.43,0.58]	[0.46,0.62]	[0.46,0.62]	[0.52,0.65]			[0.42,0.60]	[0.46,0.65]	[0.46,0.67]	[0.53,0.68]

Table 1 (continued)

Under-5 child mortality	(1)	(2)	(3)	(4)	(5)	(5a)	(6)	(7)	(8)	(9)	(10)	(10a)
	OR	OR	OR	OR	OR	OR	OR	OR	OR	OR	OR	OR
Father's education (Ref. No Education)												
Primary			1.01	1.01	1.01	0.98			1.04	1.04	1.04	0.96
			[0.96,1.07]	[0.96,1.07]	[0.96,1.07]	[0.94,1.02]			[0.95,1.13]	[0.96,1.14]	[0.95,1.13]	[0.90,1.03]
Secondary			0.90***	0.91**	0.92**	0.90***			0.97	0.98	0.96	0.94+
			[0.84,0.95]	[0.86,0.96]	[0.86,0.97]	[0.85,0.94]			[0.89,1.05]	[0.90,1.07]	[0.88,1.05]	[0.88,1.01]
Higher			0.80***	0.82***	0.83***	0.79***			0.91	0.93	0.91	0.84**
			[0.71,0.89]	[0.73,0.92]	[0.74,0.93]	[0.72,0.86]			[0.78,1.05]	[0.80,1.07]	[0.79,1.05]	[0.76,0.94]
Number of children ever born (Ref. <3)												
3–4				0.98	0.98	0.97				1.07+	1.08+	1.09**
				[0.93,1.04]	[0.93,1.03]	[0.93,1.01]				[0.99,1.16]	[1.00,1.17]	[1.02,1.15]
>4				1.14***	1.13***	1.12***				1.31***	1.32***	1.34***
				[1.07,1.21]	[1.06,1.20]	[1.07,1.18]				[1.19,1.44]	[1.20,1.46]	[1.25,1.44]
Household wealth index (Ref. Poorest)												
Poorer				1.03	1.03	1.02				1.00	1.00	1.04
				[0.98,1.08]	[0.98,1.08]	[0.98,1.07]				[0.92,1.08]	[0.92,1.09]	[0.98,1.11]
Middle				1.01	1.01	1.01				1.00	1.00	1.06
				[0.96,1.07]	[0.96,1.07]	[0.96,1.06]				[0.91,1.10]	[0.91,1.10]	[0.98,1.14]
Richer				0.99	0.99	1.02				1.04	1.04	1.11**
				[0.94,1.05]	[0.93,1.05]	[0.97,1.07]				[0.94,1.15]	[0.95,1.15]	[1.03,1.20]
Richest				0.92*	0.92*	0.93*				0.96	0.96	1.11*
				[0.86,0.99]	[0.85,0.98]	[0.87,0.98]				[0.86,1.06]	[0.86,1.07]	[1.02,1.21]
Decade (Ref. 1998–2010)												
2011–2019					0.96*	0.94***					0.92**	0.93**
					[0.92,1.00]	[0.91,0.97]					[0.87,0.98]	[0.89,0.98]
Continent (Ref. Africa)												
Asia					0.92*	0.91**					1.08*	1.06*
					[0.86,0.99]	[0.86,0.97]					[1.01,1.15]	[1.00,1.11]
var(_cons[idhspsu])						1.42***						1.26***
						[1.37,1.47]						[1.20,1.33]
N	1,093,935	736,179	458,714	458,714	458,714	482,251	623,819	415,108	237,267	237,267	237,267	239,731
F (Wald χ^2 in Model 5a and 10a)	289.71	249.75	151.34	112.66	104.01	3140.4	592.89	206.11	82.80	63.66	59.22	1986.87
p	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

Exponentiated coefficients; 95% confidence intervals in brackets

+$p<0.10$, *$p<0.05$, **$p<0.01$, ***$p<0.001$

Table 2 Synergetic relationship between indoor air pollution and outdoor air pollution

Under-5 child mortality	(1)	(2)	(3)	(4)	(5)	(6)
	OR	OR	OR	OR	OR	OR
$Z(PM2.5)_{t-1}$	1.14***	1.10***	1.29***	1.29***	1.29***	1.29***
	[1.10,1.18]	[1.05,1.15]	[1.19,1.39]	[1.19,1.39]	[1.19,1.40]	[1.21,1.37]
HH air pollution (Ref. No exposure)						
Moderate exposure	1.91***	1.66***	1.39***	1.38***	1.37***	1.38***
	[1.82,2.01]	[1.55,1.78]	[1.26,1.53]	[1.25,1.53]	[1.24,1.53]	[1.27,1.50]
High exposure	2.20***	1.83***	1.41***	1.41***	1.40***	1.39***
	[2.10,2.32]	[1.70,1.96]	[1.28,1.56]	[1.27,1.56]	[1.26,1.56]	[1.27,1.52]
HH air pollution x $Z(PM2.5)_{t-1}$						
No exposure x $Z(PM2.5)_{t-1}$	1.00	1.00	1.00	1.00	1.00	1.00
	[1.00,1.00]	[1.00,1.00]	[1.00,1.00]	[1.00,1.00]	[1.00,1.00]	[1.00,1.00]
Moderate exposure x $Z(PM2.5)_{t-1}$	1.05*	1.01	0.87**	0.86**	0.86**	0.87***
	[1.01,1.10]	[0.95,1.07]	[0.79,0.95]	[0.79,0.94]	[0.78,0.95]	[0.81,0.93]
High exposure x $Z(PM2.5)_{t-1}$	0.91***	0.92**	0.81***	0.81***	0.81***	0.83***
	[0.87,0.95]	[0.87,0.98]	[0.74,0.88]	[0.73,0.89]	[0.73,0.89]	[0.77,0.89]
var(_cons[idhspsu])						1.34***
						[1.25,1.45]
Child-related covariates	No	Yes	Yes	Yes	Yes	Yes
Parents-related covariates	No	No	Yes	Yes	Yes	Yes
Family-related covariates	No	No	No	Yes	Yes	Yes
Decade fixed effects (Ref. 1998–2010)	No	No	No	No	Yes	Yes
Continental fixed effects (Ref. Africa)	No	No	No	No	Yes	Yes
N	490,589	331,355	165,702	165,702	165,702	168,163
F (Wald χ^2 in Model 6)	294.93	148.90	69.71	54.58	50.81	1040.08
p	0.00	0.00	0.00	0.00	0.00	0.00

OR: Odds Ratios; 95% confidence intervals in brackets

+$p<0.10$, *$p<0.05$, **$p<0.01$, ***$p<0.001$

Child-related covariates in the models were childbirth size, sex of child, ANC visits. Parents-related covariates included maternal employment, mother's current age, woman's current marital status, mother's education, father's education. Family-related covariates included number of children ever born and household wealth index

deviation increase in PM2.5 consistently and significantly raises child mortality risk across all models (OR: 1.005; 95% CI: 1.004–1.006; *p*-value < 0.001). This effect remains significant in both Africa (OR: 1.005; 95% CI: 1.004–1.006; *p*-value < 0.001) and Asia (OR: 1.003; 95% CI: 1.001–1.006; *p*-value < 0.05), although the association is slightly weaker in Asia.

For household air pollution, moderate exposure increases the risk of child mortality by 23% compared to no exposure (OR: 1.230; 95% CI: 1.134–1.334; *p*-value < 0.001). This effect is more pronounced in Africa, where moderate exposure is associated with a 28% increased risk (OR: 1.278; 95% CI: 1.165–1.402; *p*-value < 0.001), while in Asia, moderate exposure shows no significant association with child mortality (*p*-value > 0.05).

High household air pollution exposure results in a 25.3% increased risk of under-5 mortality in the overall model (OR: 1.253; 95% CI: 1.154–1.361; *p*-value < 0.001). In Africa, this risk is slightly lower but still significant (OR: 1.224; 95% CI: 1.113–1.347; *p*-value < 0.001), whereas in Asia, high exposure shows a marginally significant 14% increased risk (OR: 1.140; 95% CI: 0.979–1.327; *p*-value < 0.10).

When disaggregating by income groups, the association between PM2.5 and child mortality is strong in low-income countries (OR: 1.005; 95% CI: 1.004–1.006; *p*-value < 0.001) and somewhat weaker but still significant in lower and upper-middle-income countries (OR: 1.004; 95% CI: 1.000–1.008; *p*-value < 0.10).

In terms of household air pollution, the effect of moderate exposure is not significant in low-income countries but sharply rises to a 40% increased risk in lower and upper-middle-income countries (OR: 1.400; 95% CI: 1.159–1.692; *p*-value < 0.001). High exposure to household air pollution is similarly insignificant in low-income countries but dramatically increases mortality risk by nearly 60% in lower and upper-middle-income nations (OR: 1.593; 95% CI: 1.335–1.901; *p*-value < 0.001).

Temporal variability

We also explored temporal variations in the association between air pollution and under-5 child mortality, splitting the data into two periods: 1989–2010 and

Table 3 Association between air pollution and under five child mortality: a spatial and economic disaggregation

	Continent						Income group					
	PM2.5			HAP			PM2.5			HAP		
Under-5 child mortality	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	OR	OR	OR	OR	OR	OR	OR	OR	OR	OR	OR	OR
	Full	Africa	Asia	Full	Africa	Asia	Full	LI	LUM	Full	LI	LUM
$Z(PM2.5)_{t-1}$	1.11***	1.12***	1.07*				1.11***	1.12***	1.09+			
	[1.10,1.13]	[1.10,1.14]	[1.02,1.13]				[1.10,1.13]	[1.10,1.14]	[1.00,1.18]			
HH air pollution (Ref. No exposure)												
Moderate exposure				1.28***	1.44***	0.97				1.28***	1.19***	1.36***
				[1.19,1.38]	[1.30,1.60]	[0.85,1.10]				[1.19,1.38]	[1.10,1.29]	[1.13,1.64]
High exposure				1.29***	1.36***	1.11				1.29***	1.17***	1.58***
				[1.20,1.39]	[1.23,1.51]	[0.98,1.25]				[1.20,1.39]	[1.07,1.26]	[1.33,1.87]
Child-related covariates	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Parents-related covariates	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Family-related covariates	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Decade fixed effects (Ref. 1998–2010)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Continental fixed effects (Ref. Africa)	No	No	No	No	No	No	No	No	No	No	No	No
N	717,359	636,689	80,670	378,148	229,076	149,072	717,359	644,565	72,794	378,148	264,763	113,385
F	164.71	147.28	36.10	73.01	56.62	35.37	164.71	144.70	36.75	73.01	60.71	27.88
p	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00

OR: Odds Ratios; 95% confidence intervals in brackets

$+p<0.10$, $^{*}p<0.05$, $^{**}p<0.01$, $^{***}p<0.001$

Note: LI indicates Low Income countries, LUM indicates Lower- and middle-income countries

Child-related covariates in the models were childbirth size, sex of child, ANC visits. Parents-related covariates included maternal employment, mother's current age, woman's current marital status, mother's education, father's education. Family-related covariates included number of children ever born and household wealth index

2010–2019 (Table 4). In the comprehensive model, a one standard deviation increase in PM2.5 was significantly associated with a higher risk of child mortality (OR: 1.16; 95% CI: 1.15–1.18; p-value < 0.001). However, no significant association was found for the pre-2010 period (OR: 0.99; 95% CI: 0.96–1.03; p-value > 0.05). Post-2010, the association became significant again, with a one standard deviation increase in PM2.5 associated with a 10% higher risk of child mortality (OR: 1.10; 95% CI: 1.06–1.13; p-value < 0.001).

For household air pollution, children born into moderately exposed households exhibited a 46% greater likelihood of dying before their fifth birthday compared to those from non-exposed households in the full model (OR: 1.46; 95% CI: 1.37–1.55; p-value < 0.001). Before 2010, this association was insignificant (OR: 1.00; 95% CI: 0.88–1.12; p-value > 0.05). Post-2010, moderate exposure did not show a statistically significant association with child mortality (OR: 1.03; 95% CI: 0.91–1.15; p-value > 0.05).

Children from households with high exposure to indoor air pollution demonstrated a 37% higher risk of child mortality in the full model (OR: 1.37; 95% CI: 1.29–1.46; p-value < 0.001). In the pre-2010 period, the association was insignificant (OR: 1.02; 95% CI: 0.90–1.16; p-value > 0.05). However, post-2010, high exposure was associated with an 11% higher risk of child mortality, although this association was only marginally significant (OR: 1.11; 95% CI: 0.99–1.24; p-value < 0.10).

The analysis presented in the Table 5 explores the relationship between ambient air pollution, specifically PM2.5, and household air pollution (HH Air Pollution) at moderate and high exposure levels, in relation to under-five mortality. The results are provided in terms of Population Unattributable Fractions (PUF) and Population Attributable Fractions (PAF), which quantify the burden of child mortality that can be attributed to these environmental risk factors. The baseline mortality probability is 0.034. Increasing ambient PM2.5 raises mortality to 0.038, with a PUF of 1.119 and a PAF of −0.119, indicating that reducing PM2.5 by one standard deviation could prevent 11.9% of under-five deaths (CI: −0.157 to −0.081). Similarly, moderate HH Air Pollution increases mortality to 0.038 (PUF = 1.120, PAF = −0.120), potentially preventing 12.0% of deaths (CI: −0.178 to −0.063). High HH Air Pollution results in a PUF of 1.121 and a PAF of −0.121, suggesting that eliminating high household pollution could prevent 12.1% of under-five deaths (CI: −0.178 to −0.066). All PAF estimates are statistically significant.

Discussion

Our research identified significant association between air pollution and the under-five child mortality, consistently revealing a positive connection between PM2.5 μg/

Table 4 Association between air pollution and under five child mortality: a temporal disaggregation

	PM2.5			HAP		
	(1)	(2)	(3)	(4)	(5)	(6)
Under-5 child mortality	**OR**	**OR**	**OR**	**OR**	**OR**	**OR**
	Full	**Pre-2010**	**Post-2010**	**Full**	**Pre-2010**	**Post-2010**
$Z(PM2.5)_{t-1}$	1.16***	0.99	1.10***			
	[1.15,1.18]	[0.96,1.03]	[1.06,1.13]			
HH air pollution (Ref. No exposure)						
Moderate exposure				1.46***	1.00	1.03
				[1.37,1.55]	[0.88,1.12]	[0.91,1.15]
High exposure				1.37***	1.02	1.11+
				[1.29,1.46]	[0.90,1.16]	[0.99,1.24]
Child-related covariates	Yes	Yes	Yes	Yes	Yes	Yes
Parents-related covariates	Yes	Yes	Yes	Yes	Yes	Yes
Family-related covariates	Yes	Yes	Yes	Yes	Yes	Yes
Decade fixed effects (Ref. 1998–2010)	No	No	No	No	No	No
Continental fixed effects (Ref. Africa)	Yes	Yes	Yes	Yes	Yes	Yes
N	717,359	312,389	386,017	378,148	166,871	211,273
F	263.98	74.51	66.33	145.15	38.04	33.47
p	0.00	0.00	0.00	0.00	0.00	0.00

OR: Odds Ratios; 95% confidence intervals in brackets

+p < 0.10, *p < 0.05, **p < 0.01, ***p < 0.001

Child-related covariates in the models were childbirth size, sex of child, ANC visits. Parents-related covariates included maternal employment, mother's current age, woman's current marital status, mother's education, father's education. Family-related covariates included number of children ever born and household wealth index

Note: Pre-2010 period indicates the surveys done from 1998 to 2010 and Post-2010 Period indicates the surveys done after 2010 till 2019

Table 5 Proportion of under-five child mortality attributable to air pollution

Variables	Scenario	Mean/Ratio	Std. Err.	t-value	*P*-value	95% CI	PAF Estimate
Z(PM2.5)	Scenario 0	0.034	0.001	-212.43	0.000	0.033 to 0.035	
Z(PM2.5)	Scenario 1	0.038	0.001	-135.78	0.000	0.036 to 0.040	
Z(PM2.5)	PUF	1.119	0.019	6.46	0.000	1.081 to 1.157	
Z(PM2.5)	PAF					-0.157 to -0.081	-0.119
HH Air Pollution (Moderate Exposure)	Scenario 0	0.034	0.001	-212.430	0.000	0.033 to 0.035	
HH Air Pollution (Moderate Exposure)	Scenario 1	0.038	0.001	-107.980	0.000	0.036 to 0.041	
HH Air Pollution (Moderate Exposure)	PUF	1.120	0.030	4.290	0.000	1.063 to 1.179	
HH Air Pollution (Moderate Exposure)	PAF					-0.178 to -0.063	-0.120
HH Air Pollution (High Exposure)	Scenario 0	0.034	0.001	-212.43	0.000	0.033 to 0.035	
HH Air Pollution (High Exposure)	Scenario 1	0.038	0.001	-110.18	0.000	0.036 to 0.041	
HH Air Pollution (High Exposure)	PUF	1.121	0.029	4.44	0.000	1.066 to 1.178	
HH Air Pollution (High Exposure)	PAF					-0.178 to -0.066	-0.121

Note: HH: household; PUF: population Unattributable Fractions; PAF: Population Attributable Fractions; Slightly different PUFs for Moderate and High exposure within identical scenarios (Scenario 0 and Scenario 1) are due to restricting scenario estimates to three decimal points

m3 exposure and under-five child mortality. Our results corroborate previous studies, which show PM2.5 μg/m3 exposure is a significant risk factor for children under five in various regions [37–41]. PM2.5 μg/m3 can cause numerous risks to children under five through different causal mechanisms, leading to both direct and indirect health complications. PM2.5 μg/m3 can increase child mortality by worsening respiratory and cardiovascular conditions, affecting lung functionality, compromising immunity, and negatively affecting prenatal outcomes [42–45]. The connection between PM2.5 μg/m3 exposure and respiratory problems in under-five children plays a significant role in increasing child mortality. One study revealed a significant relationship between annual PM2.5 μg/m3 exposure and acute respiratory infection (ARI) in children under five years old in developing countries [44]. The World Health Organization (WHO) issued a damning report stating that almost all children worldwide are exposed to harmful air pollution levels, leading to many deaths from stroke, lung cancer, and heart disease. The situation is particularly alarming in low- and middle-income countries, where 98% of all under-five children are exposed to PM2.5 μg/m3 levels exceeding WHO air quality guidelines [46]. These studies highlight the crucial connection between PM2.5 μg/m3 exposure, respiratory problems in under-five children, and increased child mortality rates.

PM2.5 μg/m3 exposure is associated with an increased risk of cardiovascular diseases, a link that is particularly significant for children under five, potentially contributing to higher child mortality rates. A study by the United States Environmental Protection Agency highlights that the harmful effects of PM2.5 μg/m3 encompass both short-term and long-term health outcomes, including changes to heart and vascular function, systemic inflammation, oxidative stress, and impaired vascular function that may develop type 2 diabetes [47–51]. These changes can lead to severe conditions such as heart attacks, strokes, and abnormal heart rhythms. Although these effects are primarily documented in adults, it is inferred that children, especially those with pre-existing cardiovascular conditions, could be similarly impacted. These factors, combined with the higher susceptibility of children, could link PM2.5 μg/m3 exposure to increased child mortality rates, warranting further research on this critical public health issue. A study directly links indoor air pollution, including PM2.5 μg/m3 to acute lower respiratory infections in children, a leading cause of under-five mortality. Study is evidence of the health impacts of household air pollution, including PM2.5 μg/m3 in China, a country with a high burden of under-five mortality [51, 52]. Evidence reveals a strong correlation between PM2.5 μg/m3 exposure and adverse pregnancy outcomes like low birth weight and preterm birth, impacting all geographic regions in the U.S. and suggesting a higher risk for pregnant women exposed to high PM2.5 μg/m3 levels [43]. These conditions extend their effects beyond the neonatal period, significantly raising the risk of early childhood mortality and potential developmental disabilities [53]. As a study demonstrated, PM2.5 μg/m3 exposure is directly associated with under-five mortality, with a 10-unit increase in ambient PM2.5 μg/m3 leading to 2.29 times higher odds of under-five mortality [54].

Household air pollution, specifically at high exposure levels, significantly amplified this risk, a relationship that held even when controlling for numerous covariates. Our findings are consistent with the previous evidence [7, 55, 56]. Household air pollution (HAP) from solid fuel use (SFU) in cooking, prevalent in low and middle-income countries (LMICs), leads to high concentrations of harmful indoor pollutants. These pollutants, including particulate matter, NOx, CO, SOx, formaldehyde, and toxic polycyclic aromatic hydrocarbons, often exceed WHO guidelines and pose a significant health risk [27]. Cultural practices mean women and children spend significant

time near the source of these pollutants, exposing them to harmful levels in LMICs. A study in Myanmar found that infant and under-five mortality risks were higher in households using SFU, demonstrating a direct causal link between HAP and child mortality [27].

Both outdoor pollution and indoor air pollution demonstrate individually significant positive effects on child mortality, but their synergetic effect appears negative. This finding can be understood through several theoretical frameworks in the literature. Ai, et al. [57] suggest that in nonlinear models such as logistic regression, interaction terms often capture complex relationships rather than simple additive effects. The negative interaction between outdoor and indoor pollution suggests diminishing marginal returns. In cases where exposure to outdoor pollution is already high, the additional impact of indoor pollution on child mortality may be less pronounced, reflecting a plateau in the combined effect of these stressors.

Furthermore, study [58] highlight that negative synergy, or antagonism, occurs when multiple stressors interact in ways that diminish their individual impacts. In this case, while both outdoor and indoor pollution independently elevate the risk of child mortality, their combined effect may be weaker than the sum of their parts. This negative synergism could account for the observed negative interaction term in the analysis of child mortality. A study [59] discuss how systems exposed to multiple stressors may adapt or compensate, resulting in a reduced cumulative effect. Households exposed to both indoor and outdoor pollution may take protective actions, such as improving ventilation, which could mitigate the overall risk of mortality. This adaptive behaviour may contribute to the negative interaction effect observed between the two pollutants.

Our disaggregated analysis revealed crucial geographical patterns, showing stronger association between air pollution and under-five child mortality in Africa as opposed to Asia. Though no specific study explains why the child mortality rate associated with PM2.5 μg/m3 is higher in Africa compared to Asia, some research provides possible reasons. A Nature publication found a strong link between air quality and infant mortality in Africa. Here, a 10 μg m−3 rise in PM2.5 μg/m3 concentration is associated with a 9% increase in infant mortality. This effect has remained consistent over the past 15 years and does not reduce with growing household wealth [26]. Socioeconomic elements, including education, employment status, health expenditure, improved water and sanitation facilities accessibility, and income inequality, significantly influence child mortality. Research in South Asia and Latin America demonstrates negative association between education, improved water and sanitation facilities, health expenditure, and child mortality. Conversely, unemployment and income inequality increase the risk of child mortality [60, 61]. The State of Global Air report indicates that Asian and African counties experience the most deaths related to PM2.5 μg/m3. Factors like healthcare accessibility, socioeconomic development, and population changes affect these trends. Notably, even with reduced air pollution exposure, growing population numbers can increase the overall disease burden. This factor might explain higher child mortality in Africa [62]. While the studies cited above do not offer a direct Africa-Asia comparison, they imply that differing socioeconomic conditions, healthcare access, and population dynamics could contribute to the observed differences in child mortality in both the continents.

Income level stratification echoed these results, with similar relationships observed in low-income countries and the overall model but with a weaker association in lower and upper-middle-income countries. The observed associations between outdoor (like PM2.5 μg/m3) and indoor air pollution and under-five child mortality, which are more robust in low-income countries, could be due to several factors. Higher exposure to pollutants can contribute to respiratory and other health conditions that risk child mortality, particularly in lower-income countries where indoor air pollution from cooking fuels is prevalent [26]. Socioeconomic conditions such as less access to healthcare and higher risk factors like malnutrition and infectious diseases may also heighten this association in these countries [60, 61]. Moreover, in higher-income countries, better access to medical treatments can potentially mitigate the health impacts of pollution, reducing its contribution to mortality rates4 . Additionally, a country's demographic profile, including a rapidly growing population or a higher proportion of young children, could influence the overall burden of child deaths attributable to pollution [62].

The temporal analysis suggested an intensification of associations post-2010, potentially reflecting increased pollution levels in recent years. This might indicate the compounded effect of persistent indoor and escalating outdoor air pollution. Our analysis validates existing studies confirming that in-house and outer environmental pollution significantly elevate the risk of under-five child mortality, a link that does not wane over time or with increased household wealth [26]. The intensification of this association post-2010 might stem from increased air pollution levels, particularly in populous countries like China and India, and regions including South Asia, Southeast Asia, East Asia, Oceania, Sub-Saharan Africa, North Africa, and the Middle East [61]. Factors like healthcare access, medical treatments, socioeconomic development, and demographic shifts can influence the disease burden from PM2.5 μg/m3 [61]. Socioeconomic

elements, like education, access to safe drinking water and hygiene, unemployment, and income inequality, also profoundly impact child mortality, with more marked differentials in Latin America than in developed countries [60, 62]. While data on indoor air pollution's specific impacts was unavailable during our discussion, the well-known health risks associated with solid fuel use suggest its notable contribution. Consequently, mitigating under-five child mortality necessitates addressing both types of air pollution and persistent socioeconomic disparities. To mitigate this issue, policy interventions should aim to enhance indoor ventilation, promote efficient household fuel use, and manage outdoor air pollution. These measures are particularly vital for impoverished regions and households.

Conclusion

Our study underscores the significant impact of both household (indoor) and environmental (outdoor) air pollution on under-five child mortality, with PM2.5 as a key indicator of outdoor pollution. Utilizing both standard logistic regression and multilevel logistic regression with random intercepts for Primary Sampling Units (PSUs), we found that a one standard deviation increase in PM2.5 consistently elevated the risk of child mortality. Similarly, higher levels of household air pollution significantly increased mortality risk. Population Attributable Fraction (PAF) analysis further revealed that approximately 12% of under-five deaths could be prevented by reducing ambient PM2.5 and household air pollution. While the combined effects of indoor and outdoor pollution contribute to increased mortality risk, interactions between these pollutants exhibit complex dynamics. The diverse geographical, climatic, and socioeconomic conditions across the 41 developing countries studied may influence these associations. Although PM2.5 is a crucial marker for outdoor pollution, it may not capture all pollutants affecting child mortality.

These findings suggest that policies should prioritize improving indoor air quality through better ventilation and efficient household fuels, alongside stricter regulation of outdoor pollutants. Continued monitoring of pollution levels, especially post-2010, is essential for mitigating ongoing threats to child health.

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Author contributions
RAD and RK conceptualized the study. MK supervised NN data analysis and reviewed final paper. All authors contributed to the data interpretation, revised the manuscript critically for important intellectual content, and finally approved the work.

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Data availability
IPUMS-DHS dataset set is publicly available online; https://www.idhsdata.org/idhs/.

Declarations

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Not applicable.

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Competing interests
The authors declare no competing interests.

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