

ARTICLE



In-utero exposure to multiple air pollutants and childhood undernutrition in India

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BACKGROUND: Several studies have been conducted to understand the impact of socioeconomic and maternal factors on child undernutrition. However, the past literature has not directly examined the joint impacts of fuel use and ambient pollution and have primarily focused on PM_{2.5}.

OBJECTIVE: This study explored the individual and community-level associations of both indoor (cooking fuel type) and ambient air pollution (PM_{2.5}, NO₂ and SO₂) during maternal gestation on child undernutrition.

METHODS: This study analysed stunting, being underweight, and anaemia of children aged 0–59 months ($n = 259,627$) using the National Family Health Survey. In-utero exposures to ambient PM_{2.5}, NO₂, and SO₂ were measured using satellite data and self-reported fuel type was a marker of indoor pollution exposure. The study used univariate and bivariate Moran's I, spatial lag model and multivariable logistic regression models after adjusting for other covariates to understand the effect of pollution on in-utero exposure and child health status at the individual and community-levels.

RESULTS: Higher concentration of indoor and ambient air pollution was found in the Northern and parts of Central regions of India. Estimates of spatial modelling show that each 1 $\mu\text{g}/\text{m}^{-3}$ increase in maternal exposure to ambient PM_{2.5} across the clusters of India was associated with a 0.11, 9 and 19 percentage points increase in the prevalence of stunting, underweight and anaemia, respectively. The results of multi-pollutant model show that a higher ambient PM_{2.5} exposure during pregnancy was linked to higher odds of stunting (AOR:1.38; 95% CI:1.32–1.44), underweight (AOR:1.59; 95% CI:1.51–1.67) and anaemia (AOR:1.61; 95% CI:1.52–1.69) in children. Weaker but similar associations were observed for NO₂, but not with SO₂. Indoor pollution exposure during in-utero periods was also significantly associated with childhood undernutrition and this association was modified by ambient PM_{2.5} levels, where exposure to both indoor and ambient air pollution had even greater odds of being undernourished.

IMPACT STATEMENT:

- Our research on multi-pollutant models has revealed the initial proof of the individual impacts of indoor and outdoor pollution (PM_{2.5}, NO₂, and SO₂) exposure during fetal development on children's nutrition.

Keywords: Ambient air pollution; Indoor air pollution; Undernutrition; In-utero exposure; Multipollutants; India

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INTRODUCTION

Almost half of deaths among children under the age of five are due to undernutrition [1], making this a crucial global public health issue. Undernutrition at an early age affects brain development [2] and immune function [3] resulting in lower educational attainment, later cognitive impairment [4] and increased risks of infection and chronic diseases. Altogether, impacts of undernutrition can lead to higher morbidity and mortality [5], result in substantial losses in disability-adjusted life years, inhibit productive work [6], and reduction in economic productivity [7]. As such, the 2nd Sustainable Development Goal (SDG) envisages ending hunger and all forms of undernutrition by 2030. Importantly, however, a recent evaluation of patterns in childhood undernutrition documented only minor improvements

(5–10%) between 2000 and 2015 globally, suggesting that the decreases observed are likely to be insufficient to meet the 2030 SDG goals [8].

Existing studies have identified many of the determinants of childhood undernutrition, including demographic characteristics, morbidity, economic status, sanitation practices and maternal nutrition [9, 10]. Place of residence is also an important predictor of undernutrition. Children living in Asia and Africa have the highest risk of various types of undernutrition [11], and in some of these areas, like India, the prevalence of undernutrition has worsened over time [12]. Recent observations show that the highest incidence of stunting (smaller than height-for-age) and wasting (smaller than weight-for-age) was between birth to 3 months. Collectively, this suggests that there is a need

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to identify modifiable environmental factors that may increase risk [13, 14], with a special emphasis on maternal or in-utero exposures [15].

A growing body of evidence indicates that in-utero exposure to $PM_{2.5}$ and NO_x is linked to various adverse health consequences, including pregnancy loss, preterm delivery, intrauterine growth restriction, and low birth weight (LBW) [14, 16]. Evidence exists that exposure to ambient air pollution is associated with adverse child health, anaemia, and stunted growth [17]. In addition, exposure to polluted air has been associated with acute respiratory infections (ARI), with adverse effects on childhood nutritional status [18]. The use of unclean fuel further results in poor health in women and children due to smoky indoor concentrations with $PM_{2.5}$ and other pollutants [19].

Concurrent high ambient and indoor air pollution levels in India [20] and a large fraction of deaths among children under the age of 5 years due to nutritional causes suggest that reducing maternal exposure to air pollution may be a way to reduce the burden of childhood undernutrition in this large country [21]. Although existing national-level analyses in India have shown adverse associations between high maternal and/or childhood exposures to air pollution and childhood undernutrition [22, 23], there remain important gaps in the literature. First, the effects of outdoor and indoor air pollution need to be considered together as the fuel type may influence the importance and contribution of each source. Second, much of the literature has focused on $PM_{2.5}$, which is a regional pollutant that arises from multiple sources. Examining additional pollutants may provide insight into the sources of pollution that are most impactful for health. For example, NO_2 is a more localised pollutant that often tracks with traffic sources and SO_2 is primarily generated from coal-burning activities. Investigation of the relationships of these pollutants with children's health may inform strategies for pollution control in India. Finally, as previous studies have shown, there are important spatial patterning of these associations that deserve further investigations as area-level factors may modify or confound the pollution and undernutrition association. Therefore, in this study, we examine the role of maternal exposure to both indoor and multiple ambient air pollutants ($PM_{2.5}$, NO_2 and SO_2) during pregnancy on childhood undernutrition (i.e. stunting, wasting, and anaemia) at the individual and district levels to help inform strategies to reduce childhood undernutrition.

DATA AND METHODS

Study population and health data

The study population comes from the fourth round of the National Family Health Survey (NFHS-4) of India, conducted from January 2015 to December 2016 [12]. The sample was recruited across 36 states and union territories and 640 districts using a stratified two-stage sampling design based on the 2011 Indian Population and Housing Census. Primary Sampling Units (PSUs) were villages in rural areas and census enumeration blocks (CEBs) in urban areas. Survey questionnaires were prepared to collect information on maternal and child health along with socioeconomic information. In addition to interview data, the NFHS collected biomarker data. Anthropometry was measured for children aged 0–59 months and blood samples were taken from children aged 6–59 months. The protocol for the NFHS-4 survey, including the content of all the survey questionnaires, was approved by the IIPS Institutional Review Board and the ICF Institutional Review Board. The protocol was also reviewed by the U.S. Centres for Disease Control and Prevention (CDC). Further details on the sampling design can be obtained in the Indian National Report available at <https://dhsprogram.com/pubs/pdf/FR339/FR339.pdf>. In the survey, anthropometric measurement was taken from 219,796 children and 205,035 for blood sample.

Air pollution data

Ambient air pollution. To estimate the ambient $PM_{2.5}$ and SO_2 concentrations, we used data from the Modern-Era Retrospective analysis for Research and Applications version 2 (MERRA-2) [24]. MERRA-2 integrates ground-level and satellite observations using NASA Global Modelling and Assimilation Office's (GMAO) Goddard Earth Observing System Model version 5 (GEOS 5) and the Goddard Chemistry, Aerosol, Radiation and Transport (GOCART) aerosol module. These data are available from 1980 to 2022. More details are available at https://disc.gsfc.nasa.gov/datasets/M2TMNXAER_5.12.4/summary.

Ozone Monitoring Instrument (OMI) satellite images were used to assess the mother's exposure to nitrogen dioxide (NO_2) during pregnancy. OMI satellite imagery was launched in 2004 as a part of the NASA EOS (Earth Observation System), which performs measurements of solar radiation reflected by the atmosphere and the Earth's surface with spectral resolutions of 270–500 nm and 0.5 nm. OMI provides the total NO_2 content in the vertical column of the atmosphere. The concentration was calculated by dividing the NO_2 content by the air mass value of NO_2 , which depends on a number of parameters such as the geometry of the observation, the albedo of the surface, the shape of the vertical profile of NO_2 , and the properties of the cloud (e.g. height, density, and sky coverage). To ensure the data quality, we excluded data when the cloud radiance fraction exceeded 0.3 (i.e. close to clear sky). For more details, visit <https://giovanni.gsfc.nasa.gov/giovanni/>.

We assigned average in-utero air pollution exposures to each survey participant based on the date of birth and duration of pregnancy of the children from the NFHS. For example, if a child was born in January 2015 with a pregnancy duration of 9 months, May 2014 to the end of January 2015 was considered as the in-utero period. To protect participants' privacy, the cluster points were displaced randomly by 2 and 5 km in urban and rural areas, respectively. Thus, a 3 km buffer was created to extract the pollutants of each respective cluster point. Our analysis utilized the 25th and 75th percentiles of ambient air pollutant concentrations as thresholds to define low and high exposure groups, respectively.

Indoor air pollution. Indoor air pollution was estimated using the household information available in NFHS. In the survey, respondents were asked about using primary cooking fuel. We classified wood, coal, dung cake, and crop residue as solid or unclean fuel and natural gas, liquefied petroleum gas, and electricity as clean fuels. Cooking in the household using solid cooking fuel was considered as exposure to indoor air pollution.

Variable description

Dependent variables. The survey has collected the height and weight of children aged up to 5 years during the survey through anthropometric measurement. We used two anthropometric indices, stunting (height-for-age) and underweight (weight-for-age), for physical growth assessment. These indices were derived from the standard deviation units (Z-score) and the median of the reference population based on the standards of WHO [25]. If the Z-score for height-for-age and weight-for-age is more than 2 standard deviations lower than the median, then the child is considered stunted and underweight, respectively. In addition, the survey provides the altitude adjusted haemoglobin levels. With these data, we defined anaemia as haemoglobin levels <11 g/DL [26].

Covariates. Key socioeconomic and demographic factors were selected to adjust associations of pollution with our outcome variables based on our expectations of potential confounders of the relationships between air pollution and children's health. The mother's age at delivery was calculated using information such as age of the child, mother's current age, and date of birth of the

child. The mother's highest level of education was recoded as 'literate/primary', 'secondary' and 'higher'. The wealth quintile of the household was categorized into three categories; 'poor', 'middle' and 'rich'. The religion of the household was recoded as 'Hindu', 'Muslim' and 'Others' (Sikh, Christian, Jain etc.). The toilet facility was recoded into 'no' – do not have toilet facility or used to go in field or bush for the toilet and 'yes'. Place of residence of the respondents was reported in two categories; 'urban' and 'rural'. Further, all the regions of India- 'North', 'Central', 'South', 'East', 'West' and 'North-East' were considered for the analysis (Supplementary Table A.2).

Statistical analysis

We first conducted univariate Moran's I statistics to identify the community (clusters) with higher (i.e. hot spots) and lower (i.e. cold spots) prevalence than expected of our three measures of undernutrition using a Local Indicators of Spatial Association (LISA) map. We then ran bivariate Moran's I analyse to estimate the spatial correlation between the prevalence of undernutrition and air pollution. Next, we used a spatial lag model to understand associations between air pollutants and area-level child malnutrition while accounting for spatial correlation as defined by the following equation:

$$Y_i = \delta \sum_j w_{ij} Y_j + \beta X_i + \varepsilon_i \quad (1)$$

where Y_i denotes the prevalence of childhood undernutrition for the i -th cluster, δ is the spatial autoregressive coefficient, w_{ij} denotes the spatial weight of proximity between clusters i and j , Y_j is the prevalence of childhood undernutrition in the j -th cluster, β denotes the coefficients for all X_i , which reflects the set of predictor variables, and ε_i is the residual error. Finally, we performed a multivariable logistic regression model to study the relationship between air pollution and child undernutrition after adjusting other covariates.

As corroborated by previous studies, a moderate correlation between $PM_{2.5}$ and NO_2 of 0.64 ($p < 0.05$), and NO_2 and SO_2 of 0.41 ($p < 0.05$) was observed in the study, (Supplementary Table A.1). Based on these observations, we conducted both single and multi-pollutant models for our study. The adjustment set for our models included: sex of the child, mother's age at birth, mother's education, mother's height, religion, wealth status, toilet facility, urbanicity, and region. Generalised estimating equations were used to account for community-level clustering. The interaction terms between indoor and ambient air pollutants were used to assess the combined effects of indoor and ambient pollution on childhood undernutrition after adjusting for other demographic and socioeconomic determinants. Community-level spatial analyses were performed on ArcGIS and GeoDa, while individual-level GEE models were performed in STATA Software ('xtgee').

RESULTS

Prevalence of undernutrition

In our sample of 236,116 children, the overall prevalence of stunting, underweight and anaemia was 35%, 33% and 69% respectively, in a sample of 209,210 mothers (Table 1). The highest prevalence of stunting was observed in the Northern states, such as Uttar Pradesh (45%) and Bihar (47%), and in Western India, including states like Maharashtra (Fig. 1). The prevalence of underweight children was also high in ten states having more than 1 out of 3 children being underweight. For anaemia, the highest prevalence was in Haryana (72%), followed by Jharkhand, Madhya Pradesh, Bihar and Uttar Pradesh. Fewer children with undernutrition were observed in southern and North-East India. The study utilized hot spot analyses to identify statistically significant clusters of undernutrition. The resulting map displayed dark red clusters representing the 99% significant cluster points

(hot spots) for the outcome variable. A high concentration of statistically significant hot spots of stunting was observed in most parts of Bihar, as well as some parts of Uttar Pradesh and Jharkhand. Conversely, hot spots for underweight were more than expected in states such as Jharkhand, Bihar, and Chhattisgarh, with a sparse distribution observed in the Central region of India and Odisha. The hot spots for child anaemia were densely concentrated in Delhi and its surrounding regions, as well as Madhya Pradesh, Bihar, Uttar Pradesh, and Jharkhand. In addition, statistically significant cold spots of undernutrition (represented by dark blue points) were predominantly found in the North-Eastern and Southern regions of India.

Table 2 presents the association of different socio-demographic, economic and household determinants with undernutrition. Although the study did not observe significant differences in the prevalence of undernutrition by sex of the child, children with stunting and who were underweight were more frequently males, while anaemia was higher among females. Adolescent (below 20 years of age) mothers had a higher proportion of stunted (40%) and underweight (37%) children, whereas mothers aged 30 years and above had a higher proportion of children who were anaemic (59%). Similarly, there were more stunted and underweight children among mothers with lesser height, those belonging to the Muslim community, having no toilet facilities, and living rural areas. In contrast, mothers who belonged to wealthy households and were more educated had fewer malnourished children.

Air pollution concentrations

Table 1 shows that about half (49%) of the households in India were exposed to indoor air pollution from solid fuel use, with the highest proportion of 77% in Chhattisgarh, the northern part of India. Similar to the prevalence of undernutrition, a high proportion (>70%) of indoor air pollution exposure was also observed in Jharkhand, Bihar and Madhya Pradesh, the central region of India. Figure 2 depicts the spatial concentration of the pollutants across India. The distribution map of ambient $PM_{2.5}$ shows a high concentration over the upper Gangetic region, covering states like Uttar Pradesh, Bihar, Delhi, Punjab and Haryana, whereas SO_2 concentrations were highest in some parts of the Eastern and Central region (Jharkhand and Chhattisgarh). The spatial pattern of NO_2 shows higher concentrations in the Northern (Punjab, Haryana, Delhi) and Eastern regions (West Bengal, Jharkhand and some parts of Odisha).

Cluster-level associations between air pollution and child undernutrition

Figures 3–5 illustrate the bivariate spatial correlations between ambient $PM_{2.5}$, NO_2 , SO_2 , and indoor air pollution with childhood undernutrition among the 27, 872 clusters. For all three outcomes of child undernutrition, there were consistent positive trends with ambient $PM_{2.5}$, NO_2 , and indoor air pollution, whereas relationships with SO_2 were less clear. Strong spatial relationships were observed between childhood undernutrition and ambient $PM_{2.5}$ and NO_2 most notably in the northern, and central regions of India, including most of the Empowered Action Group (EAG) states such as Uttar Pradesh, Bihar, and some parts of Jharkhand. The bivariate LISA map indicates a strong spatial autocorrelation between $PM_{2.5}$ and undernutrition; stunting; $\beta = 0.12$, $p \leq 0.00$, underweight; $\beta = 0.13$, $p \leq 0.00$ and anaemia; $\beta = 0.20$, $p \leq 0.00$. There was also evidence of clustering of these two pollutants with undernutrition in Delhi. For indoor air pollution as well, there was a notable positive spatial correlation with the outcomes (stunting; $\beta = 0.17$, $p \leq 0.00$, underweight; $\beta = 0.18$, $p \leq 0.00$ and anaemia; $\beta = 0.09$, $p \leq 0.00$), but the hotspots were more dispersed throughout Central India than for ambient $PM_{2.5}$ and NO_2 . SO_2 had a much weaker spatial correlation with the outcomes but showed evidence of clustering in Eastern India.

Table 1. The proportion of households exposed to indoor air pollution and prevalence of stunting, underweight and child anaemia across Indian states and Union Territories.

State	Indoor air pollution (%)	Stunting (%)	Underweight (%)	Anaemia (%)
<i>North</i>				
Chandigarh	12.69	29.76	25.60	72.18
Delhi	4.29	32.53	27.90	59.86
Haryana	50.00	34.62	29.89	71.84
Himachal Pradesh	52.99	27.12	22.33	53.70
Jammu And Kashmir	41.44	28.06	17.08	54.16
Punjab	35.77	26.50	22.00	56.69
Uttarakhand	44.49	34.30	27.36	60.21
Rajasthan	50.17	39.72	37.11	60.44
<i>Central</i>				
Chhattisgarh	77.22	38.16	38.70	41.74
Madhya Pradesh	70.25	42.51	43.60	69.02
Uttar Pradesh	62.50	46.87	40.32	63.35
<i>East</i>				
Bihar	71.48	48.99	44.56	63.54
Jharkhand	76.40	46.13	48.54	70.10
Odisha	53.66	34.57	34.93	44.61
West Bengal	36.80	33.13	32.45	54.47
<i>North-East</i>				
Arunachal Pradesh	54.78	29.85	19.94	54.38
Assam	42.95	36.75	30.44	35.88
Manipur	44.84	29.18	14.04	23.96
Meghalaya	62.72	44.42	29.65	48.14
Mizoram	39.96	28.47	12.19	19.63
Nagaland	50.85	29.29	17.33	26.59
Tripura	48.01	24.33	24.55	48.23
Sikkim	19.58	30.17	14.34	55.54
<i>West</i>				
Maharashtra	41.71	34.68	36.66	53.97
Dadra And Nagar Haveli	48.44	41.84	39.86	84.35
Daman And Diu	12.53	23.12	27.47	74.46
Goa	20.04	20.32	24.03	48.35
Gujarat	35.89	38.97	40.20	62.40
<i>South</i>				
Lakshadweep	31.69	27.22	24.20	52.61
Karnataka	48.64	36.82	35.79	61.25
Kerala	45.20	20.41	16.53	35.95
Tamil Nadu	16.15	27.74	24.47	50.77
Andaman And Nicobar Island	27.61	23.31	21.32	50.27
Andhra Pradesh	24.11	32.23	32.80	58.50
Puducherry	12.08	23.94	22.74	44.90
Telangana	19.71	28.06	28.40	60.63
Total	49.01	38.94	36.43	58.65

Using solid cooking fuel was considered as exposure to indoor air pollution. Stunting (height-for-age) and underweight (weight-for-age) were derived from the standard deviation units (Z-score) and the median of the reference population based on the standards of WHO. Less than 11 g/DL of haemoglobin levels defined as anaemia.

The results of the spatial lag model (Table 3) show that each $1 \mu\text{g}/\text{m}^3$ increase in maternal exposure to ambient $\text{PM}_{2.5}$ across the clusters of India was associated with a 0.11 percentage point increase in the prevalence of stunting ($p \leq 0.00$). In addition, a one-point increase in the exposure to NO_2 was statistically associated

with a 0.02-point increase in stunting prevalence ($p \leq 0.02$). Mothers exposed to higher levels of ambient $\text{PM}_{2.5}$ during pregnancy had significantly higher likelihood of having underweight ($\beta = 0.0009$, $\text{SE} = 0.00006$), while each $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ increases the prevalence of anaemia by 0.19%. Other

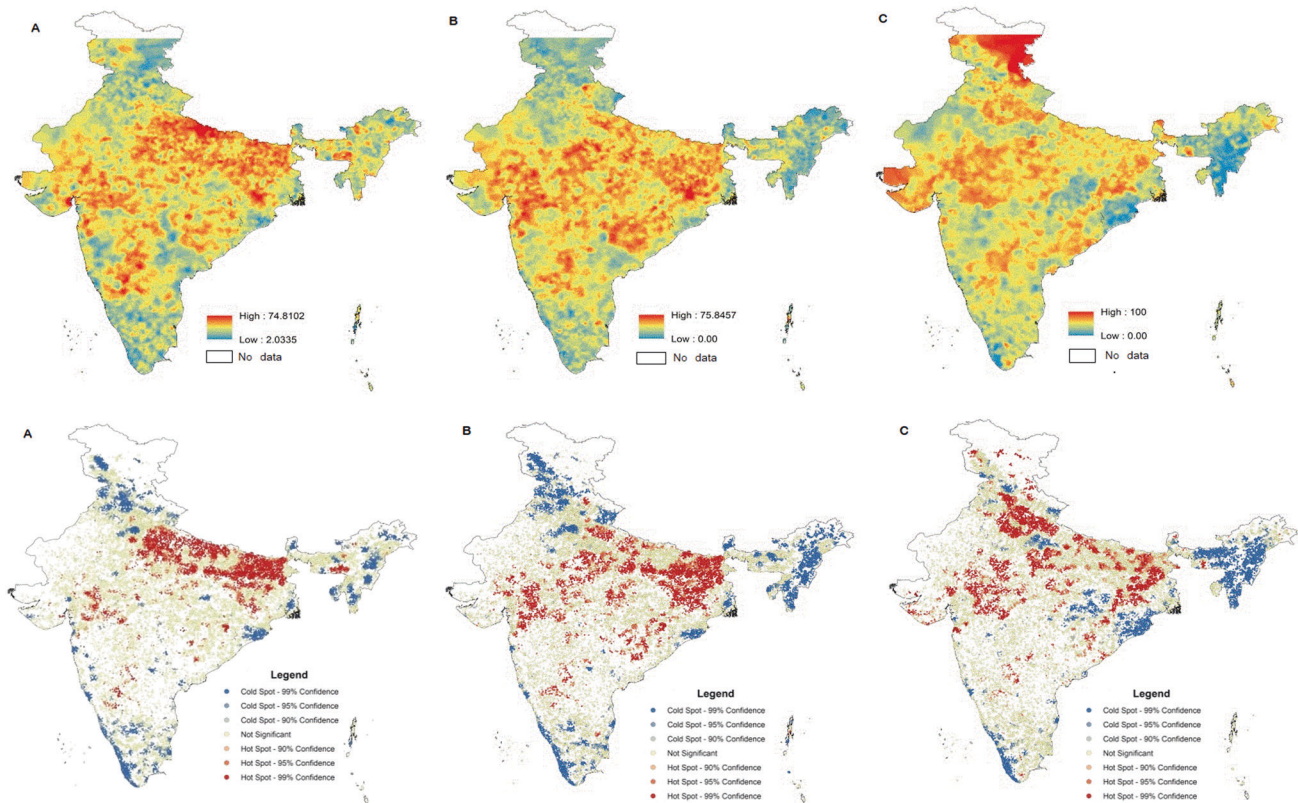


Fig. 1 Prevalence and clustering map of child undernutrition indicators across India. **A** Prevalence and clustering of stunting. **B** Prevalence and clustering of underweight. **C** Prevalence and clustering of anaemia. Source: Map prepared by authors using ArcGIS 10.8, <https://arcgis.software.informer.com/>.

socioeconomic parameters such as open defecation, poverty and proportion of educated mothers, teenage mothers and sex of the child were also associated with stunting and underweight (Supplementary Tables A3–A5). The coefficient estimate confirmed that a 1-point increase in maternal exposure to SO_2 ($\beta = 0.00014$, $\text{SE} = 0.00002$) and NO_2 ($\beta = 0.00031$, $\text{SE} = 0.00003$) was associated with an increase of 0.03 points and 0.06 points, respectively in underweight prevalence. A negative spatial relationship of SO_2 was found with stunting, which was not statistically significant.

Individual-level associations between air pollution and child undernutrition

In our minimally adjusted models with single pollutants and fully adjusted multivariable logistic regression models with multi-pollutants, we observed consistent associations between air pollution during pregnancy and childhood undernutrition (Table 4). In our multi-pollutant models, we observed that a higher ambient (75th percentile) $\text{PM}_{2.5}$ concentration during pregnancy was associated with higher odds of having stunted (AOR:1.32; 95% CI:1.25–1.38), underweight (AOR: 1.271.59; 95% CI: 1.20–1.33) and anaemic (AOR:1.19; 95% CI: 1.12–1.26) children. Mothers exposed to high concentration of NO_2 also had higher odds of having stunted (AOR:1.06; 95% CI:1.01–1.09), underweight (AOR:1.06; 95% CI:1.02–1.10) and anaemia (AOR: 1.14; 95% CI: 1.10–1.18) children as compared to their counterparts who were not exposed. Mothers exposed to indoor air pollution also had greater odds of having stunted (AOR: 1.05; 95% CI: 1.02–1.07), underweight (AOR:1.13; 95% CI:1.10–1.15) and anaemia (AOR:1.07; 95% CI:1.04–1.10) children as compared to those who were not exposed. Children who were highly exposed to SO_2 while in the uterus also had a greater chance of experiencing underweight (AOR: 1.12; 95% CI: 1.09–1.15) compared to those with low exposure to the pollutant. In the

multi-pollutant model, we found evidence of confounding between $\text{PM}_{2.5}$ and NO_2 for associations with underweight and anaemia but not stunting. After adjustment, associations with $\text{PM}_{2.5}$ strengthened in magnitude whereas associations with NO_2 became weaker. Indoor air pollution was largely robust to adjustment for outdoor pollutants. Socioeconomic and demographic variables such as sex of the child, mother's age at birth, mother's education, mother's height, religion, wealth status, toilet facility, place of residence, and region were adjusted for in the models (Supplementary Tables A6–A10).

In the interaction models, we found that ambient $\text{PM}_{2.5}$ had a stronger impact on stunting (AOR:1.54; 95% CI:1.47–1.62), underweight (AOR:1.83; 95% CI:1.73–1.93) and anaemia (AOR:1.73; 95% CI:1.63–1.83) among those women who had indoor air pollution exposures as compared to those who had no indoor air pollution exposures (AOR:1.44; 95% CI:1.37–1.52), (AOR:1.78; 95% CI:1.69–1.88) and (AOR:1.67; 95% CI:1.57–1.77), respectively. Mothers exposed to both higher concentration of SO_2 and indoor air pollution had a higher probability of having stunted (AOR:1.26; 95% CI:1.20–1.31) and underweight (AOR:1.25; 95% CI:1.19–1.29) children as compared to who were not exposed to indoor air pollution and had low concentration of SO_2 . In comparison to the previous situation, a decrease in odds ratio was observed among children whose mothers had high exposure to SO_2 but not indoor air pollution; stunting (AOR:1.15; 95% CI:1.09–1.20) and underweight (AOR:1.05; 95% CI:1.01–1.10). Women who were exposed to high levels of NO_2 but not exposed to indoor air pollution had higher odds of having underweight children and children having anaemia (AOR: 1.33; 95% CI: 1.27–1.40) and (AOR: 1.30; 95% CI: 1.24–1.37), respectively. The impact of NO_2 exposure increased when there was also exposure to indoor air pollution, resulting in higher odds of underweight (AOR: 1.46; 95% CI: 1.39–1.53) and anaemia (AOR: 1.33; 95% CI: 1.26–1.39).

Table 2. Percentage distribution of stunting, underweight and childhood anaemia by background characteristics, Na=2,19,796; Nb=2,05,035 children.

Background characteristics	Na	Nb	Stunting	Underweight	Anaemia
Sex of the child					
Male	1,23,209	1,09,243	37.33	34.85	58.49
Female	1,12,907	99,967	36.76	34.47	58.84
Mother's age at birth					
Below 20	31,403	28,278	39.76	37.08	57.48
20–24	1,07,084	95,065	36.76	34.69	59.17
25–29	65,520	57,404	35.42	32.91	58.44
30 & above	32,110	28,464	38.77	35.81	58.54
Mother's height (cm.)					
<145	27,792	24,630	54.76	49.78	61.73
15–149	63,889	56,502	43.63	41.27	59.69
150 & above	1,44,425	1,28,079	30.06	28.84	57.66
Mother's education					
Illiterate/primary	1,03,286	93,135	46.65	43.16	63.65
Secondary	1,08,004	94,898	31.80	30.30	55.77
Higher	24,825	21,176	20.05	18.34	49.63
Type of cooking fuel					
Clean	74,192	66,233	27.44	25.47	54.48
Unclean	1,61,924	1,42,977	41.47	38.88	60.59
Cooking place					
Separate kitchen	1,89,963	1,68,492	36.63	34.28	58.37
Without separate kitchen	46,153	40,718	38.83	36.25	58.73
Religion of the head of the household					
Hindu	1,85,616	1,64,680	37.20	35.22	58.85
Muslim	39,029	34,444	38.41	33.88	59.27
Others	11,470	10,086	30.26	28.39	53.46
Wealth status					
Poor	1,11,467	99,125	46.00	43.33	62.17
Middle	46,779	41,476	35.38	32.54	59.05
Rich	77,871	68,519	25.27	23.54	53.34
Has toilet facility					
No	1,04,150	93,487	45.44	42.83	62.63
Yes	1,31,966	1,15,723	30.45	28.22	55.29
Place of residence					
Urban	65,782	58,270	29.95	28.36	56.15
Rural	1,70,334	1,50,940	39.80	37.10	59.63
Region					
South	41,805	36,616	28.12	26.67	26.67
North	31,273	27,687	33.28	29.20	29
Central	64,094	56,887	43.22	38.81	62.72
North-East	8,305	7,145	33.91	26.53	36.63
East	60,938	54,376	40.75	35.26	59.21
West	29,701	26,497	33.62	26.67	56.18

Na; sample for stunting and underweight and Nb; Sample for anaemia.

DISCUSSION

These study findings reveal a clear association between exposure to indoor and outdoor air pollution during in-utero periods and poor child health outcomes, indicating the need to address the betterment of both household and ambient pollution for child health. Further, it identifies the spatial patterns of these relations to

help program interventions. Owing to the fact that this analysis was performed on a large-scale, nationally-representative dataset and adjusted for possible confounders, the findings of the study can be considered as robust and our area-level analyses will be informative in policymaking. The individual-level analysis further finds that an increase in ambient PM_{2.5} and exposure to indoor pollution are

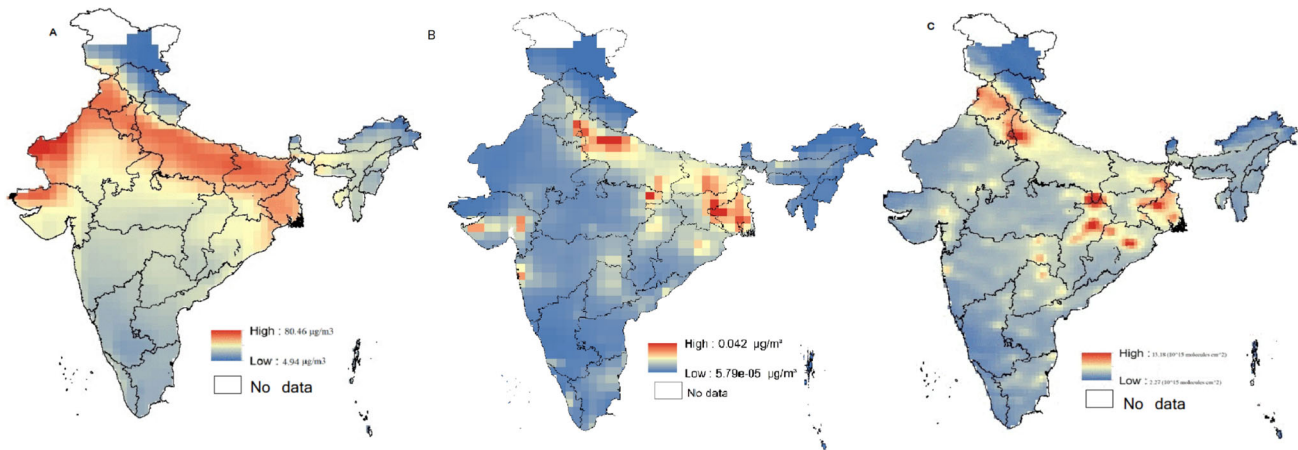


Fig. 2 Spatial distribution of ambient air pollution across India. A Spatial distribution of PM_{2.5}. **B** Spatial distribution of SO₂. **C** Spatial distribution of NO₂. Source: Map prepared by authors using GeoDa 1.8, <https://geoda.software.informer.com/>.

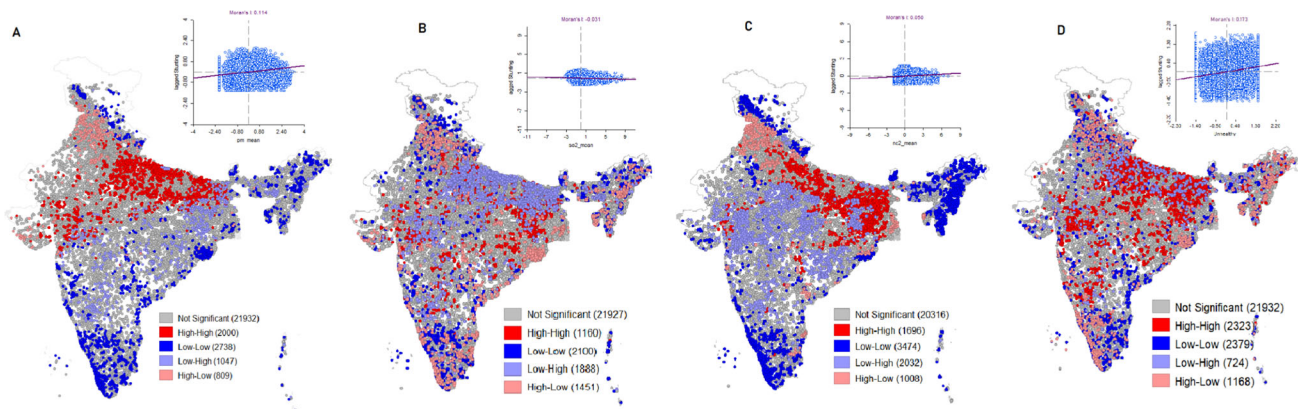


Fig. 3 Spatial relationships between stunting, ambient and indoor air pollution. A Relationship between stunting and PM_{2.5}. **B** Relationship between stunting and SO₂. **C** Relationship between stunting and NO₂. **D** Relationship between stunting and indoor air pollution. Map prepared by authors using GeoDa 1.8, <https://geoda.software.informer.com/>.

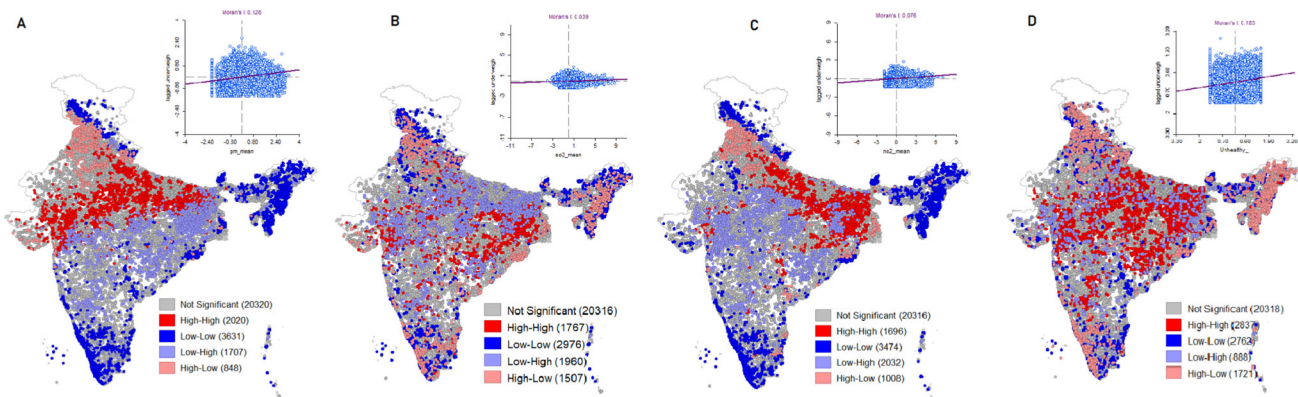


Fig. 4 Spatial relationships between underweight, ambient and indoor air pollution. A Relationship between underweight and PM_{2.5}. **B** Relationship between underweight and SO₂. **C** Relationship between underweight and NO₂. **D** Relationship between underweight and indoor air pollution. Map prepared by authors using GeoDa 1.8, <https://geoda.software.informer.com/>.

independently associated with a greater likelihood of stunting, underweight and anaemia, consistent with previous studies [23, 27]. We newly find an increase in the odds of having undernourished children among mothers who were exposed to NO₂ whereas associations with SO₂ was less clear. Our results add to the growing body of evidence suggesting that meeting targets for the National

Clean Air Program (NCAP) would improve child health and that air pollution control should be a top priority in India.

Spatial variations in the level of pollutants in India

Concentrations of ambient PM_{2.5} are higher in the Upper-Gangetic plains [28]. The deteriorating air quality has become an emerging

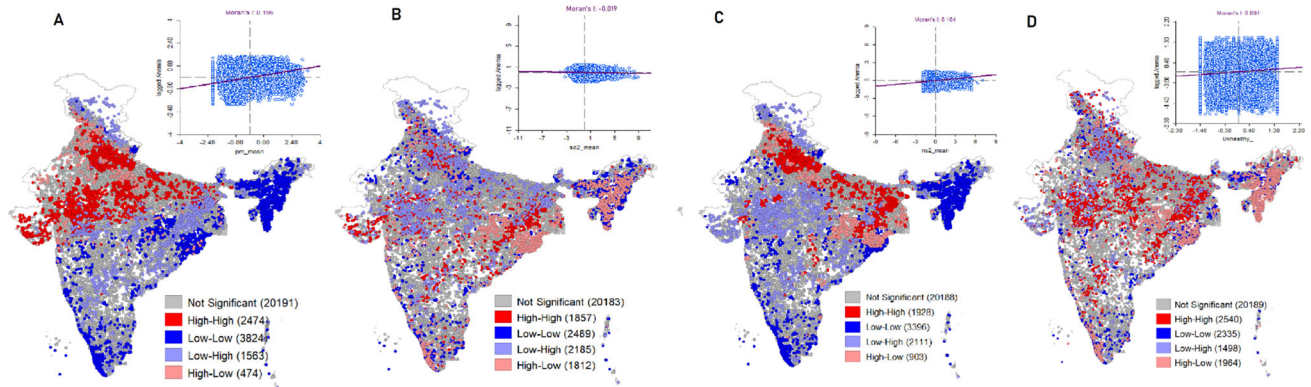


Fig. 5 Spatial relationships between childhood anaemia and ambient and indoor air pollution. **A** Relationship between childhood anaemia and PM_{2.5}. **B** Relationship between childhood anaemia and SO₂. **C** Relationship between childhood anaemia and NO₂. **D** Relationship between childhood anaemia and indoor air pollution. Map prepared by authors using GeoDa 1.8, <https://geoda.software.informer.com/>.

Table 3. Estimated result from independent Spatial Lag Model (SLM) showing the spatial association between exposure to air pollution and child nutrition.

Covariates	Stunting			Underweight			Anaemia		
	beta	SE	P	beta	SE	P	beta	SE	P
PM _{2.5} (µg /m ³)	0.00109	0.00007	<0.001	0.00093	0.00006	<0.001	0.00191	0.00008	<0.001
SO ₂ (µg /m ³)	−0.00003	0.00002	0.090	0.00014	0.00002	<0.001	0.00006	0.00003	0.007
NO ₂ molecules/m ²)	0.00023	0.00003	<0.001	0.00031	0.00003	<0.001	0.00062	0.00004	<0.001
R ²	0.20			0.24			0.21		

Exposed to indoor air pollution, proportion of male child, uneducated mother, teenaged mother, short stature mother, poor, open defecation, rural population and proportion of Hindu population adjusted in the model. Analysis conducted using 27,872 clusters available in the dataset.

public health as well as an environmental concern in India, especially in the northern part. Recently, Health Effects Institute (HEI) reported that exposure to PM_{2.5} reduces 1.5 years of life expectancy among Indians [29]. A previous study conducted using the chemical transport model has explored that about 60–70% of PM_{2.5} is emitted from industrial and residential activities in India. In addition, energy and agricultural sources also play a significant role [30]. Moreover, another study estimated that India produced 990.68 Gg/yr in 2018 PM_{2.5} due to the crop residue burning, while the Indo-Gangetic region was the hotspot [31]. Further, half of Indian households use solid or polluted fuels for cooking, heating and other domestic purposes, thus, becoming the largest contribution to ambient PM_{2.5}. The study estimated that most of the districts in the Indo-Gangetic region contributed more than 40% of ambient PM_{2.5} exposure that can be attributed to household PM_{2.5} [32]. Findings from another set of studies [28, 33] show that it is possible to achieve a significant reduction in PM_{2.5} levels by implementing stringent measures to curtail PM_{2.5} emissions.

Our findings for SO₂ and NO₂ suggest that concentrations are highest in Jharkhand, Chhattisgarh, Punjab, Haryana, and Delhi. This could be a result of large coal-fired power plants, biomass burning, vehicular and industrial pollution [32, 34, 35]. A heavy traffic network can be observed in Delhi, which is a major source of NO₂. On the other hand, areas such as Bokaro in Jharkhand, Durgapur in West Bengal, Bhilai in Chhattisgarh and Rourkela in Odisha are known as the locations of the Asia's significant iron and steel industries and a high level of NO₂. Moreover, the mining process and associated industries, thermal plants, fossil fuel extraction etc. play a major role in contributing to the high concentrations of NO₂ and SO₂, which could be a reason for having higher NO₂ and SO₂ concentrations in the East-Central and some parts of the Northern regions of India [36]. Thermal plants are the highest contributors to NO₂ emissions in India [37], which

can be found in the present study by looking at the spatial distribution.

Exposure to ambient PM_{2.5} during pregnancy and its effect on child nutrition

Our findings suggest that pregnant mothers who were exposed to ambient PM_{2.5} during pregnancy had higher odds of giving birth to anaemic children. The probable association between air pollution and childhood undernutrition has been gaining attention recently. There are several potential mechanisms by which maternal exposure to air pollution can lead to childhood stunting, wasting, and anaemia. It is well-established that exposure to air pollution can induce inflammation and when activated in pregnant women, the resulting epigenetic changes could directly affect fetal growth [38]. These mechanisms appear to explain the observed associations between maternal exposure to air pollution and adverse birth outcomes [39]. Studies show that increased prenatal exposure to ambient air pollution (PM_{2.5}) is associated with the increased risk of premature births and low birth weight at term [16, 17, 19]. Low birth weight at term is a surrogate for intrauterine growth constraint that is also independently linked to stunting and underweight [40]. Moreover, exposure to ambient air pollution could lead to respiratory infections such as pneumonia that suppress the appetite and reduce nutrient absorption [41]. Haemoglobin is a protein that is found in the bold red cells (RBCs) and transports oxygen [42]. The negative relationship between ambient PM exposure and haemoglobin concentration and RBC count could be attributed to growing evidence that adsorbed minerals in PM particles can destroy the RBCs and impede the release of haemoglobin [43–45]. Another possible pathway is decreased renal erythropoietin secretion and increased endogenous erythropoietin resistance in the marrow, thus, reducing RBC production and affecting the release of haemoglobin [46].

Table 4. Independent logistic regression model estimates showing the effects of indoor and outdoor air pollution on childhood stunting, underweight and anaemia.

Independent variables	AOR#	Stunting		AOR#	Underweight		AOR#	Anaemia	
		95% CI			95% CI			95% CI	
		Upper	Lower		Upper	Lower		Upper	Lower
Single pollutant results									
Outdoor air pollution									
PM _{2.5} (µg/m ³)									
Low	Ref.			Ref.			Ref.		
Medium	1.12***	1.07	1.17	1.23***	1.17	1.28	1.16***	1.10	1.22
High	1.32***	1.25	1.38	1.27***	1.20	1.33	1.19***	1.12	1.26
SO ₂ (µg/m ³)									
Low	Ref.			Ref.			Ref.		
Medium	0.99	0.96	1.02	1.08***	1.05	1.11	1.04**	1.01	1.07
High	0.94***	0.92	0.98	1.12***	1.09	1.15	1.00	0.96	1.04
NO ₂ (molecules/m ²)									
Low	Ref.			Ref.			Ref.		
Medium	1.11***	1.08	1.15	1.23***	1.19	1.26	1.13***	1.09	1.16
High	1.14***	1.10	1.18	1.27***	1.23	1.31	1.27***	1.22	1.32
Exposed to indoor air pollution									
No	Ref.			Ref.			Ref.		
Yes	1.05***	1.02	1.07	1.09***	1.07	1.11	1.07***	1.04	1.09
Multi-pollutant results									
Outdoor air pollution									
PM _{2.5} (µg m ³)									
Low	Ref.			Ref.			Ref.		
Medium	1.17***	1.13	1.21	1.52***	1.46	1.58	1.50***	1.43	1.56
High	1.38***	1.32	1.44	1.59***	1.51	1.67	1.61***	1.52	1.69
SO ₂ (µg m ³)									
Low	Ref.			Ref.			Ref.		
Medium	0.98	0.96	1.01	1.07***	1.04	1.10	1.02	0.99	1.06
High	0.92***	0.89	0.95	1.07***	1.03	1.10	0.93***	0.89	0.97
NO ₂ (molecules/m ²)									
Low	Ref.			Ref.			Ref.		
Medium	1.07***	1.04	1.10	1.08***	1.05	1.12	1.00	0.96	1.04
High	1.06***	1.01	1.09	1.06***	1.02	1.10	1.09***	1.05	1.14
Expose to indoor air pollution									
No	Ref.			Ref.			Ref.		
Yes	1.10***	1.07	1.11	1.13***	1.10	1.15	1.07***	1.04	1.10
Interaction results									
Indoor × PM _{2.5}									
No × low	Ref.			Ref.			Ref.		
No × high	1.44***	1.37	1.52	1.78***	1.69	1.88	1.67***	1.57	1.77
Yes × high	1.54***	1.47	1.62	1.83***	1.73	1.93	1.73***	1.63	1.83
Indoor × SO ₂									
No × low	Ref.			Ref.			Ref.		
No × high	1.15***	1.09	1.20	1.05**	1.01	1.10	0.99	0.94	1.04
Yes × high	1.26***	1.20	1.31	1.25***	1.19	1.29	1.04*	0.99	1.09
Indoor × NO ₂									
No × low	Ref.			Ref.			Ref.		
No × high	0.96*	0.92	1.01	1.33***	1.27	1.40	1.30***	1.24	1.37
Yes × high	1.04*	1.00	1.08	1.46***	1.39	1.53	1.33***	1.26	1.39

Note: reference category; Ref. and significant level; * $p < 0.10$. ** $p < 0.05$. *** $p < 0.01$.

AOR Adjusted Odds Ratio and CI Confidence Interval.

#Socioeconomic and demographic variables such as sex of the child, mother's age at birth, mother's education, mother's height, religion, wealth status, toilet facility, place of residence and region adjusted in the interaction model.

NO₂ and SO₂ exposure and its effect on child nutrition

Past evidence suggests that NO₂ exposures can create a chronic systemic inflammatory response, leading to increased endogenous erythropoietin resistance in the bone marrow, further causing a decrease in RBC production and haemoglobin levels [47, 48]. Inhaling NO₂ during pregnancy affects the fetus's growth by disrupting fetal antioxidant oxidant balance or leading to oxidative injury [49]. Moreover, it restricts the transportation of blood and glucose in the blood cells, affecting fetal growth [50]. Despite a dearth of literature on detailed biological mechanisms, there is evidence to suggest that in-utero exposure to oxides of nitrogen could lead to modulations in DNA methylation, thus, impeding fetal growth, an independent factor of stunting and underweight [51, 52]. In addition, oxidative stress and inflammatory responses triggered by prenatal exposure to oxides of nitrogen may lead to impaired neural and functional development, ultimately having an impact on child nutrition outcomes in later life [53]. Our findings do not depict a consistent and distinct association between SO₂ pollution and stunting prevalence among children, the finding of another Indian study [54]. This may be partly because we used a direct measure of SO₂ while Datt and colleagues used the number of coal-fired power plants and focused on the PM_{2.5} generated. Further studies are needed to offer a clearer explanation of the biological mechanisms of the impacts of SO₂ exposure on child health. However, evidence points to several mechanisms such as placental inflammation, oxidative stress, epigenetic changes such as DNA methylation and microRNA [52]. In addition, SO₂ absorbed in the human body likely has negative consequences for fetal growth and development, leading to the destruction of function and microstructure of germ cells [14]. Adverse birth outcomes and childhood illness increase the probability of being malnourished [55].

In-utero exposure to indoor air pollution and its effect on child nutrition

Past studies found that releasing pollutants from uncleaned biomass burning in the household restricts fetal growth, increasing the probability of having a child with low weight and preterm birth [56]. Moreover, respiratory infection and childhood diseases such as pneumonia and a weak immune system could result from in-utero exposure to indoor air pollution [57]. Smoke from unclean fuels is a complex mixture of pollutants that harm human health [58]. The severity of impact, however, depends on the pollutants' nature and the exposure duration. There are several pathways through which indoor air pollution impacts children directly [59]. Studies also suggest that pregnant women exposed to indoor air pollution are more likely to give birth to premature and low birth weight babies [19]. In addition, exposure to indoor air pollution during the first trimester of pregnancy has been shown to have a negative impact on child growth indicators [60]. Understanding the impact of exposure to pollutants during pregnancy is also important because pregnant women tend to spend most of their time indoors and this time only increases once the pregnancy progresses. There is enough evidence to support the fact that this causes detrimental birth outcomes, and its impact continues much later than immediately after birth [16, 17, 19].

Strengths and limitations

The study is unique for several reasons: First, this is the first study that takes into account multiple pollutants in explaining child health conditions. As such, it builds upon the one study in India [23] that published on in-utero exposure to PM_{2.5} and child malnutrition. In addition, the study evaluates the effects of ambient and indoor pollutants independently and altogether using nationally representative data. The study indicates a need for an intervention in both rural and urban areas of India as pollution of different kinds are well observed across the country with spatial hotspots. Conducting an

in-depth epidemiological study is necessary to understand the biological mechanism of the relationship between air pollution and undernutrition that cannot be explored through the existing data set. The effects of PM by its composition have not been examined in the study due to the unavailability of the data. The study has not considered the different pollutants emitted from the household cooking fuel due to the lack of information in the dataset. Similarly, we were unable to account for individual-level smoking behaviours though we do not expect these to correlate strongly with ambient or indoor pollution after adjustment for maternal socioeconomic status, urbanicity, and region. Finally, our research assumes that mothers did not change their residence from pregnancy period to the survey date. While this may introduce some error, our estimates should be reasonable if they remain in the same general area and if the children's health was unrelated to if a mother moved.

CONCLUSION

The central premise of the study is that there is an association between child nutrition outcomes and maternal exposure in-utero to ambient air pollution. The findings of the study suggest that, in addition to distinct spatial variations in the prevalence of child undernutrition in India, there is a presence of hotspots and cold spots of child undernutrition in the country. Maternal exposure during pregnancy to ambient air pollution leads to a higher risk of having undernourished and anaemic children. A significant role of both indoor and ambient air pollution during maternal gestation and the consequent child health can be observed in the study, which indicates an urgent requirement of continued efforts of cleaner air, fuel policy and awareness generation. Besides addressing the immediate determinants of nutrition, it is essential to target the distal factors like indoor and ambient air pollution in a synchronised way in order to be able to make progress in child health.

Reporting summary

Further information on research design is available in the Nature Research Reporting Summary linked to this article.

DATA AVAILABILITY

The study uses secondary data which are available on reasonable request through <https://dhsprogram.com/data/> and <https://giovanni.gsfc.nasa.gov/giovanni/>. The data and code used in this study are available from the corresponding author on reasonable request.

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AUTHOR CONTRIBUTIONS

Conceptualisation: AJ, AC. Data curation: AJ. Formal analysis: AJ. Methodology: AJ, AC. Software: AJ. Writing: AJ, AC, AS. Review & editing: AC, AS, AJ, SDA, JD.

COMPETING INTERESTS

The authors declare no competing interests.

ETHICS APPROVAL

The organization committee of the survey received ethical approval from the Ministry of Health and Family Welfare (MoHFW) to collect the human data. The Indian Demographic and Health Survey (DHS) is known as National Family Health Survey (NFHS) in India. We used published large-scale national data where every respondent was anonymized in the data set itself. As it is not based on a primary survey- cases, we were not required to do any anonymization in the study as the data is already made in that fashion following all ethical protocols. Thereby, it is certified that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the course of the survey. Also, verbal as well as written informed consent was obtained from all the participants. The informed consent was taken from their parent or legal guardian. The dataset is publicly available, thus, consent for publication is not applicable for the study.

ADDITIONAL INFORMATION

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