The association of in-utero exposure to ambient fine particulate air pollution with low birth weight in India

Goyal, Nihit; Canning, David

DOI
10.1088/1748-9326/abf18e

Publication date
2021

Document Version
Final published version

Published in
Environmental Research Letters

ENVIRONMENTAL RESEARCH
LETTERS

LETTER • OPEN ACCESS

The association of in-utero exposure to ambient fine particulate air pollution with low birth weight in India

To cite this article: Nihit Goyal and David Canning 2021 *Environ. Res. Lett.* **16** 054034

ENVIRONMENTAL RESEARCH
LETTERS

LETTER

The association of in-utero exposure to ambient fine particulate air pollution with low birth weight in India

Nihit Goyal[1,*] and David Canning[2]

[1] Faculty of Technology, Policy and Management, Delft University of Technology, Delft, the Netherlands
[2] Department of Global Health and Population, Harvard TH Chan School of Public Health, Boston, Massachusetts, United States of America
* Author to whom any correspondence should be addressed.

E-mail: nihit.goyal@tudelft.nl

Keywords: ambient air pollution, child anthropometry, environmental health, fine particulate matter ($PM_{2.5}$), Indian Demographic and Health Survey (DHS), low birth weight (LBW), National Family Health Survey (NFHS)

Supplementary material for this article is available online

OPEN ACCESS

RECEIVED
26 November 2020

REVISED
24 February 2021

ACCEPTED FOR PUBLICATION
23 March 2021

PUBLISHED
29 April 2021

Abstract

A growing body of research indicates that in-utero exposure to ambient fine particulate matter ($PM_{2.5}$) is a risk factor for low birth weight (LBW). However, research on India, where the high and rising level of ambient air pollution is a significant health concern, is limited. In this study, we analyze the association between ambient $PM_{2.5}$ and LBW in India. We match data on birth weight from the National Family and Health Survey (NFHS) conducted in India in 2015–16 with high-resolution spatial data on annual ambient $PM_{2.5}$ concentration to calculate in-utero exposure based on the residential location of each child. We estimate the association of in-utero exposure to ambient $PM_{2.5}$ with birth weight and LBW, after adjusting for child level, maternal, and household level characteristics that predict birth weight. In our sample ($n = 149\,416$), in comparison to the reference category of in-utero $PM_{2.5}$ level less than 26.7 $\mu g\ m^{-3}$, the adjusted OR of LBW increases non-linearly from 1.098 (95% CI: 0.954, 1.263) for children in the exposure band 39.3–44.7 $\mu g\ m^{-3}$ (i.e., the fourth octile) to 1.241 (95% CI: 1.065, 1.447) for those in the exposure band 44.7–51.6 $\mu g\ m^{-3}$ (i.e., the fifth octile) and 1.405 (95% CI: 1.126,1.753) for those with in-utero $PM_{2.5}$ level greater than 77.3 $\mu g\ m^{-3}$ (i.e., the last octile). Our findings show that exposure to ambient $PM_{2.5}$ is strongly associated with LBW in India and suggest that policies that improve air quality may be necessary for achieving the World Health Assembly target of 30% reduction in LBW by 2025.

1. Introduction

At least 15%–20% of children around the world—over 20 million newborns in 2015—are estimated to weigh less than 2500 g at birth [1]. While this is a global issue, the prevalence of low birth weight (LBW) is high in low- and middle-income countries, especially in South Asia and Sub-Saharan Africa. LBW is not only a predictor for neonatal morbidity and mortality, but also a risk factor for poor cognitive development, noncommunicable illness, and cardiovascular disease in later life [2]. Consequently, the World Health Assembly has adopted a target of 30% reduction in LBW by 2025 in comparison to the year 2012, but progress has been insufficient [3]. Although emphasis on nutrition; water, sanitation, and hygiene; access to healthcare facilities; and women empowerment may help reach this target [4], a focus on air quality—both indoor and outdoor—is also likely to be important.

The World Health Organization has estimated that only one in ten people breathe clean air [5]. The problem is especially severe in low- and middle-income countries which experience poor and worsening air quality. Ambient air pollution is a major risk factor for the global burden of disease and has been associated respiratory infection, lung cancer, heart disease, brain hemorrhage, and diabetes [6, 7]. Exposure to fine particulate matter—i.e., particles less than 2.5 μm in diameter, or $PM_{2.5}$—is especially harmful for children, and has been associated with impaired lung function, respiratory infection, stunting, and mortality [8–10]. A growing body of research has also identified ambient $PM_{2.5}$ as a risk factor for adverse

pregnancy outcomes, including pregnancy loss, preterm birth, intrauterine growth restriction, and LBW [11, 12].

We examined the existing literature on the relationship of LBW with ambient $PM_{2.5}$ through a search for ('birth weight') AND ('fine particulate*' OR 'particulate matter' OR 'PM2.5') in PubMed. Out of 298 studies returned by this search, we excluded 153 studies that did not include (low) birth weight as an outcome variable or ambient $PM_{2.5}$ as an exposure variable based on a scan of each article title and abstract. We found that most of the remaining studies on the relationship between (low) birth weight and ambient $PM_{2.5}$ have been conducted in high-income countries, which have a relatively low level of ambient air pollution [13]. Illustratively, a European cohort study (ESCAPE) that pooled data from 14 representative studies in 12 countries consisted of ambient $PM_{2.5}$ concentration in the range 0–40 approximately [14]. Research on the impact of ambient air pollution on birth weight in low- and middle-income countries such as China and India—which have significantly higher concentrations of ambient $PM_{2.5}$—is still limited.

While the sources of $PM_{2.5}$ typically include the burning of biomass and crop residue, energy production using fossil-fuel in industries, thermal power plants, urban transportation, and construction activities, their apportionment varies significantly based on region and the level of economic development [15]. Consequently, the composition and toxicity of ambient $PM_{2.5}$ are also likely to vary from one setting to another, leading to variation in the relationship of $PM_{2.5}$ with human health [13, 16]. Yet, in the context of India, only two studies have examined this relationship [17, 18]; these studies present contradictory evidence and neither is representative for India. In the absence of such context-specific evidence, the burden of disease due to ambient air pollution is typically estimated using concentration response curves (CRCs) extrapolated from research conducted in settings with different concentration, composition, and toxicity of ambient $PM_{2.5}$ [6].

In this study, we directly estimate the relationship between ambient $PM_{2.5}$ and LBW in India. Although India has made some progress over the past decade in improving child health, the prevalence of LBW is still estimated to be over 25% [19]. The proximate factors for LBW in India—several of which are linked to household poverty—include low maternal nutritional status, a high burden of infectious diseases, low levels of maternal education, poor sanitation and hygiene, and the lack of antenatal care [20, 21]. However, the country also has one of the worst levels of air quality in the world and 22 of the world's 30 most polluted cities are from India [22]. Thus, a better understanding of the relationship between ambient air pollution and LBW is essential for an effective policy response. We contribute to the literature by providing evidence on the relationship between ambient $PM_{2.5}$ and LBW over a wide concentration range (approximately 0–130 $\mu g\ m^{-3}$) and adding to the small but growing research on adverse health outcomes due to air pollution in low- and middle-income countries using nationally representative survey data.

2. Materials and methods

2.1. Study design

This cross-sectional study is based on data on health of children in India from the National Family and Health Survey (NFHS) conducted in 2015–2016. The NFHS is a nationally representative household survey on reproductive and child health [23]. It employs a two-stage sampling design stratified by rural and urban location. The primary sampling unit is a (segment of a) village in a rural area or a Census Enumeration Block in an urban area (hereafter, cluster) [24]. In each cluster, 22 households are sampled systematically and women of reproductive age—between 15 and 49 years old—are identified through a household questionnaire. The 'eligible' women are then administered a survey to collect information on the health of children born to them during the 5 years prior to the survey date.

The NFHS has observations on 259 627 children born during 2010–2016. We drop observations for children whose mother is a visitor ($n = 8487$) or moved to the place of her current residence after the conception period of the child ($n = 40\,147$), as our exposure measurement is not valid for those children. Therefore, the total sample consists of data on 219 480 children. In addition, we drop observations with missing information on birth weight and other covariates, resulting in a final sample of 149 416 children (see supplementary information: table S1 (available online at stacks.iop.org/ERL/16/054034/mmedia)). Thus, all children in the NFHS for whom the relevant data are present are included in our analysis.

2.2. Outcome

The primary outcome of interest for this study is LBW. Additionally, we use birth weight (in g) as an outcome to corroborate the findings. Data on birth weight in the NFHS is based on a written health record, if available, or the mother's report. LBW is defined as weight at birth less than 2500 g regardless of gestational age.

2.3. Exposure assessment

For information on air pollution, we use high-resolution spatial data on fine particulate matter from the Atmospheric Composition Analysis Group 15]. They estimate the annual average ambient $PM_{2.5}$ concentration at a resolution of $0.01° \times 0.01°$ (approximately, 1 km × 1 km) through a triangulation of

satellite data, ground-based air quality monitoring, and modelling of pollution sources and wind dispersion patterns. Although earlier versions of these estimates were questionable due to a lack of ground-based monitoring in the Indian subcontinent [25], the latest version we use has a strong correlation with an expanded ground-based monitoring network (correlation coefficient $\rho = 0.81$).

We match data on child health with data on ambient $PM_{2.5}$ concentration using the GPS coordinate of each cluster. In the NFHS, the location of each urban cluster is randomly displaced by up to 2 km for an urban cluster and up to 5 km for a rural cluster to protect respondent confidentiality [26]. Following the recommended methodology [27], we correct for this by calculating the mean of the annual average $PM_{2.5}$ concentration over the displacement radius of each cluster. As the annual average $PM_{2.5}$ concentration at the GPS coordinate of the cluster is highly correlated with the mean annual average $PM_{2.5}$ concentration over the displacement radius (correlation coefficient $\rho > 0.999$), the noise in the cluster GPS coordinates is unlikely to bias our findings.

We calculate in-utero $PM_{2.5}$ exposure by weighting the annual average ambient $PM_{2.5}$ concentration for each year overlapping the in-utero period of the child by the fraction of the in-utero period in that year. We use information on pregnancy duration, recorded by the NFHS, to approximate the in-utero period. For example, the in-utero period for a child born in July 2013 with a pregnancy duration of 9 months is assumed to extend from the beginning of November 2012 till the end of July 2013. Therefore, we weight the annual average $PM_{2.5}$ concentration in 2012 by 2/9 and the annual average $PM_{2.5}$ concentration in 2013 by 7/9 (i.e., number of months in-utero during that year divided by the pregnancy duration).

2.4. Control variables

We control for various child level, maternal, and household level characteristics that have been identified as determinants of LBW in India [20, 21]. At the child level, these include birth order, whether the child is the first born, whether the child is a twin or triplet, the sex of the child, and the preceding birth interval. At the maternal level, the covariates include an indicator for teenage motherhood, the education level of the mother, whether the mother has short stature (height less than 160 cm), whether the mother is underweight (BMI less than 18.5 kg m^{-2}), whether the mother has anemia, whether the mother uses tobacco, and whether the mother smokes *bidis* (a bidi is a hand-rolled cigarette consisting of tobacco wrapped in a plant leaf; in the NFHS, data on this is recorded separately from that on tobacco use). Household level characteristics include whether the household uses solid cooking fuel, whether the household has access to an improved sanitation facility, whether the household has access to an improved drinking water source, whether the household is located in an urban area, and the wealth quintiles of the household in the state and the country. In addition, we include birth month dummies, birth year dummies, and district dummies to control for unobserved characteristics.

2.5. Statistical analysis

We estimate the effect of air pollution on birth weight at the individual child level. As the outcome on birth weight is continuous, we estimate this relationship using linear regression. As the outcome on LBW is binary, we estimate this relationship using logistic regression. We cluster standard errors at the DHS cluster level to account for a sampling methodology and allow for correlation between outcomes for children within a cluster. We present the estimates from the regressions as change in outcome in g (for linear regression) and odds ratios (for logistic regression) with their 95% CI.

Various CRCs have been proposed in the literature to estimate or model the relationship between ambient $PM_{2.5}$ exposure and health based on studies on high-income settings or second-hand smoke [28]. However, the sources and toxicity of ambient $PM_{2.5}$ in low- and middle-income settings might be different [15, 29, 30]. Therefore, rather than imposing a functional form, we estimate the relationship by dividing the exposure into octiles, each comprising 12.5% of the sample.

We conduct additional analysis to check the robustness of the findings. First, we examine whether the estimates are sensitive to additional control for ante-natal visit. Second, we estimate the relationship for the sample consisting only of children with birth weight information recorded from health information card (and not mother's recall). Third, we test for sensitivity to variation in the control for birth year (no time trend, linear time trend, and state level birth year effect). Fourth, we also test for sensitivity to variation in the level of fixed effect (state fixed effect, cluster fixed effect, and sibling fixed effect). Finally, we analyze the robustness of the findings to alternate specifications of the CRC for in-utero $PM_{2.5}$ level (linear specification, log specification, and spline specification with three nodes).

2.6. Software

The geospatial matching and analysis are done using QGIS software (version 3.4.6-Madeira; Open Source Geospatial Foundation Project). The statistical analyses are conducted using STATA software (version STATA/SE 14.1; StataCorp LP, College Station, TX, USA).

3. Results

The spatial distribution of annual average ambient $PM_{2.5}$ concentrations in 2009 and 2016 is shown

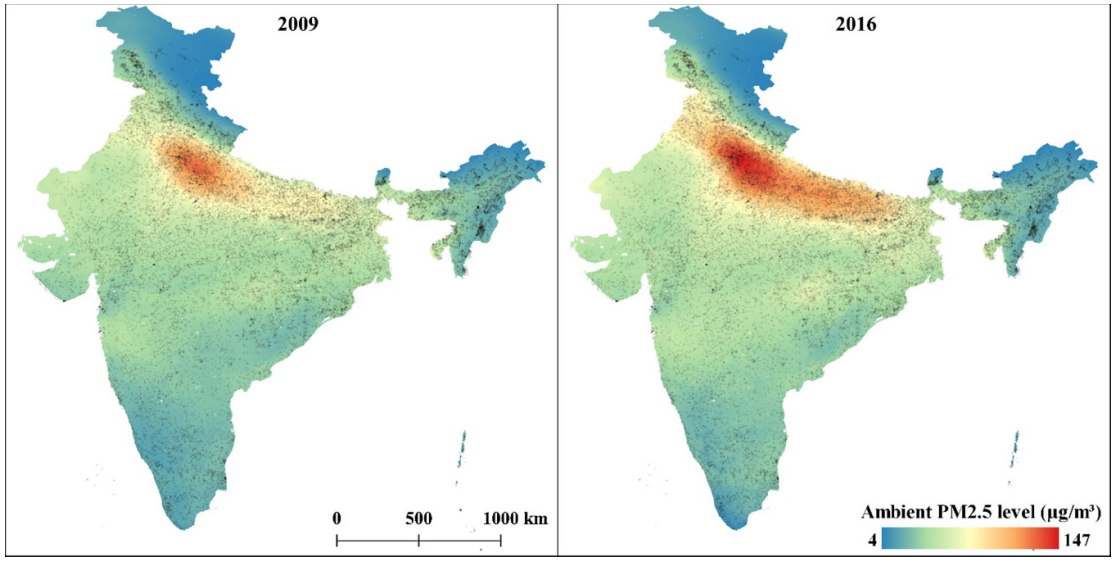

Figure 1. The DHS cluster location (black marker) overlaid on the annual average ambient $PM_{2.5}$ concentration in India in 2009 (left) and 2016 (right).

in figure 1. We observe that air quality in India is poor and worsening, with the geographic mean $PM_{2.5}$ concentration increasing from 42 $\mu g\ m^{-3}$ in 2009 to 49 $\mu g\ m^{-3}$ in 2016. Further, $PM_{2.5}$ concentration exhibits significant spatial heterogeneity in India. While a detailed examination of this heterogeneity requires granular characterization of the sources of air pollution [31]—data for which are largely unavailable at the pan India level [32, 33]—we observe that the Indo-Gangetic plain has particularly severe air pollution. The reasons for this include the presence of many inefficient brick kilns, high use of coal and biomass, and a landlocked geography [34].

The descriptive statistics for our sample are shown in table 1. The mean birth weight of children is 2819.32 g (SD: 603.86). With 25 886 cases of children weighing less than 2500 g at birth, the prevalence of LBW is 17%. The mean in-utero $PM_{2.5}$ level in our sample is 50.01 $\mu g\ m^{-3}$ and children with LBW are exposed to a slightly higher level of $PM_{2.5}$ in-utero (52.84 $\mu g\ m^{-3}$) than children with normal weight at birth (49.42 $\mu g\ m^{-3}$). Moreover, over 99% of the children are exposed to ambient $PM_{2.5}$ concentration exceeding the WHO guideline of 10 $\mu g\ m^{-3}$ (figure 2). In addition, we observe that 67% children were from a household that used solid cooking fuel while 9% of children were exposed to second-hand smoke from maternal tobacco use (table 1). The correlation matrix is shown in supplementary information: table S3.

The results for the regression of LBW on in-utero $PM_{2.5}$ level are shown in table 2. The reference group for the analysis is children with exposure in the lowest octile, with in-utero $PM_{2.5}$ level less than 26.7 $\mu g\ m^{-3}$. We find that exposure to higher in-utero $PM_{2.5}$ level has a negative, non-linear, and strong association with birth weight. In comparison to the reference group, children with in-utero $PM_{2.5}$ level above approximately 50 $\mu g\ m^{-3}$ especially weigh lesser at birth. After adjusting for various characteristics, children in the exposure band 51.6–63.5 $\mu g\ m^{-3}$ weigh 47.281 g lesser (95% CI: −82.944, −11.618) and those in the exposure band > 77.3 $\mu g\ m^{-3}$ weigh 57.866 g lesser (95% CI: −104.482, −11.251) than those in the reference group.

Exposure to higher in-utero $PM_{2.5}$ level is also associated with LBW. The adjusted odds ratio of LBW increases from 1.098 (95% CI: 0.954, 1.263) for children in the exposure band 39.3–44.7 $\mu g\ m^{-3}$—i.e. the fourth octile—to 1.241 (95% CI: 1.065, 1.447) for children in the exposure band 44.7–51.6 $\mu g\ m^{-3}$—i.e. the fifth octile—and 1.405 (95% CI: 1.126, 1.753) for children with exposure greater than 77.3 $\mu g\ m^{-3}$, i.e. the last octile (also see figure 2). Various sensitivity analyses based on control for antenatal visit, quality of information on birth weight, variation in control for birth year, variation in control for fixed effect, and functional form of the exposure confirmed that the findings are robust (supplementary information: figures S1–S2 and tables S5–S12).

The estimates for child level and maternal covariates are in the expected direction. Consider the regression of birth weight (supplementary information: table S4, column 1). Being the first child (β: −38.813; 95% CI: −48.467, −29.160), a twin or a triplet (β: −655.052; 95% CI: −693.116, −616.987), or a girl (β: −65.497; 95% CI: −71.386, −59.609) is associated with lower weight at birth. Similarly, being born to a mother who uses tobacco (β: −6.412; 95% CI: −19.478,6.655) or smokes *bidis* (β: −124.402; −185.604, −63.201) is associated with lower weight at birth. Also, in comparison to children whose mother did not complete primary school, children whose

Table 1. Descriptive statistics for the study sample.

	Mean or *n*	SD or %
Birth weight (g)	2819.32	603.86
Low birth weight		
No	123 530	83%
Yes	25 886	17%
In-utero $PM_{2.5}$ level (band)		
<26.7 $\mu g\ m^{-3}$	18 719	13%
26.7–34.1 $\mu g\ m^{-3}$	18 637	12%
34.1–39.3 $\mu g\ m^{-3}$	18 675	13%
39.3–44.7 $\mu g\ m^{-3}$	18 677	13%
44.7–51.6 $\mu g\ m^{-3}$	18 677	13%
51.6–63.5 $\mu g\ m^{-3}$	18 682	13%
63.5–77.3 $\mu g\ m^{-3}$	18 672	13%
>77.3 $\mu g\ m^{-3}$	18 677	13%
Use of solid cooking fuel		
No	48 691	33%
Yes	100 725	67%
Mother uses tobacco		
No	135 234	91%
Yes	14 182	9%
Mother smokes *bidis*		
No	149 060	100%
Yes	356	0%
Birth order (number)		
Case	2.19	1.36
Control	2.21	1.33
First child		
No	97 141	65%
Yes	52 275	35%
Multiple birth		
No	147 259	99%
Yes	2157	1%
Female child		
No	78 620	53%
Yes	70 796	47%
Short birth interval		
No	94 890	64%
Yes, <18 months	9367	6%
Yes, 18–35 months	45 159	30%
Teenage motherhood		
No	140 868	94%
Yes, age at birth <18 years	3237	2%
Yes, age at birth 18–19 years	5311	4%
Education level of mother		
None	39 079	26%
Primary	21 445	14%
Secondary	73 935	49%
Tertiary	14 957	10%
Maternal height <160 cm		
No	11 902	8%
Yes	137 514	92%
Maternal BMI <18.5 $kg\ m^{-2}$		
No	114 566	77%
Yes	34 850	23%
Mother has severe anemia		
No	148 077	99%
Yes	1339	1%
Improved sanitation facility		
No	71 990	48%
Yes	77 426	52%
Improved drinking water source		
No	18 129	12%
Yes	131 287	88%
Type of residence		
Rural	112 842	76%
Urban	36 574	24%
Wealth quintile in state		
Poorest	30 556	20%
Poor	32 562	22%
Middle	31 783	21%
Rich	29 957	20%
Richest	24 558	16%
Wealth quintile in country		
Poorest	32 983	22%
Poor	34 273	23%
Middle	31 981	21%
Rich	27 239	18%
Richest	22 940	15%
Number of observations		149 416

For continuous variables, the mean and standard deviation are presented. For discrete (categorical, binary) variables, the number of cases as well as the proportion for that category are presented.

mother completed primary education (β: 5.571; 95% CI: −5.615, 16.757), secondary education (β: 23.099; 95% CI: 13.571, 32.626), or tertiary education (β: 69.243; 95% CI: 54.936, 83.550) have higher weight at birth, *ceteris paribus*.

Surprisingly, however, household level characteristics such as use of solid cooking fuel (β: 5.181; 95% CI: −4.994, 15.356), improved sanitation facilities (β: 4.036; 95% CI: −5.308, 13.381), and improved drinking water source (β: 1.002; 95% CI: −9.545, 11.549)—which have been identified as risk factors for LBW in previous research [35, 36]—are not strongly associated with lower weight at birth in this analysis. This could be due to high correlation with ambient $PM_{2.5}$ concentration (for example, in the case of use of solid cooking fuel) or measurement error due to a lag between the birth of the child and the time of data collection. The estimates based on the regression of LBW are consistent with those based on the regression of birth weight (supplementary information: table S4, column 2).

In post estimation analysis, we computed the predicted probability of LBW at different values of in-utero $PM_{2.5}$ level (figure 3). The analysis indicates that if all children in the sample were exposed to in-utero $PM_{2.5}$ level exceeding 51.6 $\mu g\ m^{-3}$—i.e., sixth octile or higher—the prevalence of LBW might have been more than 0.19 (in comparison to sample mean of 0.17). In contrast, a reduction in the maximum in-utero exposure to less than 26.7 $\mu g\ m^{-3}$—i.e., the first octile—might reduce the prevalence of

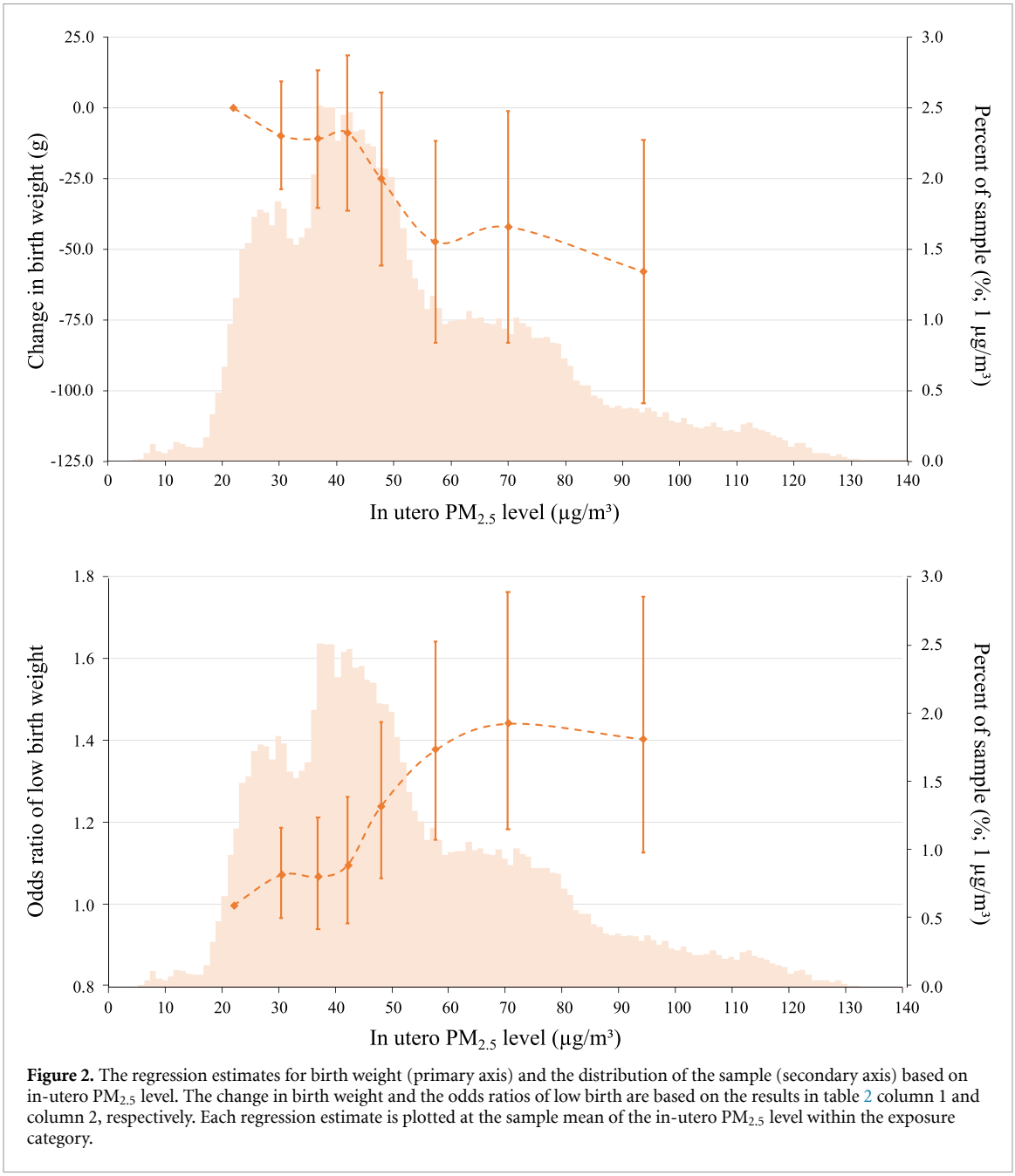

Figure 2. The regression estimates for birth weight (primary axis) and the distribution of the sample (secondary axis) based on in-utero $PM_{2.5}$ level. The change in birth weight and the odds ratios of low birth are based on the results in table 2 column 1 and column 2, respectively. Each regression estimate is plotted at the sample mean of the in-utero $PM_{2.5}$ level within the exposure category.

Table 2. The adjusted regression estimates of birth weight for in-utero $PM_{2.5}$ level band.

	Birth weight (1)	Low birth weight (2)
In-utero $PM_{2.5}$ level (reference: < 26.7 $\mu g\ m^{-3}$)		
26.7–34.1 $\mu g\ m^{-3}$	−9.640 [−28.757, 9.477]	1.074 [0.969, 1.190]
34.1–39.3 $\mu g\ m^{-3}$	−10.829 [−35.242, 13.583]	1.069 [0.942, 1.215]
39.3–44.7 $\mu g\ m^{-3}$	−8.692 [−36.235, 18.850]	1.098 [0.954, 1.263]
44.7–51.6 $\mu g\ m^{-3}$	−25.096 [−55.794, 5.602]	1.241 [1.065, 1.447]
51.6–63.5 $\mu g\ m^{-3}$	−47.281 [−82.944, −11.618]	1.378 [1.156, 1.643]
63.5–77.3 $\mu g\ m^{-3}$	−41.981 [−83.044, −0.918]	1.443 [1.182, 1.763]
>77.3 $\mu g\ m^{-3}$	−57.866 [−104.482, −11.251]	1.405 [1.126, 1.753]
Number of observations	149 416	149 416

The unit of observation is the child. Adjusted regression estimates are presented with their 95% confidence interval. For birth weight, the estimates are change in birth weight (in g) while for LBW the estimates are odds ratios. The regressions include birth month fixed effect, district fixed effect, and birth year fixed effect. The standard errors are clustered at the primary sampling unit (DHS cluster) level. The complete result is shown in supplementary information: table S4.

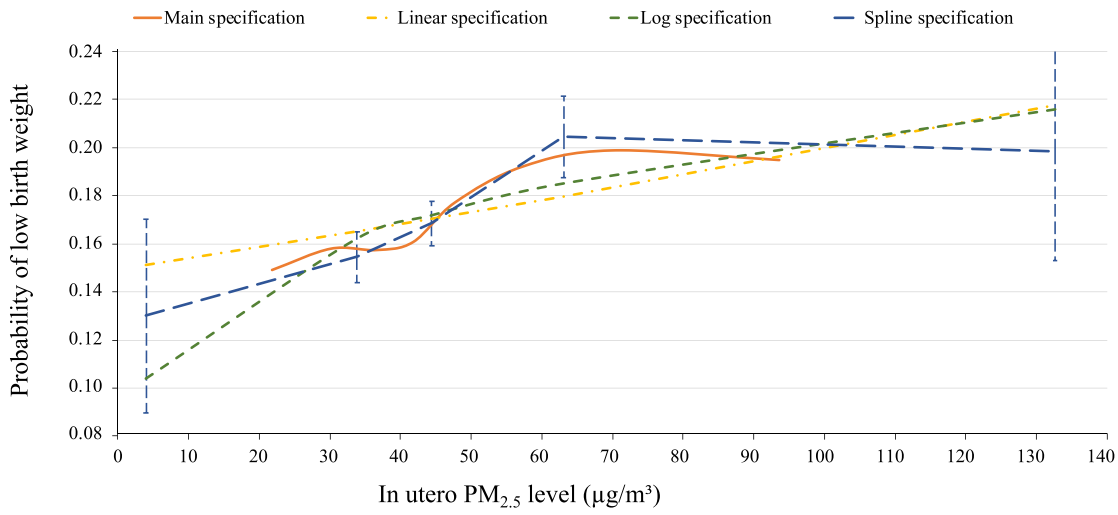

Figure 3. The probability of low birth weight based on variation in-utero $PM_{2.5}$ level. The values for the spline specification are accompanied by their 95% confidence interval. The figure is based on predicted marginal analysis, at existing values of other covariates, using the result in supplementary information: table S12.

LBW to 0.15. Further, we also observe that the CRC is non-linear. While our main specification based on exposure categories representing octiles of in-utero $PM_{2.5}$ exposure is consistent a spline specification, the linear and log specifications do not model the relationship accurately.

4. Discussion

In this study, we combine individual level data on child health in India with high-resolution spatial data on annual average ambient $PM_{2.5}$ concentration to calculate exposure to ambient $PM_{2.5}$ in-utero and examine the relationship between in-utero $PM_{2.5}$ level and LBW. We find that in-utero exposure to $PM_{2.5}$ level above approximately 50 $\mu g\ m^{-3}$ is associated with lower weight at birth. In addition, and relatedly, we observe that children with in-utero exposure to $PM_{2.5}$ level above 45 $\mu g\ m^{-3}$ have higher odds of LBW and the odds of LBW increase with an increase in $PM_{2.5}$ until approximately 70 $\mu g\ m^{-3}$ before leveling off. The findings are consistent even after adjusting for confounding variables at the child, maternal, and household level and controlling for unobserved spatial and temporal characteristics; further, they are robust to changes in model specification and study sample. Our findings suggest that a reduction in maximum ambient $PM_{2.5}$ level to approximately 25 $\mu g\ m^{-3}$ or less could reduce the prevalence of LBW to 15%, about 2% less than the sample mean of 17%.

Two other studies have examined the relationship between ambient $PM_{2.5}$ and birth weight in India. In a multi-country study that also covered India, Fleischer *et al* [17] investigated the association of ambient $PM_{2.5}$ with LBW using a cross-sectional design based on data on birth weight and satellite data on air pollution. Overall, they found that a higher $PM_{2.5}$ exposure level was associated with higher odds of LBW. However, their analysis on India indicated an inverse relationship between ambient $PM_{2.5}$ and LBW; they found that for India maternal $PM_{2.5}$ exposure above approximately 70 $\mu g\ m^{-3}$, in comparison to exposure below approximately 19 $\mu g\ m^{-3}$, was associated with lower odds of LBW (OR: 0.82; 95% CI: 0.75, 0.90). The contradiction between these findings and those of our study possibly stem from differences in the study design and methodology. Our scope is limited to India and we are to we use a larger, more representative sample based on individual birth data and incorporate subnational dummies and birth year fixed effect to account for spatial and temporal variation in LBW.

In a more recent study, Balakrishnan *et al* [18] analyzed the relationship between $PM_{2.5}$ exposure during pregnancy and birth weight amongst children in the Indian state of Tamil Nadu using a prospective cohort design based on direct measurement of $PM_{2.5}$ exposure performed across each trimester of pregnancy. They found that a 10 $\mu g\ m^{-3}$ increase in $PM_{2.5}$ level is associated with a 4 g (95% CI: 1.08, 6.76) decrease in birthweight and a 2% increase in the odds of LBW (OR: 1.02; 95% CI: 1.005, 1.041). In comparison, our study covered the entire country using a cross-sectional design based on geospatial data on annual average ambient $PM_{2.5}$ concentration. While we find evidence of a potentially stronger, but non-linear association of in-utero $PM_{2.5}$ level with (low) birth weight, our findings are broadly consistent with their study.

This study also complements existing research on the relationship between exposure to $PM_{2.5}$ during pregnancy and LBW in the rest of the world. In a meta-analysis of 32 such studies, mainly covering high-income settings in North America and Europe, Sun *et al* [13] found a strong association of LBW (OR = 1.090, 95% CI: 1.032, 1.150) with in-utero

$PM_{2.5}$ exposure (per 10 $\mu g\,m^{-3}$ increment). In a study conducted in a setting with ambient $PM_{2.5}$ level more comparable to that in India, Liang *et al* [37] examined the association between ambient $PM_{2.5}$ and LBW in nine Chinese cities. They estimated that a 10 $\mu g\,m^{-3}$ increase in ambient $PM_{2.5}$ was associated with a hazard ratio of 1.18–1.20 (95% 1.15, 1.23), depending on the trimester of exposure. Our findings are broadly consistent with these and present robust evidence for India.

This study has several limitations that must be borne in mind while interpreting its findings. First, the estimated annual average ambient $PM_{2.5}$ concentrations used for the analysis may be subject to measurement error due to the limited ground-based pollution monitoring network in India. Second, although we correct for the noise added to the GPS location of the DHS cluster, and find a 99% correlation between the annual average ambient $PM_{2.5}$ concentrations at the point location and over the displacement radius, we cannot rule out the possibility of classification error in exposure assessment. Third, the annual frequency of our exposure data means we are unable to identify how the exact timing of exposure affects birth weight; moreover, acute exposure or diurnal and seasonal variability in ambient $PM_{2.5}$ concentrations might affect the relationship between ambient $PM_{2.5}$ exposure and child health. Fourth, we are unable to measure the composition of the particulate matter, which might influence its effect on LBW. Fifth, our estimates might be biased by the concentrations of other pollutants such as nitrogen oxides and ozone, which may be correlated with ambient $PM_{2.5}$ concentrations; the estimates we report might, therefore, pertain to a more general relationship between ambient air pollution and health, rather than fine particulate matter specifically.

Sixth, as an observational study, our work is subject to omitted variable bias. Seventh, the missingness of data might also bias our estimates. Specifically, we see that the household level characteristics for observations with information on birth weight are different than those without (supplementary information: table S2). As children with missing information are more likely to be from households in lower wealth quintiles, with higher exposure to secondhand smoke due to maternal tobacco use and pollution due to the use of solid cooking fuel, it is plausible that our estimated association is conservative. Finally, in terms of the measurement of our outcomes, nearly half of the observations on birth weight in our sample are based on mother's recall and the indicator on LBW is, hence, subject to recall bias.

These limitations notwithstanding, this study was based on a large, nationally representative survey on women and child health in India and high-resolution spatial data on ambient $PM_{2.5}$ concentration. Further, it adjusted for several of child level, maternal, and household level characteristics, and incorporated birth month effect, birth year fixed effect, and district dummies to account for spatial and temporal variation in unobserved characteristics. Its findings shed light on the relationship between ambient air pollution and birth weight in India and can inform strategies for reducing low birth weight and improving child health.

Data availability statement

The data that support the findings of this study are available upon reasonable request from the authors.

Acknowledgments

N G thanks the Lee Kuan Yew School of Public Policy, National University of Singapore for the Graduate Research Scholarship and the Fox family and the Whitney and Betty MacMillan Center for International and Area Studies, Yale University for the Fox International Fellowship.

Author contributions

Conceptualization, N G and D C; methodology, N G and D C; formal analysis, N G; data curation, N G; writing—original draft preparation, N G; writing—review and editing, N G and D C; visualization, N G and D C.

Conflicts of interest

The authors declare no conflict of interest.

ORCID iDs

Nihit Goyal https://orcid.org/0000-0002-1025-7585
David Canning https://orcid.org/0000-0003-4041-1229

References

[1] UNICEF Data 2019 Low birth weight May 2019 (available at: https://data.unicef.org/topic/nutrition/low-birthweight/) (Accessed 19 April 2020)
[2] Risnes K R *et al* 2011 Birthweight and mortality in adulthood: a systematic review and meta-analysis *Int. J. Epidemiol.* **40** 647–61
[3] Blencowe H *et al* 2019 National, regional, and worldwide estimates of low birthweight in 2015, with trends from 2000: a systematic analysis *Lancet Global Health* **7** e849–e60
[4] World Health Organization 2015 Global nutrition targets 2025: low birth weight policy brief. 2014 *Glob. Nutr. Targets* **2025**
[5] WHO 2016 *Ambient Air Pollution: A Global Assessment of Exposure and Burden of Disease* (Geneva: World Health Organization)
[6] Murray C J L *et al* 2020 Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019 *Lancet* **396** 1223–49

[7] Shamsipour M *et al* 2019 National and sub-national exposure to ambient fine particulate matter ($PM_{2.5}$) and its attributable burden of disease in Iran from 1990 to 2016 *Environ. Pollut.* **255** 113173
[8] Goyal N and Canning D 2018 Exposure to ambient fine particulate air pollution in utero as a risk factor for child stunting in Bangladesh *Int. J. Environ. Res. Public Health* **15** 22
[9] Heinrich J and Slama R 2007 Fine particles, a major threat to children *Int. J. Hyg. Environ. Health* **210** 617–22
[10] Mehta S, Shin H, Burnett R, North T and Cohen A J 2013 Ambient particulate air pollution and acute lower respiratory infections: a systematic review and implications for estimating the global burden of disease *Air Qual. Atmos. Health* **6** 69–83
[11] Xue T, Zhu T, Geng G and Zhang Q 2019 Association between pregnancy loss and ambient $PM_{2.5}$ using survey data in Africa: a longitudinal case-control study, 1998–2016 *Lancet Planet. Health* **3** e219–ee25
[12] Klepac P, Locatelli I, Korošec S, Künzli N and Kukec A 2018 Ambient air pollution and pregnancy outcomes: a comprehensive review and identification of environmental public health challenges *Environ. Res.* **167** 144–59
[13] Sun X, Luo X, Zhao C, Zhang B, Tao J, Yang Z, Ma W and Liu T 2016 The associations between birth weight and exposure to fine particulate matter ($PM_{2.5}$) and its chemical constituents during pregnancy: a meta-analysis *Environ. Pollut.* **211** 38–47
[14] Pedersen M *et al* 2013 Ambient air pollution and low birthweight: a European cohort study (ESCAPE) *Lancet Respir. Med.* **1** 695–704
[15] Van Donkelaar A, Martin R V, Brauer M, Hsu N C, Kahn R A, Levy R C, Lyapustin A, Sayer A M and Winker D M 2016 Global estimates of fine particulate matter using a combined geophysical-statistical method with information from satellites, models, and monitors *Environ. Sci. Technol.* **50** 3762–72
[16] Kelly F J and Fussell J C 2012 Size, source and chemical composition as determinants of toxicity attributable to ambient particulate matter *Atmos. Environ.* **60** 504–26
[17] Fleischer N L, Merialdi M, van Donkelaar A, Vadillo-Ortega F, Martin R V, Betran A P and Souza J P 2014 Outdoor air pollution, preterm birth, and low birth weight: analysis of the world health organization global survey on maternal and perinatal health *Environ. Health Perspect.* **122** 425–30
[18] Balakrishnan K *et al* 2018 Exposures to fine particulate matter ($PM_{2.5}$) and birthweight in a rural-urban, mother-child cohort in Tamil Nadu, India *Environ. Res.* **161** 524–31
[19] Every Preemie–SCALE 2019 India: profile of preterm and low birth weight prevention and care (available at: www.healthynewbornnetwork.org/hnn-content/uploads/India-1.pdf) (Accessed 19 April 2020)
[20] Khan N, Mozumdar A and Kaur S 2020 Determinants of low birth weight in India: an investigation from the National Family Health Survey *Am. J. Hum. Biol.* **32** e23355
[21] Bharati P, Pal M, Bandyopadhyay M, Bhakta A, Chakraborty S and Bharati P 2011 Prevalence and causes of low birth weight in India *Malays J. Nutr.* **17** 301–13
[22] Griffiths J 2019 22 of the top 30 most polluted cities in the world are in India *CNN*
[23] Rutstein S O and Rojas G 2006 *Guide to DHS Statistics* (Calverton, MD: ORC Macro)
[24] International Institute for Population Sciences II, ICF 2017 *India National Family Health Survey NFHS-4 2015–16* (Mumbai: IIPS and ICF)
[25] Dey S, Di Girolamo L, van Donkelaar A, Tripathi S N, Gupta T and Mohan M 2012 Variability of outdoor fine particulate ($PM_{2.5}$) concentration in the Indian Subcontinent: a remote sensing approach *Remote Sens. Environ.* **127** 153–61
[26] Burgert C R, Colston J, Roy T and Zachary B 2013 *Geographic Displacement Procedure and Georeferenced Data Release Policy for the Demographic and Health Surveys* (Calverton, MD: ICF International)
[27] Perez-Haydrich C, Warren J L, Burgert C R and Emch M E 2013 *Guidelines on the Use of DHS GPS Data* (Calverton, MD: ICF International)
[28] Burnett R T *et al* 2014 An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure *Environ. Health Perspect.* **122** 397–403
[29] Tuomisto J T, Wilson A, Evans J S and Tainio M 2008 Uncertainty in mortality response to airborne fine particulate matter: combining European air pollution experts *Reliab. Eng. Syst. Saf.* **93** 732–44
[30] Lelieveld J, Evans J S, Fnais M, Giannadaki D and Pozzer A 2015 The contribution of outdoor air pollution sources to premature mortality on a global scale *Nature* **525** 367
[31] Faridi S, Niazi S, Yousefian F, Azimi F, Pasalari H, Momeniha F, Mokammel A, Gholampour A, Hassanvand M S and Naddafi K 2019 Spatial homogeneity and heterogeneity of ambient air pollutants in Tehran *Sci. Total Environ.* **697** 134123
[32] Pant P, Guttikunda S K and Peltier R E 2016 Exposure to particulate matter in India: a synthesis of findings and future directions *Environ. Res.* **147** 480–96
[33] Guttikunda S K, Goel R and Pant P 2014 Nature of air pollution, emission sources, and management in the Indian cities *Atmos. Environ.* **95** 501–10
[34] Guo H, Kota S H, Sahu S K, Hu J, Ying Q, Gao A and Zhang H 2017 Source apportionment of $PM_{2.5}$ in North India using source-oriented air quality models *Environ. Pollut.* **231** 426–36
[35] Epstein M B, Bates M N, Arora N K, Balakrishnan K, Jack D W and Smith K R 2013 Household fuels, low birth weight, and neonatal death in India: the separate impacts of biomass, kerosene, and coal *Int. J. Hyg. Environ. Health* **216** 523–32
[36] Tielsch J M, Katz J, Thulasiraj R D, Coles C L, Sheeladevi S, Yanik E L and Rahmathullah L 2009 Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in south India *Int. J. Epidemiol.* **38** 1351–63
[37] Liang Z, Yang Y, Qian Z, Ruan Z, Chang J, Vaughn M G, Zhao Q and Lin H 2019 Ambient PM(2.5) and birth outcomes: estimating the association and attributable risk using a birth cohort study in nine Chinese cities *Environ. Int.* **126** 329–35
[38] Goyal N, Karra M and Canning D 2019 Early-life exposure to ambient fine particulate air pollution and infant mortality: pooled evidence from 43 low- and middle-income countries *Int. J. Epidemiol.* **48** 1125–41