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Air pollution and child development in India

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Air Pollution and Child Development in India

Abstract

In this paper, we study the impact of air pollution on child growth in India. We rely on wind direction to capture quasi-random variation in three main criteria air pollutants. We show that an increase in the average concentration of fine particulate matter by one standard deviation is accountable for almost 5 and 2.4 percentage points of stunting and severe stunting rates, respectively. We also find that ozone and carbon monoxide impact weight-related outcomes. Stunting has critical long-term health and economic consequences; through its impact on stunting, pollution exacerbates the height premium in earnings, with girls being more adversely affected than boys in India.

Keywords: Ambient air pollution; child health; anthropometry; wind direction; height premium.

JEL Classification Codes: C31, C36, I15, I32, Q53.

1 Introduction

Air pollution is a global health threat with 7 million deaths attributable to the joint effects of household and ambient air pollution in 2016 (WHO, 2016; Cohen et al., 2017). About 94% of these deaths occur in low- and middle-income countries. In addition, air pollution causes acute respiratory infections, blindness, heart diseases, low birth weight and stillbirth (Branca and Ferrari, 2002; Dewey and Begum, 2011; Hodinott et al., 2013; Feng et al., 2019).

In economics, a large body of literature attempts to document the causal impacts of pollution on different health outcomes (Chay and Greenstone, 2003; Currie and Neidell, 2005; Currie et al., 2009; Jayachandran, 2009; Currie and Walker, 2011; Moretti and Neidell, 2011; Arceo et al., 2016; Knittel et al., 2016; Schlenker and Walker, 2016; Deryugina et al., 2019). An overwhelming share of these studies focuses on the developed world, exploring the link between pollution and mortality or respiratory and heart-related hospitalizations.

Using data from India, this paper studies the impact of air pollution on child growth indicators, such as stunting and wasting. Children are stunted for their age and gender when their height is more than two standard deviations below the World Health Organization (WHO) Child Growth Standards median. Stunting is of high relevance for public health and economic policies, being associated with critical short- and long-term physical and socio-economic disadvantages (for a review, see [Currie and Vogl, 2013](#)). In the short term, stunting is linked to (i) weakened immune systems and higher risk of infection ([Rodríguez et al., 2011](#); [Schlaudecker et al., 2011](#)); (ii) lower cognitive development ([Pollitt et al., 1995](#); [Brown and Pollitt, 1996](#); [Case and Paxson, 2008a](#)); (iii) adverse educational achievements ([Maluccio et al., 2009](#); [Hoddinott et al., 2013](#)); and (iv) higher mortality rates ([Caulfield et al., 2004](#); [Olofin et al., 2013](#)). Evidence on the negative long-term consequences of stunting is growing, with stunted children being more likely to experience (i) short stature in adulthood ([Tanner et al., 1956](#); [Sachdev et al., 2005](#); [Gigante et al., 2009](#)); (ii) reduced cognitive skills ([Glewwe and Miguel, 2007](#); [Guven and Lee, 2015](#)), (iii) functional limitations, such as reduced work capacity ([Spurr, 1988](#)); (iv) higher risks of obesity and chronic diseases ([Gluckman et al., 2007](#)); (v) lower income and wealth ([Hoddinott et al., 2008](#); [Case and Paxson, 2008a](#); [Almond and Currie, 2010](#)), (v) poorer marriage outcomes ([Hoddinott et al., 2013](#)); and (vi) worse reproductive outcomes, such as lower birthweight of offsprings ([Victora et al., 2008](#)), having firstborns at younger ages, and more pregnancies and children ([Hoddinott et al., 2013](#)).

Air pollution is expected to affect child growth and development through its impact on respiratory diseases and the loss of nutrients when fighting infectious agents ([Schlaudecker et al., 2011](#)). The existing literature has so far focused on estimating the correlation between child stunting and air pollution, in particular household air pollution. When assessing the link between air pollution and health outcomes, OLS estimates are likely biased due to measurement errors and omitted variables ([Duflo et al., 2008b](#); [Greenstone and Jack, 2015](#); [Deryugina et al., 2019](#)). Households are usually not randomly exposed to different levels of air pollution, in particular indoor pollution, and their exposure correlates with a multitude of factors that are also likely to impact child health. For example, the use of specific fuel types for cooking, which directly impacts air pollution levels, correlates highly with household wealth and education. Poorer households may be more likely to have both stunted children and choose free or cheap fuel sources (like dung or wood gathered from nearby fields or forests) ([Duflo et al., 2008a](#)).

This study focuses on India as a case-in-point for a developing economy with persistently high levels of stunting and increasing air pollution ([Greenstone and Hanna, 2014](#); [Banerjee and Dwivedi, 2020](#)). We combine anthropometric and socio-economic data from the Indian National Family Health Survey 2015/2016 (hereafter NFHS-4) with granular information on air pollutant concentrations, wind direction, wind speed, and weather variables from the second Modern-Era Retrospective analysis for Research and Applications (MERRA-2) from NASA's atmospheric reanalysis of satellite data. This unique dataset allows us to estimate the causal impact of air pollution on child growth by exploiting the quasi-random variation in pollution attributable to the transportation of pollutants by wind. We focus on three criteria pollutants, *viz.* fine particulate matter (PM_{2.5}), carbon monoxide (CO), and ozone (O₃), and show their disparate impacts on height- and weight-related child growth outcomes.

Based on a long time-series of hourly data, we build aggregate measures of frequency of wind direction and pollution concentrations that reflect the personal exposure of each child in our sample from the gestational period till the time of the interview. Employing an instrumental variable (IV) approach, we use the exogenous variation in wind direction to instrument for local pollution concentrations and estimate the impact of pollution on anthropometric measurements for children.

With our methodology, we join the recent literature that explores changes in wind direction as an exogenous source of variation in air pollution for assessing health impacts. [Deryugina et al. \(2019\)](#) use changes in daily average wind direction as an instrument for variation in daily average fine particulate matter concentrations to identify its effect on mortality and health care use in the US. [Anderson \(2020\)](#) investigates the impact of air pollution on adult mortality by using variation in wind orientation with respect to highways in the Los Angeles area. [Sheldon and Sankaran \(2017\)](#) use wind direction and the occurrence of Indonesian fires to instrument air pollution in Singapore and study the impact of pollution on respiratory diseases. Similar identification strategies have also been used when researching the impact of wildfires on different health outcomes ([Jayachandran, 2009](#); [Pullabhotla, 2018](#); [Rangel and Vogl, 2019](#)).

Our analysis shows that an increase by one standard deviation in average annual outdoor PM_{2.5} concentrations increases the frequency of stunting and severe stunting by 5 and 2.4 percentage points, respectively. We also find that ozone and carbon monoxide impact weight-related outcomes. Back-of-the-envelope estimates suggest that, if average pollution levels in India were brought down to WHO recommended stan-

dards, i.e., an eightfold reduction from current levels, the shares of stunted and severe stunted children would decrease by 10.4 and 5.17 percentage points, respectively, *ceteris paribus*. This would correspond to approximately 14.3 million less stunted children in India.

We perform a series of heterogeneity analyses by age, gender, birth order, and place of residence and show how impacts can vary across these dimensions, pointing, among others, to important cultural practices that shape the effects of pollution on child growth. Moreover, we rely on an aggregated air quality index to estimate the joint impact of the different criteria pollutants on the child growth indicators. A series of tests demonstrates the robustness of our results. The findings are stable to changing the aggregation level of the data and accounting for spatial correlation in the error terms. Moreover, the results are consistent when employing an alternative IV approach, where we restrict the sample to include only children living close to the coastline and use the share of time wind is blowing from the sea as an instrument for air pollution.

The consequences of air pollution exposure, similarly to other health shocks during childhood, are expected to be long-lasting. Based on estimates from the literature, we compute the additional contribution of air pollution exposure to the well-known height premium in earnings and find that an increase in $PM_{2.5}$ by one standard deviation during childhood translates into lost yearly earnings in adulthood in the range of 1.4 – 1.8% for men and 1.6 – 2% for women, pointing to a potential new dimension of the gender gap in India.

Our paper contributes to the existing literature by being, to the best of our knowledge, one of the first papers to perform a causal estimation of the impacts of air pollution on child growth outcomes. Moreover, it focuses on India, one of the largest emerging economies in the world and currently among the most polluted places. So far, only a handful of economic papers have focused on identifying the causal impact of air pollution on health outcomes in a developing country context (Jayachandran, 2009; Chen et al., 2013; Arceo et al., 2016; Deschenes et al., 2020). Existing evidence comes from Indonesia, Mexico, and China with a focus on infant and elderly mortality. If the pollution - health relation is convex or the costs of avoidance behavior are different in developing countries (Arceo et al., 2016), it is important to understand what are the impacts at high levels of pollution. Our paper is somewhat close to Deschenes et al. (2020), who use thermal inversions to instrument for the impact of $PM_{2.5}$ on adult weight and obesity in China. In contrast to their findings, we find no evidence that

PM_{2.5} significantly affects the weight-for-age and weight-related growth outcomes of children in India; however, we do find that these outcomes are negatively impacted by ozone and carbon monoxide, two other important criteria pollutants not considered in their analysis. Our paper is also one of the first to study the joint impact of the three main criteria pollutants on child health, decreasing the risk of overestimating the impact of one particular pollutant when included separately, as in most previous studies.

Our results indicate that the negative impacts of pollution on child development are substantial, underlying once again the urgency to reduce air pollution in India. In light of recent evidence that the willingness to pay for clean air is extremely low in India (Greenstone et al., 2021), it seems that change will not be led by, at least in the short term, citizen-led initiatives, but needs to be mediated by policy makers.

The remainder of the paper is organized as follows. Section 2 describes the conceptual framework of the article. Section 3 describes the data. Section 4 introduces the proposed identification strategy to tackle endogeneity issues. Sections 5 and 6 present the main results, the heterogeneity analysis, and the robustness checks. Section 7 estimates the additional impacts of pollution on the height premium in earnings, while Section 8 concludes.

2 Conceptual Framework

This section describes the selected outcome variables that capture growth and developmental impairments in children. In particular, we explain the mechanisms evoked in the medical literature linking air pollution and reduced child growth.

2.1 Measuring Physical Growth Deficiencies in Children

In a well-nourished and healthy population, there is a statistically predictable distribution of height and weight for children of a given age. The standard index used for physical growth — height-for-age — reflects the long-term effects of genetic and environmental conditions and gene-environment interactions on skeletal growth. The height-for-age (HAZ) measure is expressed in standard deviation units (z-score) as the difference between the current height and the median of the reference popula-

tion:

$$HAZ_i = \frac{\text{Height}_i - \text{Median}(\text{reference population of same age and gender})}{\text{Standard Deviation}(\text{reference population of same age and gender})} \quad (1)$$

where HAZ_i is the height-for-age indicator of child i , whose height is given by Height_i . The WHO classifies children with a HAZ score below -2 as being stunted and below -3 as severely stunted.¹ Another standard measure of growth relates weight and age. The weight-for-age (WAZ) index is expressed in standard deviation units from the median of a reference population and is computed similarly to the HAZ. A WAZ score below -2 indicates being wasted and below -3 being severely wasted.

Although HAZ and WAZ are both measures of restricted growth, they tend to indicate different stages of the condition. While wasting is linked to very low muscle tissue and fat mass for a certain age, stunting reveals a significantly slowed skeletal development (Ricci and Becker, 1996). Impacts on body mass can be transitory, caused by recent diseases and limited dietary intake. In contrast, stunting reflects skeletal growth impairments that tend to be the result of repeated acute illnesses and nutritional deficiency, capturing long-term irreversible growth impairments (Ricci and Becker, 1996; Subramanyam et al., 2010). In this study, we show results for both height- and weight-related growth indicators.

2.2 Air Pollution and Growth Deficiencies

Long-term exposure to air pollution, in particular fine particulate matter, is detrimental to human health, including cardio-respiratory health endpoints, such as chronic obstructive pulmonary disease, ischemic heart disease, stroke, lung cancer, and child acute lower respiratory infections (Burnett et al., 2018; Upadhyay et al., 2015, among many others). Sinharoy et al. (2020) offer a recent summary of the medical literature dedicated to explaining the physiological channels linking air pollution and child health.

Respiratory infections lead to the activation of the immune system to fight off disease-

¹We rely on the most recent international reference population, which was released by the WHO in January 2018. The standard for the reference population is based on children of non-smoking mothers around the world (Brazil, Ghana, India, Norway, Oman, and the USA), who are raised in healthy environments and are fed with recommended feeding practices (exclusive breastfeeding for the first 6 months and appropriate complementary feeding from 6 to 24 months). The z-score of the reference population is normally distributed; then, a child in the reference population will have a chance of less than 2.3% to be stunted (Imai et al., 2014).

causing agents, which requires a broad range of nutrients to defend against pathogens (Dewey and Mayers, 2011). For children, the problem is reinforced by the fact that their immune system is still developing and weaker when fighting infectious agents (Rodríguez et al., 2011). The necessary metabolic energy consumed to fight the disease is diverted away from growth (Schlaudecker et al., 2011).

Repeated and lasting exposure to air pollution also leads to sub-clinical biological responses similar to mechanisms resulting from poor sanitation and lack of hygiene and water purity, in which gut barrier function is reduced by pathogens, and which induces chronic immune activation, increased inflammation and resistance of growth hormones (Sanitation Hygiene Infant Nutrition Efficacy (SHINE) Trial Team et al., 2015).

Figure 1 depicts the relation between the rate of stunting and average lifetime ambient fine particulate matter (PM_{2.5}) exposure for the Indian children 0 - 5 years old in our sample. The basic pattern is striking: the higher the PM_{2.5} concentrations, the higher the share of stunted children, with a steeper effect in rural areas, likely due to more limited access to health care and other confounding factors. This paper aims to identify the relationship between pollution concentrations and child growth outcomes, net of other socio-economic and behavioral factors.

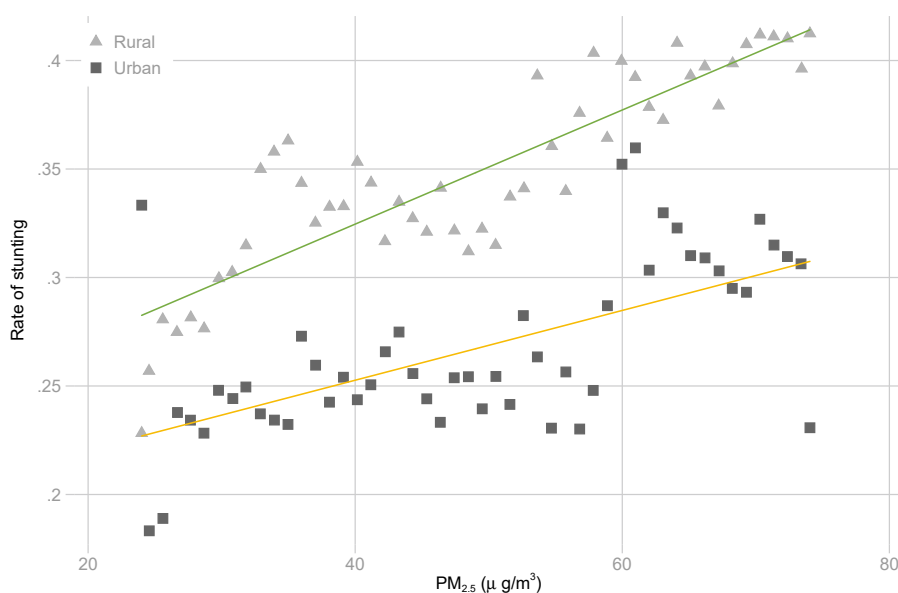


Figure 1: PM_{2.5} concentrations and shares of stunted children in urban and rural India.

Note: The figure plots stunting rates in 50 bins of PM_{2.5} concentrations, separately in urban and rural areas. The colored lines represent linear regression lines separately for urban (in orange) and rural (in green) areas. Data source: the 2015/2016 India National Family Health Survey (NFHS-4).

3 Data

We combine different sources of data to study the impact of air pollution on child growth in India. The 2015/2016 National Family Health Survey (NFHS-4) contains information on the socio-economic background of children and their parents, as well as anthropometric information on child development. We take advantage of the georeferenced information included in the household surveys² and link it to satellite data on air pollution, wind direction and speed, and related weather variables. Our identification strategy relies on temporal and spatial variation in wind direction for which the available data has a longitudinal-latitudinal resolution of $0.625^\circ \times 0.5^\circ$.³ Our main

²While there have been three previous waves of the NFHS, NFHS-4 is the first that provides geocoordinates of the primary sampling units (PSUs) where interviewed households reside. These coordinates are geomasked for ethical reasons, using a displacement buffer of up to two kilometers for urban PSUs and up to five kilometers for rural ones. As our main unit of analysis is cells with longitudinal-latitudinal resolution of $0.625^\circ \times 0.5^\circ$, such geomasking is not a big concern for our specifications.

³Given India's latitudinal and longitudinal coordinates, cell areas vary between 3,157 and 3,798 km², or polygons with edges of roughly 69×55 km.

specifications are at the individual child level to which we match the gridded environmental data.

3.1 Anthropometric and Socio-economic Data

We use data from India's NFHS-4 conducted in 2015/2016 – the most recent available – collected through the Demographic and Health Surveys (DHS) Program.

For each child in the NFHS-4 dataset, we match cell-level environmental variables that reflect personalized averages over the lifetime of the child. To account for the well-known impacts of pollution and climate variables also during the gestation period (Goyal and Canning, 2017; Sanders, 2012), we average hourly data starting nine months before the birth of the child⁴ up to the date of the interview. Our results are robust to including only the period after the actual birth.⁵

Our final dataset consists of 223,049 children aged 0 - 5 years old for which we have complete anthropometric measures and relevant socio-economic information. Table 1 presents key summary statistics characterizing the children in our sample. All child characteristics reflect averages at the child level, while household characteristics reflect household-level statistics. Overall, stunting affects about a third of all children in India, while severe stunting affects almost 13%. The incidence of wasting and severe wasting is about 40% and 10%, respectively. Looking at the age distribution, the youngest children aged 0-12 months old appear to fare best in terms of all growth indicators, despite coming from households with very similar profiles to those in other age groups. This is suggestive of how stunting (and its severe form) indicates *cumulative* growth deficiencies and a general worsening of health as the child ages, despite being exposed to constant living conditions.

⁴We call this the *date of concept*, but we can only estimate the month of concept, not the precise day. The analysis abstracts from accounting for early births.

⁵This approach assumes that a child has lived in the same cell since birth till the date of the interview. To check the strength of this assumption, we compare the age of each child with the reported number of years of residence in the current household. Over the entire sample, about 8.2% of children appear to have changed residence during their lifetime and only 0.4% to have only very recently moved in, although we do not know if relocation took place within the same cell or from another cell.

Table 1: Summary statistics of variables in the 2015-2016 India National Family and Health Survey IV (NFHS-4).

	Age group 1	Age group 2	Age group 3	Age group 4	Age group 5	All
1. Child characteristics						
Height-for-age score (HAZ)	-0.623 (1.594)	-1.602 (1.589)	-1.281 (1.620)	-1.520 (1.500)	-1.607 (1.390)	-1.323 (1.587)
Stunted	0.175 (0.380)	0.409 (0.492)	0.313 (0.464)	0.371 (0.483)	0.374 (0.484)	0.328 (0.469)
Severely stunted	0.0631 (0.243)	0.168 (0.374)	0.122 (0.327)	0.142 (0.349)	0.147 (0.354)	0.128 (0.334)
Weight-for-age score (WAZ)	-1.068 (1.269)	-1.824 (1.171)	-1.781 (1.176)	-1.745 (1.078)	-1.722 (1.054)	-1.628 (1.187)
Wasted	0.225 (0.418)	0.469 (0.499)	0.456 (0.498)	0.426 (0.494)	0.418 (0.493)	0.399 (0.490)
Severely wasted	0.0536 (0.225)	0.135 (0.342)	0.131 (0.337)	0.102 (0.303)	0.0898 (0.286)	0.103 (0.303)
Child age (months)	6.496 (3.435)	18.44 (3.421)	30.50 (3.455)	42.44 (3.437)	53.99 (3.139)	30.09 (16.98)
Child is a boy	0.523 (0.499)	0.517 (0.500)	0.518 (0.500)	0.515 (0.500)	0.525 (0.499)	0.520 (0.500)
2. Household characteristics						
Household wealth index	-0.133 (0.964)	-0.129 (0.968)	-0.140 (0.964)	-0.156 (0.972)	-0.169 (0.972)	-0.145 (0.968)
Nightlights Index	2.912 (7.453)	3.000 (7.679)	3.019 (7.624)	3.051 (7.636)	3.075 (7.697)	3.011 (7.617)
No. of household members	6.769 (2.903)	6.515 (2.883)	6.512 (2.843)	6.535 (2.872)	6.468 (2.796)	6.561 (2.863)
Hindu	0.733 (0.442)	0.722 (0.448)	0.723 (0.448)	0.726 (0.446)	0.729 (0.444)	0.727 (0.446)
No. of children	44,994	44,581	45,392	47,042	41,040	223,049

Note: The table captures summary statistics of key variables of interest in the 2015-2016 India National Family and Health Survey IV (NFHS-4). The statistics show mean values and standard deviation (in parentheses) by age group (1-5 years). Outcome variables and children's characteristics have been aggregated from data at the child level, whereas household and maternal characteristics are from data at the household level. NFHS-4 gathered information on a total of 265,653 children living in the households interviewed. Due to missing or fully out-of-range values for some key variables of interest, our final estimation sample includes 223,049 children, fairly well-balanced across the different age groups.

Household and child characteristics are well-balanced across the different age groups, with similar wealth, family size, and religion. The nightlights index further indicates similarity in average economic development and population density.

3.2 Air Pollution and Weather Data

We focus on the criteria pollutants such as fine particulate matter (PM_{2.5}), ground-level ozone (O₃), carbon monoxide (CO), sulphur dioxide (SO₂), and nitrogen dioxide (NO₂). These five pollutants have a significant detrimental impact on human health (Geyh et al., 2000; Suh et al., 2000; Townsend and Maynard, 2002; Currie and Neidell, 2005; Chen et al., 2007; Moretti and Neidell, 2011; Schlenker and Walker, 2016) and their values are usually daily monitored around the globe and reported to the general public in the form of an air quality index (AQI) for preventive use. High concentrations of air pollution increase the risk of respiratory diseases, cardiovascular mortality, and cardiopulmonary illnesses.

In India, as in many emerging and developing countries, ground-level information on air pollution is sparsely collected and not representative of the national profile (Brauer et al., 2019).⁶ To obtain a representative quantification of air pollution, we use satellite data from NASA's second Modern-Era Retrospective analysis for Research and Applications (MERRA-2) database, with a longitudinal-latitudinal resolution of $0.625^\circ \times 0.5^\circ$ and available at hourly frequency.⁷ Accounting for all cells where at least one PSU has been surveyed by the NFHS-4, our spatial structure consists of 987 cells.⁸

PM_{2.5} is the standard indicator to measure the impacts of air pollution on health (WHO, 2016) and, often, the only included measure of air pollution in empirical analyses. Depending on location and emission sources, major composites of PM_{2.5} can include windblown mineral dust (DS_{2.5}), sea salt (SS_{2.5}), organic carbon (OC), black carbon (BC), and particles derived from the oxidation of primary gases like sulfur and nitrogen oxide, such as sulfate (SO₄) (Perraud et al., 2012; Dominici et al., 2015). Main sources of PM_{2.5} are fossil fuels combustion, biomass burning, and biofuel consumption, with additional biogenic sources of particulate organic matter (Koster, 2016).⁹

⁶In 2010-2016, about 200 ground monitors collected data on PM_{2.5} in India, a monitor density much lower than in China and Brazil (Brauer et al., 2019).

⁷A few recent studies compare satellite-derived PM_{2.5} against surface measurements from the Central Pollution Control Board (CPCB) India network (Dey et al., 2020; Navinya et al., 2020) and find high correlations for the available sites.

⁸Fig. A.1 in the Appendix illustrates the number of PSUs interviewed in each cell, with a median of 22 PSUs per cell.

⁹Black carbon is emitted as soot including soot from coal burning, internal-combustion engines, power-plant boilers, central steam-heat boilers, waste treatment by combustion, field burning, forest fires, fireplaces, and furnaces among others (Omidvarborna et al., 2015). In the environment, sulfate (SO₄) is produced from the oxidation of elemental sulfur, sulfide minerals, or organic sulfur. Industrially, sulfate is produced during the burning of sulfur-containing fossil fuels, household wastes (e.g.,

PM_{2.5} values are not readily available in MERRA-2, but can be estimated based on values provided for its composites, applying a mass reconstruction method.¹⁰ We follow Provençal et al. (2017) and calculate PM_{2.5} as:

$$PM_{2.5} = BC + 1.8 \times OC + DS_{2.5} + SS_{2.5} + 1.375 \times SO_4. \quad (2)$$

Ozone and carbon monoxide mixing ratios are available from MERRA-2 with the same spatial and temporal resolution as the PM_{2.5} composites. On one hand, tropospheric (ground-level) ozone is a result of chemical reactions between oxides of nitrogen (NO_x) and volatile organic compounds (VOC), emitted by cars, power plants, industrial boilers, refineries, and chemical plants (US EPA, 2021). Ozone levels usually peak in urban areas during dry hot days. On the other hand, carbon monoxide results from the incomplete oxidation of carbon in combustion. One of the main sources of CO in India is the burning of biomass and other solid fuels and kerosene for cooking and heating of indoor spaces. CO levels usually peak during the cold season in rural areas.

Table 2 presents summary statistics of the different air pollutants. The national yearly average PM_{2.5} concentrations over 2010 - 2016 are about 41 µg/m³, more than eight times the WHO recommended level. The average SO₂ concentrations across the country are less worrying, being below the levels recommended by the European Environment Agency (EEA).¹¹ Fig. 2 indicates significant spatial variations in the PM_{2.5} average distribution, with the industrialized North facing, on average, much higher pollution.¹²

detergents), and effluents from tanneries, steel mills, sulfate-pulp mills, and textile plants. 80% of estimated commercial sulfur production comes from sulfuric acids (Appel et al., 2005).

¹⁰The MERRA-2 reconstructed values for PM_{2.5} are highly consistent with the Van Donkelaar et al. (2016) PM_{2.5} measurements, another satellite-based widely used dataset available only at yearly frequency (Fig. A.3 in the Appendix).

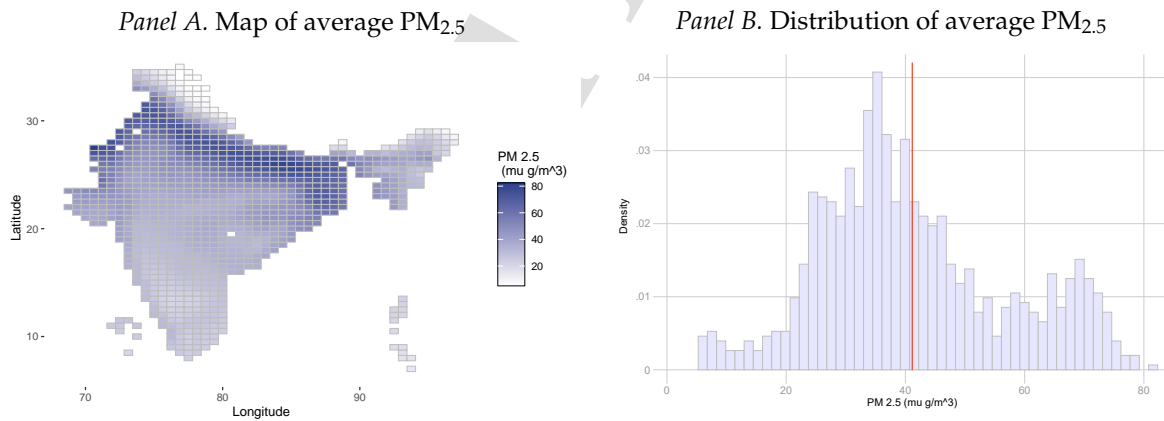
¹¹The WHO and EEA recommendations in terms of O₃ and CO are based on shorter time scales (8-hour or 24-hour), which makes comparison to the annual average level less appropriate.

¹²The spatial and seasonal distribution of average CO and O₃ levels is captured in Fig. A.2 in the Appendix.

Table 2: Summary statistics of air pollution and weather variables from MERRA-2.

	Mean	SD	Min	Median	Max
Pollutant surface mass concentration					
PM _{2.5} ($\mu\text{g}/\text{m}^3$)	41.146	24.835	1.331	36.450	318.977
CO ($\mu\text{g}/\text{m}^3$)	113.389	47.620	49.025	106.129	962.638
O ₃ ($\mu\text{g}/\text{m}^3$)	64.647	11.415	39.036	63.359	136.217
SO ₂ ($\mu\text{g}/\text{m}^3$)	5.307	5.176	0.004	3.871	67.591
Weather					
Total precipitation from atm. model physics ($\text{g}/\text{m}^2\text{s}$)	0.050	0.081	0.000	0.012	1.151
Surface air temperature (K)	297.5	7.3	252.0	298.8	311.7
Surface wind speed (m/s)	4.8	1.4	1.6	4.6	11.6
No. of observations					82,908
No. of obs. with mean monthly PM _{2.5} levels above WHO threshold of $5 \mu\text{g}/\text{m}^3$					81,967

Note: The table presents summary statistics of air pollution concentrations and weather variables, computed from monthly averages at the cell level, using data over 2010-2016. The number of observations reflects 7 years of monthly data for each of the 987 cells. All data have been aggregated from MERRA-2 (Surface Flux Diagnostics).

Figure 2: India map (*Panel A*) and histogram (*Panel B*) of PM_{2.5} concentrations.

Note: The figure uses cell-level averages over monthly observations during 2010 - 2016. Only territorial cells, where at least one primary sampling unit (PSU) is represented in the NFHS-4, are included. The vertical red line in the histogram (*Panel B*) represents the sample mean value.

Weather variables

Data on precipitation, temperature, and wind speed and direction comes from the MERRA-2 Surface Flux Diagnostics datasets, also available at $0.625^\circ \times 0.5^\circ$ spatial resolution and hourly frequency. Similarly to the procedure followed in the case of air pollution variables, we match the weather outcomes with the NFHS-4, by constructing averages representative of the lifetime horizon of each child, beginning nine months prior to the reported birth date until the date of interview when the anthropometric measures have been taken.

The key weather variable for our identification strategy is wind direction, and in particular its temporal and spatial variation. Wind direction frequency is fairly equally distributed on average in our sample, with only wind from the East being slightly less observed. Variation in wind direction is high, both across time (within variation) and across space (between variation). This high variability is important for our identification, as children from the same cell might be exposed differently to pollution, depending on when exactly they are conceived. Moreover, the high within-cell variability is also suggestive of the difficulty to predict changes in wind direction and take compensatory actions.¹³ Finally, when analysing how pollution levels vary with the prevailing winds, we find no consistent pattern.¹⁴

4 Identification Strategy

Our aim is to estimate the impact of air pollution on child growth variables. This relation can be modeled using the following equation:

$$y_{icg} = \beta_1 \text{PM2.5}_{ic} + \beta_2 \text{CO}_{ic} + \beta_3 \text{O3}_{ic} + X'_{ic} \gamma + \alpha_g + \epsilon_{icg} \quad (3)$$

where the outcome variable y_{icg} is the growth measure of child i , residing in cell c of the geographical region g . The coefficients of interest are the β s, the parameters indicating the link between the different pollutant concentrations and child's growth measurements.

¹³Table A.2 in the Appendix presents summary statistics regarding the overall, between, and within variation in wind direction over 90-degree wind angle bins using monthly data over 2010 - 2016 for all cells.

¹⁴Section A.4 in the Appendix provides details on the spatial variation in prevailing winds and on how this relates to air pollution concentrations.

The outcome variables are the height-for-age score, the indicator for being stunted, the indicator for being severely stunted, the weight-for-age score, the indicator for being wasted, and the indicator for being severely wasted. These values are provided in the NFHS-4 and reflect the measurements on the day the household was surveyed. Thus, they reflect a snapshot of a child's health status, shaped by all genetic, environmental, and behavioral factors that have impacted the child's development since conception and until the date of measurement.

The pollution concentrations are computed for each child separately and reflect averages of hourly measurements in the cell of residence c from child conception until the date of the survey. In this sense, computed pollution concentrations are equal for all children born on the same month and year in cell c . Hence, our results quantify the impact of pollution on the average child residing in the cell.

The granularity of our data allows us to control for a rich set of socio-economic and environmental factors that can impact child growth (X_{ic}). We control for gender and age to capture remaining variation in the outcome variables across these two dimensions.¹⁵ As the NFHS-4 collected data over two years (2015/2016), we also control for the year of interview. As a measure of a household's financial resources, we include the 5-digit wealth index provided by the NFHS-4, which is expected to account for the household's access and usage of various resources, including nutrition and health care. Similarly, to control for location-specific economic development across India, we include the nightlights index of the corresponding primary sampling unit (PSU). The index is expected to help account for employment and educational opportunities of the care givers, as well as access to medical care. We further include basic household characteristics, such as the number of household members and the religion, as indicators of household-level resource scarcity and cultural practices. Children's growth can also be impacted by environmental conditions that have shaped both the short- and the long-term setting in which the child has developed. We include averages of precipitation, temperature, and wind speed observed over the child's life. To capture unexplained spatial variation in growth outcomes and pollution, we group cells into geographical regions (see the discussion below for details on the procedure). In the

¹⁵All outcome variables reflect deviations from age- and gender-specific averages in the way they are computed, see Section 2. However, mean values of the outcome variables can still vary by age and gender, due to potential cultural and environmental practices. To account for this variation, we include gender and age group fixed effects. Moreover, we control for the child's age in months to account for potential within age group variations.

benchmark model, we include fixed effects for the geographical region of residence α_g .¹⁶ In our preferred specification, we cluster standard errors at the district level, but our results are robust to different levels of clustering, as shown in the robustness section.

The basic link between air pollution and child growth has been previously estimated in the literature, with the primary choice of cooking fuel in the household often used as a proxy for pollution exposure. Linear models or multinomial logistic regressions have been commonly used to estimate the drivers of the height-for-age score and the probability of stunting.

[Mishra and Retherford \(2007\)](#) use a multinomial logistic regression approach and find a significant and positive link between solid fuel use and the predominance of anaemia and stunting among Indian children aged 6–35 months, using NFHS data from 1998–1999. Relying on a similar methodological approach, [Machisa et al. \(2013\)](#) find, in contrast, no significant correlation between the use of solid fuels and stunting in children from Swaziland. [Kim et al. \(2017\)](#) assess the relative importance of 13 correlates of child stunting in South Asia employing data from nationally representative cross-sectional surveys. Using mutually adjusted logistic regression models, they find no significant effect of indoor pollution on stunting among children under the age of 23 months. [Fenske et al. \(2013\)](#) revisit the analysis of stunting drivers in India, relying on an additive quantile regression using the 2005–2006 NFHS data. They find that children from households using gas or electricity as primary source for cooking tend to be at a lower risk of stunting, especially in the lower 15th percentile of the height-for-age distribution. While the previous studies focused on indoor pollution, [Goyal and Canning \(2018\)](#) find that stunting is related to ambient air pollution in Bangladesh.

OLS estimates of the relationship between pollution and health are prone to bias; children's exposure to pollution is not randomly assigned but reflects a complex set of cultural and socio-economic factors that even the richest set of controls might fail to fully account for. In such specifications, endogeneity concerns arise due to omitted variable bias and measurement error; households are likely to make decisions that affect both child health and domestic purchases (such as solid fuel types) simultaneously. In general, poorer and less educated households tend to have a higher exposure to pollution and have both lower demand for and access to defensive mechanisms, such as face masks and air purifiers ([Greenstone et al., 2021](#)).

¹⁶For a discussion on including cell fixed effects, see Section [B.3](#) in the Appendix.

To correct for the omitted variable bias, we employ an IV approach where we rely on wind direction to capture quasi-random variation in air pollution, similarly to [Deryugina et al. \(2019\)](#) and [Bondy et al. \(2020\)](#). With this approach, we aim to pick up the variation in pollution induced by non-local sources and transported across space by wind. Intuitively, due to a generally unequal spatial distribution of emitting sources, air pollution in a particular place can be higher or lower at a point in time depending on the wind direction at that instant and the relative position of pollution sources.

The impact of wind direction on air pollution can be different across the country. For example, wind blowing from the West can clear the air for some locations but bring heavy pollutants for others, depending on where the pollution source is located. To account for this, we allow the impact of wind direction on local pollution to vary by geographical region. Our first-stage specification is given by:¹⁷

$$p_{icg} = \sum_g \rho_1^g \text{Share}_{ic}^S + \sum_g \rho_2^g \text{Share}_{ic}^N + \sum_g \rho_3^g \text{Share}_{ic}^E + X'_{ic} \theta + \alpha_g + \varepsilon_{icg} \quad (4)$$

where p_{ic} represents the estimated concentrations of $\text{PM}_{2.5}$, CO and O_3 . The excluded instruments, Share_{ic}^ω with $\omega \in \{S, N, E\}$, represent the respective shares of time the wind blew from the South, North, and East in the cell where child i resides, over his or her lifetime. Share_{ic}^W is the omitted category. This division corresponds to a classification of wind direction according to 90-degree wind angle bins.¹⁸

We divide the Indian territory into G geographical regions using a k -means clustering algorithm over the latitude and longitude coordinates of cell centroids, similar to [Deryugina et al. \(2019\)](#). [Fig. 3](#) illustrates the resulting territorial division into 30 groups, the total number of geographical regions we use in our benchmark specifications. [Section 6](#) shows the robustness of the results when varying the total number of regions. Intuitively, the higher the number of total geographical regions, the better are the instruments at predicting air pollution; in contrast, the lower the number of regions, the more likely it is that the captured variation in pollution is driven by non-local sources.

¹⁷All IV estimations are executed employing the `reghdfe` command in Stata ([Correia, 2016](#)). This estimates the first and second stages simultaneously and produces adjusted standard errors.

¹⁸Our main results remain unchanged when the shares of wind direction are constructed around eight cardinal points, i.e., using 45-degree wind angle bins instead of 90-degree; see our robustness section below.

The empirical specification is constructed to exploit the variation in pollution induced by wind direction in a similar manner across all cells belonging to a specific geographical region, i.e., the coefficients ρ^g are estimated based on variation in all cells from region g . Restricting the impact of wind direction on pollution to be the same for all cells in a region ensures that the pollution variation captured by Eq. 4 is more likely driven by non-local pollution sources that have a similar relative position to the entire region. In contrast, wind direction is less likely to affect locally produced pollution the same way for all cells in a region, due to differential positioning of sources with respect to the cells.

Figure 4 illustrates the variation in pollution we harness to estimate the causal impact on child growth outcomes. We take the example of Region 30, located in the center of India, and estimate the relation between annual average $PM_{2.5}$ concentrations and the annual share of wind blown from each of the four cardinal directions, with the share of wind blowing from the West as the base category. The regression controls for year and region dummies. Here, wind from the South significantly reduces $PM_{2.5}$ on average, compared to wind blowing from the West; wind from the North or East has the same average impact as from the West. This seems intuitive, as Region 30 is to the East of the Western Ghats mountain range, so clean air from the sea hardly reaches it. The region is surrounded by coal power plants to the North and East; only wind from the South manages to clear the air. In Appendix B, we include results from the same exercise for all 30 regions, with both four and eight cardinal directions to illustrate the variation in pollution. In all cases, the share of wind from the different directions is a strong predictor of local $PM_{2.5}$.

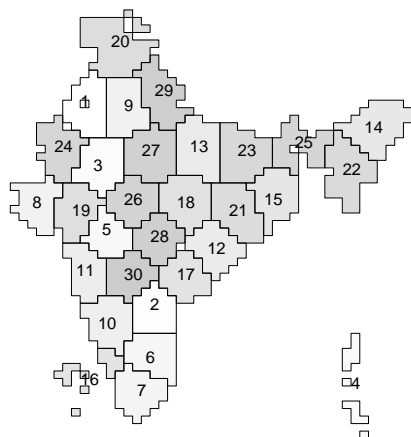


Figure 3: Territorial division into 30 regions.

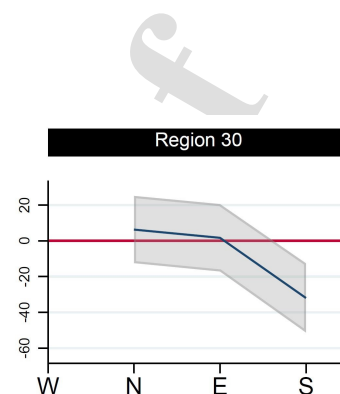


Figure 4: Impact of wind direction on average $PM_{2.5}$ in Region 30.

Note: Fig. 3 illustrates the categorization of territorial cells into 30 geographical regions based on a k -means algorithm over the latitude and longitude of cell centroids. Fig. 4 shows regression estimates of annual average $PM_{2.5}$ concentrations in Region 30 on the share of wind blown from different cardinal directions, while accounting for year and region indicators. West is the base category. Standard errors are clustered at the district level (for the specification, see Eq. B.2 in Appendix B).

The illustrative case above suggests that there is no bias due to weak instruments. The results of the first-stage estimation in our benchmark model (Eq. 4) using the entire dataset are presented in Section B.2 in the Appendix. Furthermore, the first-stage Anderson-Rubin and the Kleibergen-Paap F -statistics in the table of results in Section 5.1 also reinforce that there is no weak instrument bias.

5 Results

This section presents the main results of estimating the impact of air pollution on child growth indicators (Eq. 3). Besides the pollution concentration variables, all specifications include the following controls: the household wealth index, the nightlights index, the number of household members, whether or not the child lives in a Hindu household, child's age in months, age group, child's gender, the year of interview, and climate variables (average temperature, precipitation, and wind speed over the child's lifetime). All models include geographical zone fixed effects.¹⁹ Standard errors are clustered at the district level.

¹⁹In the benchmark models, we use a total of 100 zones for the spatial fixed effects. The robustness section relaxes this assumption.

5.1 Air Pollution and Child Growth

Table 3 reports OLS and IV estimates of the impact of air pollution on child growth indicators. As reported in Panel 1, the OLS estimates indicate a detrimental effect of pollution on children's anthropometric measurements, but the estimates are generally not statistically significant, with the exception of carbon monoxide (CO) which appears to reduce the weight-for-age score and increase the probability of being wasted in children.

Panel B reports the IV estimates of the causal impact of air pollution concentrations on anthropometric measurements. These are sizably larger than the estimates in Panel A, suggesting a significant downward bias in the OLS.²⁰

PM_{2.5} impacts

The IV estimates imply a highly significant impact of PM_{2.5} on children's height-for-age score, as well as on stunting and severe stunting rates. The results suggest that each 1 µg/m³ increase in PM_{2.5} over the lifetime exposure of a child, reduces his/her HAZ score by 0.011 and increases the probability of being stunted and severely stunted by 0.29 and 0.14 percentage points, respectively. We do not find evidence that PM_{2.5} concentrations affect the weight-for-age score or the probability of being wasted and severely wasted.

As variations in PM_{2.5} concentrations both across time and space are considerably high, we report our estimates in relation to the observed variation. Table 4 converts the significant IV coefficients into shares of standard deviation explained, in a similar fashion to Arcand et al. (2015). An increase of one standard deviation in PM_{2.5} concentrations, i.e., by 17.012 µg/m³ in our sample, corresponds to a reduction in the average HAZ score by about 0.19 (i.e., 0.01104 × 17.012). This would explain 11.8% of

²⁰The explanation beyond the downward bias in the OLS estimates can be threefold. First, our estimates of pollution concentration can suffer from measurement error, as we are using information at the available MERRA-2 resolution, which can imprecisely reflect personal exposure especially for periods spent indoors. This measurement error in pollution biases the OLS estimate of the treatment effect toward zero. IV estimates, in contrast, should be unaffected by the measurement error and are hence larger than the OLS estimates. Second, while OLS estimates reflect the average treatment effect (ATE) over the entire sample, the IV estimates can be interpreted as local average treatment effects (LATE) and can be larger than the OLS due to heterogeneity in the studied population. We explore this heterogeneity in Section 5.2. Third, the downward bias in the OLS can suggest the presence of omitted variables that are correlated with pollution and health. For example, pollution concentrations can be higher in more industrialized areas, where access to health care is easier, offsetting thus some of the negative impacts of pollution on health (Sanders, 2012).

the sample standard deviation of HAZ. At a one standard deviation increase in PM_{2.5} concentrations, stunting and severe stunting rates increase by almost 5 and 2.4 percentage points, respectively. This corresponds to 10.9% and 7.1% of their respective standard deviations.

Table 3: OLS and IV estimates of the effect of air pollution on child growth variables.

	HAZ (1)	Stunted (2)	Sev. stunted (3)	WAZ (4)	Wasted (5)	Sev. wasted (6)
Panel A: OLS estimates						
PM _{2.5} (µg/m ³)	-0.00083 (0.00161)	0.00015 (0.00042)	-0.00007 (0.00034)	0.00130 (0.00140)	-0.00027 (0.00047)	-0.00029 (0.00027)
CO (µg/m ³)	-0.00008 (0.00017)	0.00006 (0.00004)	-0.00002 (0.00005)	-0.00030*** (0.00011)	0.00011** (0.00004)	0.00001 (0.00003)
O ₃ (µg/m ³)	-0.00783 (0.00520)	0.00239* (0.00136)	0.00111 (0.00089)	-0.00370 (0.00448)	0.00266* (0.00152)	0.00101 (0.00081)
Mean of outcome variable	-1.323	0.328	0.128	-1.628	0.399	0.103
Adjusted R ²	0.128	0.092	0.052	0.168	0.112	0.045
Panel B: IV estimates						
PM _{2.5} (µg/m ³)	-0.01104*** (0.00315)	0.00288*** (0.00079)	0.00143** (0.00056)	0.00113 (0.00253)	-0.00093 (0.00084)	-0.00025 (0.00046)
CO (µg/m ³)	0.00060 (0.00055)	-0.00011 (0.00016)	-0.00024* (0.00014)	-0.00067* (0.00035)	0.00033** (0.00013)	0.00001 (0.00007)
O ₃ (µg/m ³)	0.01689 (0.01374)	-0.00592 (0.00363)	-0.00228 (0.00218)	-0.01670 (0.01073)	0.00747** (0.00352)	0.00302* (0.00178)
Mean of outcome variable	-1.323	0.328	0.128	-1.628	0.399	0.103
Anderson-Rubin <i>F</i> -stat (joint sig.)	35.325	23.193	17.158	47.052	40.605	36.633
Anderson-Rubin <i>F</i> -stat <i>p</i> -value	0.000	0.000	0.000	0.000	0.000	0.000
Kleibergen-Paap <i>F</i> -stat	16.6	16.6	16.6	16.6	16.6	16.6

Note: The table reports OLS (Panel A) and IV (Panel B) estimates of the impact of air pollution concentrations on child growth outcomes (Eq. 3 in the main text). Each column represents the effect of pollution on a different anthropometric measure. The data is at the individual child level. All regressions include the following controls: the household wealth index, the nightlights index, the number of household members, whether or not the child lives in a Hindu household, child's age in months, child's age group, child's gender, the year of interview, and climate variables (average temperature, precipitation, and wind speed over the child's lifetime). For ease of presentation, these coefficients are not reported here; instead, full model estimations are presented in Appendix C.1. All models include geographical zone fixed effects, with cells being grouped into 100 regions. In the first-stage of the IV models, the impact of wind direction on air pollution concentrations is allowed to vary across 30 regions (i.e., $G = 30$ in Eq. 4). Standard errors, clustered by district, are reported in parentheses. Statistical significance at the 1%, 5%, and 10% levels is denoted by ***, **, and *, respectively.

Table 4: Quantification of PM_{2.5} impacts on child growth outcomes as share of standard deviation explained.

	HAZ (1)	Stunting rate (2)	Sev. stunting rate (3)
PM _{2.5}	11.8%	10.9%	7.1%

Note: The table converts the estimated significant PM_{2.5} IV coefficients from Tables 3 (Panel B) into shares of standard deviation (sd) of the dependent variables explained. Conversions are done according to the following formula: share of $sd = [100 \times \hat{\beta} \times sd(p)] / sd(y)$. The sample standard deviations of the growth indicators, $sd(y)$, take the following values: 1.587 for HAZ, 0.469 for the stunting rate, and 0.334 for severe stunting. The sample standard deviation for PM_{2.5}, $sd(p)$, is 17.012.

Next, we report impact sizes in relation to the WHO guidelines for pollution exposure. The target threshold for mean annual PM_{2.5} concentrations is 5 $\mu\text{g}/\text{m}^3$, around eight times lower than the observed mean national concentrations in India over 2010-2016 (Table 2). Back-of-the-envelope calculations suggest that, at a reduction in PM_{2.5} concentrations from 41.146 to 5 $\mu\text{g}/\text{m}^3$, the stunting and severe stunting rates would decrease by 10.41 and 5.17 percentage points respectively, *ceteris paribus*. In our sample, this would correspond to a decrease in national stunting rates from 31.1% to 20.7%, or about 14.26 million less stunted children.²¹

Other pollutants

Although PM_{2.5} is the standard air quality indicator for health risks, other pollutants are responsible for negative health impacts, most importantly carbon monoxide (CO) and ozone (O₃). Similarly to PM_{2.5}, these pollutants are transported by wind across territories and need to be accounted for in the empirical estimation. Simultaneously including all pollutants in our models reduces the risk of omitting important health risk factors that are co-transported by wind, and of over-estimating the impact of PM_{2.5} on health impacts. Other two critical air pollutants that are known to have negative health impacts and are usually accounted for in the computation of daily air quality indices are sulfur dioxide (SO₂) and nitrogen dioxide (NO₂). However, both pollutants convert to PM_{2.5} within a few days. As such, our specification, which employs long-term pollution aggregates, does not allow us to assess their independent impacts from those of PM_{2.5}, similarly to Deryugina et al. (2019).

²¹ According to the Indian Ministry of Statistics, India had a population of around 137 million children aged 0-5 years in 2018 (<http://www.mospi.gov.in>).

In our results, both OLS and IV estimates suggest limited independent impacts of CO and ozone on children's height-related outcomes. However, in both Panels 1 and 2, carbon monoxide appears to significantly impact weight-related measures, such as the weight-for-age score and wasting rates. The IV estimates suggest that an increase in CO by $1 \mu\text{g}/\text{m}^3$ reduces WAZ on average by 0.0007 and the probability of being stunted by 0.03 percentage points. This corresponds to 2.84% of the sample standard deviation of WAZ and 2.95% of the sample standard deviation of the wasting rate, at an increase by one standard deviation in CO.

Each $1 \mu\text{g}/\text{m}^3$ increase in ozone concentrations leads to an increase by 0.75 and 0.3 percentage points in wasting and severely wasting rates, respectively. An increase by one standard deviation in O_3 explains 9.87% and 6.37% of the sample standard deviation of the wasting and severe wasting rate, respectively.

All results are robust to including the pollutants individually in the models, as reported in Appendix C.2, suggesting that their independent impacts on health are not masked by correlations with $\text{PM}_{2.5}$ as main pollutant.

Overall, we find that fine particulate matter has the strongest associated health risk on height-related outcomes, while ozone and CO are responsible for negative impacts on weight-related growth measures.²² Otherwise stated, it appears that $\text{PM}_{2.5}$ is responsible for long-term growth impairments, while CO and O_3 are responsible mostly for short-term deficiencies. This difference could be related to the different health risks the three pollutants raise and the pollutant-specific sensitive groups (see Fig. A.7 in the Appendix). While $\text{PM}_{2.5}$ brings health risks to all children, CO is most harmful for individuals with heart disease, and ozone is especially dangerous, among others, for people with diets limited to certain nutrients. We revisit these findings in Section 5.3.

Comparison to previous findings

Our results are in line with previous studies that investigate the link between air pollution and anthropometric measures. In India, Mishra and Retherford (2007) find that severe stunting is more prevalent among children from households using biofuels for cooking and heating. In a similar vein, Fenske et al. (2013) show that children from

²²The impact of CO is weakly statistically significant and negative on severe stunting, reflecting the fact that CO is often negatively correlated with other pollutants (Deryugina et al., 2019; Currie and Neidell, 2005).

households using kerosene for cooking are more likely to be affected by severe stunting than households using other fuel types.²³ The methodological differences between the present paper and the previous two studies are that they: (i) rely on the main fuel type reported by the household as a proxy for (indoor) air pollution, and (ii) do not account for endogeneity issues linked to air pollution exposure. Using a panel structure and ambient air pollution data from satellite observations in Bangladesh, [Goyal and Canning \(2018\)](#) find that an increased exposure to pollution during the prenatal period is associated with a higher risk of stunting during childhood. None of these studies considers other air pollutants, such as CO and ozone, in their analysis. Our results are well-aligned with the previous literature in terms of direction of the general pollution-stunting relation, and try to improve on the magnitude of estimated effect sizes by accounting for endogeneity. However, due to the differences in empirical specifications, a direct comparison of the magnitude of estimates is difficult.

5.2 Age, gender, and birth order

The models reported in Table 3 are estimated using the full set of controls, albeit not reported there. The full estimation results are included in Section C.1 of the Appendix. All coefficients have the expected sign. The household wealth index is one of the variables with highest explanatory power of child growth indicators, being highly significant across all models and positively related to the height- and weight-for-age z-scores, and negatively related to stunting and wasting rates, in both moderate and severe forms. The nightlights index shows similar patterns, although not statistically significant in HAZ and WAZ models. Children residing in households with more family members tend to have poorer growth indicators. Furthermore, children from Hindu households also fare worse than the rest.

A child's age in months appears to play an important role in explaining growth deficiencies, even after controlling for the age group. To gain a better understanding of how the impact of pollution on health varies with age, we estimate the benchmark model with the full set of controls for five age groups separately. The coefficients capturing the impacts of PM_{2.5} on height-related growth outcomes are captured in Fig. 5 below. The magnitudes appear to increase monotonically with age, in line with the theory that each new year of exposure to pollution contributes to a cumulative worsening of the health status. The estimated impacts of PM_{2.5} seem to gain statistical significance

²³The combustion of kerosene results in high indoor CO and SO₂ concentrations.

from age one onward.

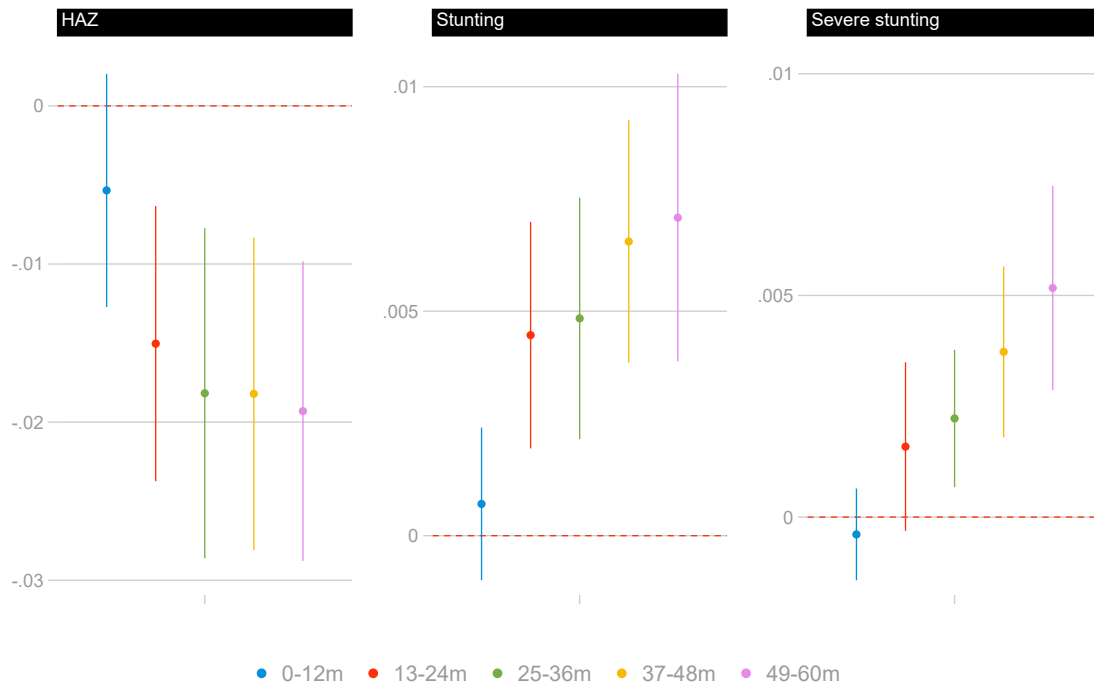


Figure 5: IV estimates of the impact of $PM_{2.5}$ on child growth outcomes, by age group.

Note: The figure presents IV estimates of the impact of $PM_{2.5}$ on HAZ, stunting and severe stunting rates (Eq. 3), separately by age group. The usual controls are included and the standard errors are clustered by district.

In the pooled models across all five age groups, male children seem to fare worse across all growth indicators (see Table C.1 in the Appendix). Gender differences in the growth outcomes of children might be explained by both genetic and cultural factors.

The impacts of $PM_{2.5}$ by gender and birth order are captured in Fig. 6, showing that girls' height-related growth indicators are relatively more sensitive than boys'. These results are unfortunately not surprising, as health discrimination against women in India is well-documented (Vulimiri et al., 1996). Worryingly, as the Indian economy has been growing, the average height in India is on the rise, but the observed rate of increase is three times higher for Indian men than women, likely due to differentiated access to improvements in nutrition and health care (Deaton, 2008). We revisit this point in Section 7.

Furthermore, Fig. 6 shows the differential impact of $PM_{2.5}$ on firstborns and juniors (children that have older siblings). We find stronger negative impacts on children higher in the birth order than on firstborns, for both genders. The results are suggestive of cultural differences in the upbringing of children with respect to the adoption of pollution defensive mechanisms, with preferential treatment given to firstborn sons. A general favoritism toward eldest sons in India has been previously documented in [Jayachandran and Pande \(2017\)](#). Hence, our results point to one channel through which the growth indicators of juniors are poorer.

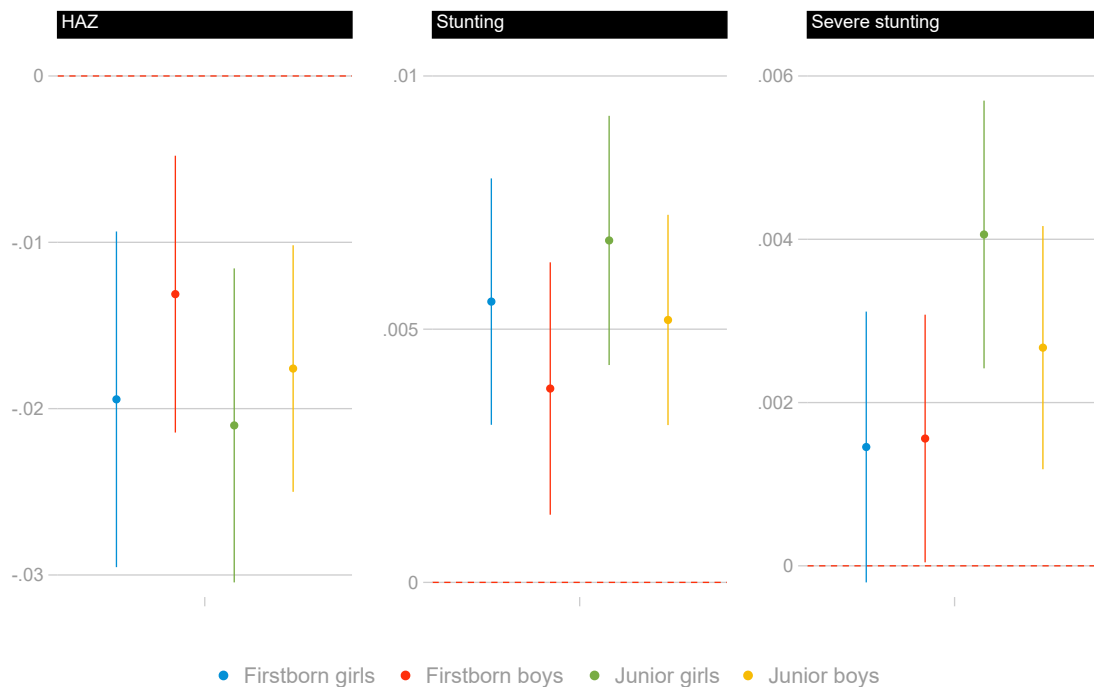


Figure 6: IV estimates of the impact of $PM_{2.5}$ on child growth outcomes, by gender and birth order.

Note: The figure presents IV estimates of the impact of $PM_{2.5}$ on HAZ, stunting and severe stunting rates (Eq. 3), separately by gender and birth order (firstborn versus juniors). The usual controls are included and the standard errors are clustered by district.

5.3 Rural versus urban

The pollution effects we measure are net of any behavioral responses to health risks and are averaged across India, despite stark variation in pollutant concentrations lev-

els across the country (see Fig. A.2). While $PM_{2.5}$ is the main critical pollutant with high concentrations all over the country, ground-level ozone is typically found at higher concentrations in urban areas, usually associated with vehicle emissions. Carbon monoxide levels can be high in both rural and urban areas, with the burning of unclean fuels for cooking and heating as main sources. Children can thus be exposed to different pollutants, depending on their place of residence.

Table 5 shows that $PM_{2.5}$ has indeed comparable results on height-related growth outcomes in both rural and urban areas. However, the impacts on weight-related outcomes seem to be driven by CO in rural areas, where the burning of solid fuels indoors is still largely present, and O_3 in urban areas, where ozone is produced when pollutants emitted by cars, power plants, industrial boilers, refineries, chemical plants, and other sources chemically react in the presence of sunlight (Lal et al., 2008; Sharma et al., 2016; Sujith et al., 2017).

Table 5: Rural versus urban areas: IV estimates of the effect of air pollution on child growth variables.

	HAZ (1)	Stunted (2)	Sev. stunted (3)	WAZ (4)	Wasted (5)	Sev. wasted (6)
Panel A: Rural						
PM _{2.5} (µg/m ³)	-0.00971*** (0.00354)	0.00276*** (0.00092)	0.00164** (0.00064)	0.00264 (0.00264)	-0.00110 (0.00090)	-0.00027 (0.00051)
CO (µg/m ³)	0.00012 (0.00052)	-0.00003 (0.00016)	-0.00027* (0.00014)	-0.00123*** (0.00046)	0.00046*** (0.00017)	0.00004 (0.00008)
O ₃ (µg/m ³)	0.02357 (0.01540)	-0.00760* (0.00420)	-0.00438* (0.00265)	-0.00974 (0.01161)	0.00515 (0.00405)	0.00181 (0.00204)
Observations	169,842	169,842	169,842	169,842	169,842	169,842
Mean of outcome variable	-1.397	0.348	0.139	-1.685	0.419	0.111
Anderson-Rubin <i>F</i> -stat <i>p</i> -value	0.000	0.000	0.000	0.000	0.000	0.000
Kleibergen-Paap <i>F</i> -stat	20.6	20.6	20.6	20.6	20.6	20.6
Panel B: Urban						
PM _{2.5} (µg/m ³)	-0.01251*** (0.00439)	0.00227** (0.00103)	0.00101 (0.00069)	-0.00241 (0.00375)	-0.00117 (0.00118)	-0.00004 (0.00066)
CO (µg/m ³)	0.00112* (0.00065)	-0.00014 (0.00017)	-0.00013 (0.00014)	0.00024 (0.00056)	0.00025 (0.00017)	0.00008 (0.00008)
O ₃ (µg/m ³)	-0.00766 (0.01601)	0.00156 (0.00428)	0.00444 (0.00270)	-0.03332*** (0.01255)	0.01326*** (0.00419)	0.00822*** (0.00239)
Observations	53,207	53,207	53,207	53,207	53,207	53,207
Mean of outcome variable	-1.087	0.262	0.092	-1.444	0.334	0.077
Anderson-Rubin <i>F</i> -stat <i>p</i> -value	0.000	0.000	0.000	0.000	0.000	0.000
Kleibergen-Paap <i>F</i> -stat	8.0	8.0	8.0	8.0	8.0	8.0

Note: The table reports IV estimates of the impacts of air pollutants on child growth variables (Eq. 3) in urban and rural areas, separately. Each column represents the effect of pollution on a different outcome variable. The data is at the individual child level. The usual controls have been included. Regressions include geographic group fixed effects, with cells being grouped into 100 regions. In the first stage of the IV models, the impact of wind direction on air pollution concentrations is allowed to vary across 30 regions (i.e., $G = 30$ in Eq. 4). Standard errors, clustered by district, are reported in parentheses. Statistical significance at the 1%, 5%, and 10% levels are denoted by ***, **, and *, respectively.

6 Robustness Tests

We test the robustness of our results across several dimensions and show that our main findings are consistent with alterations in the benchmark specification. We discuss the results here, while the supporting tables and figures are relegated to the Appendix.

6.1 The air quality index

Our benchmark models account for the three most important criteria pollutants that pose health risks in India. The estimations so far have included *level* concentrations of the pollutants and served to identify the health impacts of one additional $\mu\text{g}/\text{m}^3$. However, health impacts of pollutant concentrations might not be linear, but different effects could occur at different ranges of concentrations. Moreover, although all pollutants included are measured in terms of $\mu\text{g}/\text{m}^3$, their relative impact on health outcomes might not be directly comparable. We convert pollutant concentrations into the corresponding air quality index (AQI)²⁴ and estimate the IV models with pollutant-specific and total AQI, separately. The results are presented in Section C.6 of the Appendix. The estimated coefficients are consistent in both sign and significance to our benchmark results. When comparing coefficient sizes across models using either concentrations or air quality indices, the magnitude is fairly close between $\text{PM}_{2.5}$ concentrations and AQI $\text{PM}_{2.5}$, suggesting that the impact of one additional $\mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$ is comparable to one unit increase in AQI $\text{PM}_{2.5}$.

6.2 Using sea wind for an alternative specification

To check the robustness of our results, we design an alternative identification strategy, where we only rely on the sub-sample of children living in the vicinity of the Indian coastline. We use the share of time the wind blew from the direction of the sea as an instrument for local pollution concentrations. Full details on the specification and the results are included in Section D of the Appendix. Despite lower statistical significance, the magnitude of the effects is consistent with our benchmark findings.

6.3 Clustering of standard errors

In our main specification, we cluster standard errors at the district-level, to correct for potential heteroskedasticity. Section E.2 in the Appendix demonstrates how our results remain unchanged when we instead cluster at the cell or geographical region level.

Ideally, we should correct the standard errors for spatial correlation as in [Conley \(1999\)](#);

²⁴An AQI aims to offer a comprehensive measure of ground-level air pollution, taking into account the health risks imposed by various criteria pollutants. Appendix C.6 presents summary statistics and key descriptive figures of air pollution as measured by air quality indices. We follow the US EPA guidelines for computing the individual pollutant-level and total air quality index ([US EPA, 2018](#)).

however, the cross-sectional data structure at the individual level, with multiple children located in the same place (sharing the same latitude and longitude coordinates) is not well-suited for spatial error correction models, at least not under current advances that we are aware of. Nevertheless, in order to draw a rough comparison between models with spatially corrected errors versus models with errors clustered at the district level, we modify the data structure and run the analysis in a cross-section at the cell level, where variables represent cell means constructed from individual-level observations. The description of the entire exercise and the results are included in Section E.5 of the Appendix. Overall, we find straight consistencies among the different error correction models, suggestive of robustness of our main specifications. However, this analysis needs to be interpreted with caution, as we rely on a different data structure when running the tests.

6.4 Pollution from non-local sources and instrument definition

In order to be able to interpret IV estimates as a LATE, the monotonicity assumption regarding the consistent impact of wind direction on air pollution needs to hold (Angrist and Imbens, 1995, see also the discussion in Deryugina et al. (2019)). This means that an increase in the share of wind blowing from a particular direction should affect air pollution concentrations similarly for all cells in a geographic region. We investigate the validity of this assumption by estimating alternative specifications where we vary (i) the number of cardinal points for defining wind direction, and (ii) the size of the geographical regions (the number of cells in a region).

First, Table E.1 in the Appendix captures the IV estimates when wind direction is allowed to vary across 8 cardinal points, i.e., when using 45-degree wind angle bins instead of 90-degree as in the benchmark specification. The sign and magnitude of the results remain consistent, indicating robustness.

Second, we vary the size of the geographical region and thus the level of spatial aggregation at which the impact of the excluded instruments on air pollution is allowed to vary. Fig. E.1 captures the robustness of the impact of $PM_{2.5}$ on height-related outcome variables, when dividing the Indian territory into 10, 20, 30, 40, or 50 regions, alternatively. Beyond providing evidence that the monotonicity assumption is not violated in our analysis, this test also brings evidence that the variation in pollution we are capturing is more likely to come from non-local sources than local ones. In contrast, if the variation in pollution we are capturing came from local sources, our results would vary

with the number of cells in a region.

Additionally, we run tests to see whether the results remain stable when varying the geographical region dummies. Section E.3 in the Appendix allows for different levels of aggregation when defining the regional dummies and shows that results are very similar to those in our benchmark specification.

Based on these different tests, we find no evidence so far of the violation of the monotonicity assumption. Hence, we interpret this as supportive evidence that our results can be interpreted as LATE.

7 Discussion: Height and labor market outcomes

There exists evidence that the height of an individual as a child predicts not only his or her height as an adult, but it is also an important predictor of health and social status and earnings in adulthood (see, e.g., [Persico et al., 2004](#); [Case and Paxson, 2008b](#); [Lundborg et al., 2009](#)). Several possible underlying mechanisms have been highlighted: (i) physical capacity has a significant impact on adult health, which is correspondingly rewarded on the labor market ([Steckel, 1995](#); [Strauss and Thomas, 1998](#); [Metter et al., 2002](#); [Thomas et al., 2006](#); [Lundborg et al., 2009](#)); (ii) height is strongly predictive of cognitive capacity, where the link becomes particularly important in early childhood and continues throughout the adult life ([Richards et al., 2002](#); [Case and Paxson, 2008a,b](#)); and (iii) height also affects non-cognitive skills, such as confidence and self-image, which can impact perseverance and social skills ([Persico et al., 2004](#); [Heckman et al., 2006](#)). Shorter height may increase the probability of being discriminated against, particularly in social dealings and negotiations ([Frieze et al., 1990](#)).

The strength of the height - earnings relation is particularly apparent at the tails of the income distribution ([Lundborg et al., 2009](#)) and the height premium is often higher in developing countries ([Schultz, 2002](#); [Sohn, 2015](#)).

We compute the reduced earnings related to shorter height associated with air pollution. Table 6 summarizes some of the main contributions in the literature to the calculation of the height premium in earnings, based on differences in adult height. We anchor our calculations to the estimates provided by [Sohn \(2015\)](#), where the height premium in earnings per year is estimated at 7.5% for men and 13% for women per 10 centimeters of adult height, based on data from Indonesia. As a first step, we specify the

relation between child height and adult height following [Cole and Wright \(2011\)](#):

$$\text{Height}_a = sd_a \times \text{HAZ}_a + \text{Median}_a \quad (5)$$

where Height_a stands for adult height, Median_a and sd_a are the median and standard deviation in adult height of the WHO reference population, and HAZ_a is the height-for-age z-score with respect to the adult WHO reference population. The link to child characteristics comes from expressing the adult's height-for-age z-score (HAZ_a) as a function of child's height-for-age z-score (HAZ_c), with $\text{HAZ}_a = \rho \times \text{HAZ}_c$. ρ is the correlation coefficient between child's current height and their adult height, varying with age and gender (as tabled in [Molinari et al. \(1995\)](#), see Appendix F).²⁵ Then, Eq. 5 becomes:

$$\text{Height}_a = sd_a \times \rho \times \text{HAZ}_c + \text{Median}_a \quad (6)$$

We measure the impact of an increase in the mass concentrations of $\text{PM}_{2.5}$ on adult height as:

$$\begin{aligned} \text{Height}'_a - \text{Height}_a &= (sd_a \times \rho \times \text{HAZ}'_c + \text{Median}_a) - (sd_a \times \rho \times \text{HAZ}_c + \text{Median}_a) \\ &= sd_a \times \rho \times \hat{\beta}_1 \end{aligned} \quad (7)$$

where Height'_a is the height of an adult that has been exposed to pollution in childhood and their corresponding HAZ score in childhood is HAZ'_c . $\hat{\beta}_1$ is the change in the HAZ score of children at an increase in the mass concentration of $\text{PM}_{2.5}$.

Figure 7 (Panel A) plots the estimated reduction in adult height at an increase by one standard deviation in $\text{PM}_{2.5}$, based on the relation described in Eq. 7, holding all other things equal. These estimates are based on age- and gender-varying correlations between height in childhood and height in adulthood (data from [Molinari et al. \(1995\)](#)), the standard deviation of height for nineteen-year-old men and women in the WHO reference population,²⁶ and the estimated impact of one standard deviation increase in $\text{PM}_{2.5}$, by gender and age group (based on Fig. C.4). Despite higher responses in women's HAZ scores to air pollution during childhood ($\hat{\beta}_{1,\text{women}} < \hat{\beta}_{1,\text{men}}$), their adult height is relatively less affected than men's, following from lower estimated correlations between childhood height and adult height in women ($\rho_{\text{women}} < \rho_{\text{men}}$), as well

²⁵The correlation between child height and adult height increases with child's age [Cole and Wright \(2011\)](#).

²⁶It is generally the case that individuals reach their adult height in the early twenties ([Deaton, 2008](#)).

as lower standard deviations in adult height ($sd_{a,women} < sd_{a,men}$).

Next, we translate the “lost” height into labor market outcomes, relying on the estimates of [Sohn \(2015\)](#) for Indonesia, the most similar country to India in which the height premium has been estimated. Considering a 7.5% height premium for men and 13% for women per 10cm, the estimated missed earnings are plotted in [Fig. 7](#) (Panel B) for Indian boys and girls five-year old or younger. Results are expected to be more robust after age two, when the correlation coefficient between child and adult height becomes more precise ([Cole and Wright, 2011](#)). Moreover, in our IV estimations, the impact of $PM_{2.5}$ on HAZ is not significant for boys below one year old (see [Fig. C.4](#)). Thus, we prefer to give more weight to the estimated lost earnings for children in age groups 2–5.

These rough estimates point to a loss in earnings in the range of 1.4–1.8% for men and 1.6–2% for women due to an increase in $PM_{2.5}$ by one standard deviation during their childhood, all other things equal. Our results are thus suggestive of stronger long-term negative consequences of $PM_{2.5}$ exposure on girls than boys, especially with respect to missed earnings in adulthood. The size of the estimated coefficients rely on the limiting assumption that the height premium computed by [Sohn \(2015\)](#) using Indonesian data holds in India as well. From the scarce existing evidence for India, the height premium appears to be comparable, if not higher in India, at least for labor-intensive professions such as coal mining ([Dinda et al., 2006](#)).

Moreover, according to the Global Gender Gap Report 2020, India is and has been performing more poorly than Indonesia in terms of women’s access to economic participation and opportunity, educational attainment, health and survival, and political engagement, relative to men’s ([World Economic Forum, 2020](#)). We then expect that the gender-differentiated height premium in earnings is higher in India than Indonesia, suggesting that our results regarding the $PM_{2.5}$ gender discrimination might be underestimating the true impacts.

Table 6: Height premium in earnings in developing and developed countries.

Study	Country	Sample	Height Premium
1. Developing countries			
Sohn (2015)	Indonesia	Adult workers	7.5% (men) and 13% (women) per 10 cm ^{a,c}
Vogl (2014)	Mexico	Men aged 25-65	12% per 10 cm ^b
Thomas and Strauss (1997)	Brazil	Men aged 25-50	2.4% per 1% of height ^c
Dinda et al. (2006)	India	Coal miners in the east of India	9–17% above average - below average, 6–13% above average - average; average = 155.0–164.99 cm ^a
LaFave and Thomas (2017)	Indonesia	HH members aged 15 and older	2.3% per 1% increase in height ^{c,b}
2. Developed countries			
Persico et al. (2004)	US and UK	Cohort data, aged 7-33/16-27 (UK/US)	2.1% (UK) and 2.6% (US) per 1 inch at age 16 ^b
Case and Paxson (2008b)	US and UK	Cohort data, aged 7-33 (UK)	1.8-2.6% for men and 0.6-2.4% per 1 inch for women ^c
Lundborg et al. (2009)	Sweden	Males aged 28-38	6% per 10 cm
Rashad (2008)	US	Adults aged 21-45	5.4% per 10 cm
Harper (2000)	UK	Adult men	5.9% between 80-89th and 20-79th height percentile ^c

Note: The table presents a list of existing studies that investigate the height premium in earnings using large-scale longitudinal data for a set of developing and developed countries. Studies include different sets of covariates in their specifications: ^a workplace characteristics associated with risk and health measures at the workplace; ^b childhood characteristics as covariates; ^c body measurements, cognitive skills, and educational attainment.

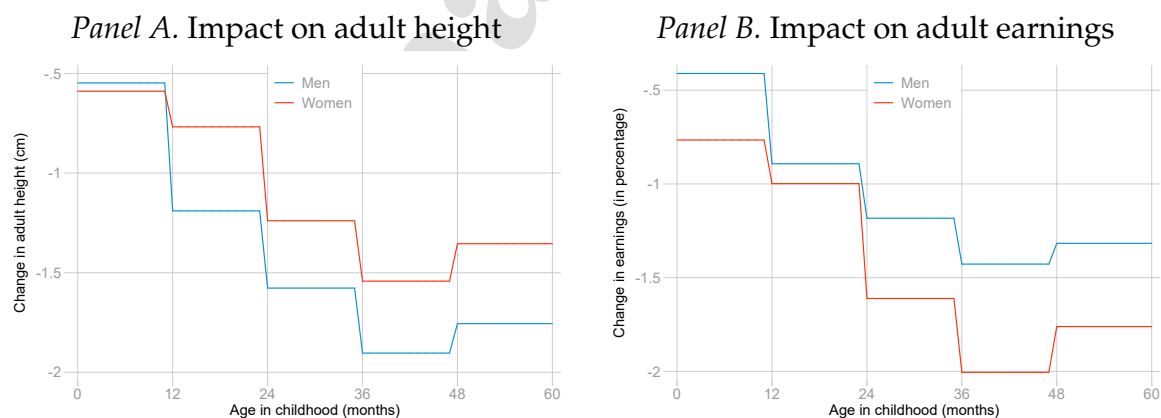


Figure 7: Sensitivity of adult height and of earnings in adulthood to exposure to PM_{2.5} concentrations during childhood, by age and gender.

Note: The figure presents changes in adult height (Panel A) and in earnings in adulthood (Panel B) at an increase by one standard deviation of PM_{2.5}. The impact is computed by child's age and gender.

8 Conclusions

The detrimental effects of air pollution on children's health are becoming increasingly salient, particularly in India, where the bad air quality is a constant problem, exacerbated by the high prevalence of solid fuel combustion. We rely on a unique dataset formed by merging the latest available National Family Health Survey (NFHS-4) in India with satellite information on ambient air pollution, wind direction and speed, and related weather variables. We use the quasi-random variation in pollution induced by wind direction. Correcting for endogeneity, this paper is, to the best of our knowledge, the first to move away from correlation analysis and take steps toward demonstrating the causal link between air pollution and chronic restrictions in the physical development of children.

We find that higher exposure to air pollution causes significant limitations to child growth. An increase by one standard deviation in $PM_{2.5}$ induces higher rates of stunting and severe stunting by 5 and 2.4 percentage points, respectively. Ozone and CO appear to impact weight-related growth outcomes. The WHO recommends an average annual $PM_{2.5}$ level of $5 \mu\text{g}/\text{m}^3$, which is eight times lower than the annual average in India for the period 2010-2016. Back-of-the-envelope calculations indicate that reducing pollution levels in India to the WHO standard, assuming all other things equal, could decrease stunting rates by about 10.4 percentage points, impacting about 14.3 million children. Our estimates are consistent across a series of robustness tests.

We use previous findings to compute a rough estimate of the additional impact of pollution on the height premium in earnings. The results illustrate once more how early-life investments in human health capital are essential for personal and public economic development in the long-term.

One limitation of the current analysis is linked to the data structure used. Whereas the NFHS is already in its fourth round, only the last wave offers georeferenced information of primary sampling units to which we could link satellite information on pollution, wind, and weather-related variables. Increasing the availability of geocoded surveys would not only allow to account for individual time-invariant characteristics but also to better estimate the long-term consequences of child growth impairments.

We use a gridded data structure to explore exogenous variation in ambient air pollution. Modest estimation errors could be expected due to imprecise pollution concentration measurements along India's geopolitical borders. Moreover, this approach

relies on ambient air pollution levels, as information on direct exposure to pollution is unavailable. Our results might underestimate the impact of pollution on growth outcomes. Nevertheless, the current analysis is expected to be informative for designing regulations that are guided by ambient air pollution indicators.

The results of this paper suggest several areas for further research. Important questions remain as to the marginal role of different pollutants on health outcomes, or the possible interactions between them. Moreover, the estimates of pollution impacts are net of adaptation behavior, remaining silent about how much caregivers act in the background to ameliorate health shocks. We find suggestive evidence that pollution discriminates girls relatively more than boys, indicative of differentiated access to nutrition and health care. Understanding the drivers and role of compensating or reinforcing behavior by child caregivers is essential for designing appropriate supporting programs and public policies (see [Currie and Vogl \(2013\)](#) for a discussion).

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Air pollution and child development in India

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Abstract

In this paper, we study the impact of air pollution on child growth in India. We rely on wind direction to capture quasi-random variation in three main criteria air pollutants. We show that an increase in the average concentration of fine particulate matter by one standard deviation is accountable for almost 5 and 2.4 percentage points of stunting and severe stunting rates, respectively. We also find that ozone and carbon monoxide impact weight-related outcomes. Stunting has critical long-term health and economic consequences; through its impact on stunting, pollution exacerbates the height premium in earnings, with girls being more adversely affected than boys in India.

Keywords: Ambient air pollution; child health; anthropometry; wind direction; height premium.

JEL Classification Codes: C31, C36, I15, I32, Q53.

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