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Household solid fuel exposure and linear growth faltering in rural Indian children

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ABSTRACT

Introduction: Stunting affects approximately one-quarter of children globally. This study aims to examine how exposure to Household Air Pollution (HAP) from solid fuel use contributes to growth faltering among children.

Materials and methods: The study utilized data from the National Family Health Survey (NFHS-5, 2019–21) and included 185,721 rural children aged 0–59 months. Bivariate and multivariable logistic regression analyses were performed to evaluate the association between household cooking fuel and childhood stunting, with findings reported as 95% Confidence Intervals (CIs). A mediation analysis was further undertaken to examine the potential mediating role of child morbidity.

Results: Using clean fuels was inversely associated with stunting; the odds of stunting were 62% and 54% lower among children in households using only clean fuels compared to those exposed to solid fuels. Based on the adjusted multivariable logistic regression, clean cooking fuel was significantly associated with a decreased prevalence of stunting ($\beta = -0.029$, $p = 0.050$). Mediation analysis indicated significant effects of cooking fuel on stunting for the controlled direct effect (CDE = 0.972, $p = 0.050$) and natural direct effect (NDE = 0.973, $p = 0.044$) but not for the natural indirect effect via morbidity (NIE = 1.000, $p = 0.893$). The marginal total effect (MTE=0.973, $p = 0.044$) supported a small but significant protective association of child stunting with clean cooking fuel.

Conclusion: The findings suggest that reducing exposure to household air pollution through clean cooking fuels, alongside broader socio-economic interventions, may contribute to lowering childhood stunting in India.

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Introduction

Malnutrition, defined as deficiencies or excesses in nutrient intake of overall nutrition status associated with numerous adverse health consequences and represents a significant global health concern; it is estimated to cause 45% (3.1 million deaths) of all under-five child mortality directly and indirectly [1]. Undernutrition compromises immunity, reduces lung function and its ventilator capacity, and increases vulnerability to both infectious diseases as well as non-communicable diseases such as respiratory illnesses [2]. In 2020, the majority of stunted (94%), wasted (97%), and overweight children (75%) were residing in Africa and Asia [3]. Stunting is a widely recognized indicator of chronic undernutrition and is defined as a Height-for-Age Z-score (HAZ) below −2 standard deviations from the median of the World Health Organization (WHO) Child Growth Standards. Children affected by stunting are at increased risk of adverse health outcomes, including impaired lung function, asthma, and pneumonia [4-6]. In addition, stunting is associated with a higher risk of morbidity and mortality during childhood and can have lasting consequences for physical growth, cognitive development, educational attainment, and productivity in adulthood [7]. In 2011, approximately 165 million children younger than five years were affected by stunting worldwide, with more than one in three children residing in low- and middle-income countries experiencing chronic undernutrition [8].

According to the WHO conceptual framework, childhood stunting is influenced by both immediate determinants, such as inadequate nutrition and infections, and broader underlying factors, including poor water and sanitation, adverse socioeconomic conditions, environmental influences, and limited access to healthcare and education. Persistent wasting during early childhood and the coexistence of wasting and stunting markedly increase the risk of mortality [9].

Childhood malnutrition remains a significant burden on India. In India, as reported in second rounds of the National Family Health Survey [10], nearly half of children under three had extremely poor height-for-age ratios [11]. With significant gains in some maternal and child health indicators, stunting has emerged as one of the most prevalent forms of malnutrition globally: >20% are born with a stunted growth [12] and together contribute to almost half of all growth faltering among <5 children in India. This same persisting problem is due to various environmental and socio-economic elements. Exposure to household air pollution generated by the use of solid fuels for cooking and heating represents a major environmental determinant of ill health and remains an underappreciated global public health concern. Biomass fuels (firewood, crop residue, and dung) are widely used by low-income households in rural settings [13]. Certain fuels produce more than ten times the particulate matter of cleaner types [14]. Children in those families are obviously influenced by high quantity of indoor smoke because they stay inside or carried by mothers during cooking [11]. Household Air Pollution (HAP) is one of the most concerning environmental risk factors and causes almost 4 million premature deaths annually around the globe [15–16]. Because the respiratory and immune systems of children are still developing in early childhood, they are uniquely sensitive to the harmful effects of air pollutants. IAP is also an important contributor to child mortality [17]. In India, nearly 56% of children younger than five years spend much of their time indoors, frequently accompanying their mothers while meals are being prepared, which may increase their exposure to household air pollution [18–19]. Previous studies in India have suggested that solid fuel use is responsible for approximately 20% of child deaths and that ARI accounts for 24% of all deaths amongst children under-five [11]. Due to their smaller lung

volume, an immature immune system and higher respiratory rate relative to body size, children are particularly sensitive to toxicity through inhalation [20]. Household air pollution exposure is also associated with respiratory infections, low birth weight, stunting and developmental delays [21-22].

Although the critical role of child malnutrition as health and population priority problems has been demonstrated worldwide and nationally, links between Household Air Pollution (HAP) exposure and nutritional outcomes among children in India remains less investigated. While undernutrition due to dietary deficiency, infection, or poverty remains a major focus of most studies on child health in India, less empirical attention has been given to environmental factors such as indoor air pollution. The nexus of malnutrition and air pollution is especially worrying in rural, low-income households dependent on solid fuels. Since child growth is a reflection of the nutrients they consume and the environment in which children are raised [6], it is important to assess whether exposure to household air pollution contributes to growth faltering or stunting. Understanding this relationship is important for designing integrated interventions that address food security, healthcare and also environmental determinants of child health.

Materials and methods

Data source

This study utilized data from the fifth round of the National Family Health Survey (NFHS-5, 2019–21), a nationally representative household survey conducted by the Ministry of Health and Family Welfare, Government of India. The survey covered all 36 states and union territories and provides comprehensive information on demographic, socioeconomic, health, nutrition, and environmental characteristics. NFHS-5 employed a multistage stratified sampling design, using a three-stage sampling procedure in rural areas and a two-stage sampling procedure in urban areas. In rural settings, villages were selected as Primary Sampling Units (PSUs) using Probability Proportional to Size (PPS) sampling, followed by the systematic selection of households within each PSU. Information on anthropometric measurements, household characteristics, and environmental exposures, including the primary type of cooking fuel used as a proxy for Household Air Pollution (HAP), was collected using standardized survey instruments.

The present analysis was restricted to rural children aged 0–59 months who had valid anthropometric measurements and complete information on household cooking fuel, child stunting, child morbidity, and all selected covariates included the study. Children residing in urban areas, and observations with missing or implausible anthropometric measurements were excluded (N=47,199). For the multivariable regression and mediation analyses, observations with missing values on the outcome, exposure, mediator, or any covariates included in the models were excluded using a complete-case approach (N=34,406). The Fig. 1 shows final analytical sample for the regression analyses comprised 151,315 children. All analyses incorporated sampling weights to account for the complex survey design and to produce nationally representative estimates.

Outcome variable

The primary outcome variable was child stunting, an indicator of chronic undernutrition. Stunting was defined according to the World Health Organization (WHO) as a Height-for-Age z-score (HAZ) more than two standard deviations below the reference median (WHO, 2020). It was coded as a binary variable, with a value of 1 if the child was stunted and 0 otherwise.

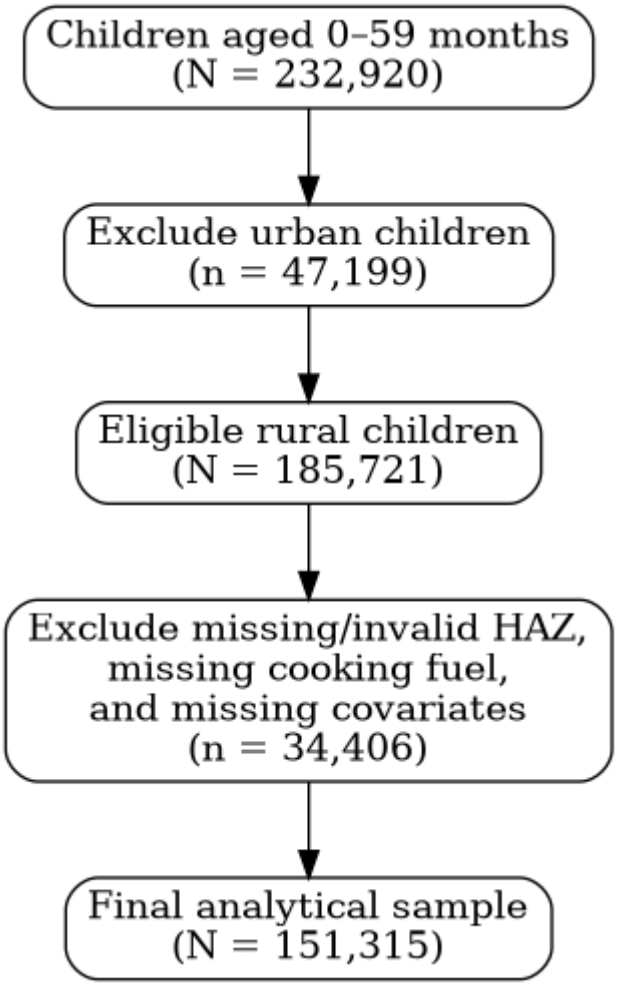

Fig. 1. Flow chart of sample selection

Independent variable

The main exposure variable of study was type of cooking fuel used in the household, which served as a proxy for Household Air Pollution (HAP). However, this measure does not capture variations in actual exposure arising from household ventilation, stove technology, cooking location, cooking duration, fuel stacking (simultaneous use of multiple fuel types), or individual time–activity patterns, which were not available in the NFHS-5 dataset. Cooking fuels were divided into two categories: solid fuels (wood, charcoal, crop residues, dung cakes, coal, and lignite) and clean fuels (electricity, Liquefied Petroleum Gas [LPG], biogas, and natural gas).

Control variables

According to earlier studies, various characteristics related on childhood stunting, covariates were selected to account for child-, maternal-, household-, and environmental-level characteristics [23-25]. Child-level variables included age (months), sex, recent morbidity (having diarrhea or fever in the last two weeks), birth order, and birth weight. Maternal characteristics included age at childbirth, educational attainment, Body Mass Index (BMI),

and anaemia status. Household characteristics included caste, religion, and wealth index.

Statistical analysis

Descriptive statistics, such as cross-tabulations and Chi-square (χ^2) tests, were utilized to assess the relevance of bivariate relationships. Multivariate logistic regression models with 95% Confidence Intervals (CIs) were applied to estimate the association between household fuel type and child stunting, controlling for all covariates. To further explore the potential pathway linking household cooking fuel and childhood stunting, mediation analysis was performed using child morbidity as the hypothesized mediator. The analysis estimated the Controlled Direct Effect (CDE), Natural Direct Effect (NDE), Natural Indirect Effect (NIE), and Marginal Total Effect (MTE). All analyses considered the intricate survey design through the "svy" commands in Stata version 18.

Integration with the Gut–Lung axis framework

Fig. 2 illustrates the conceptual framework where exposure to household air pollution from solid fuels may trigger inflammatory reactions in the respiratory system, potentially altering gut microbiota composition, impairing nutrient absorption, and leading to stunted linear growth.

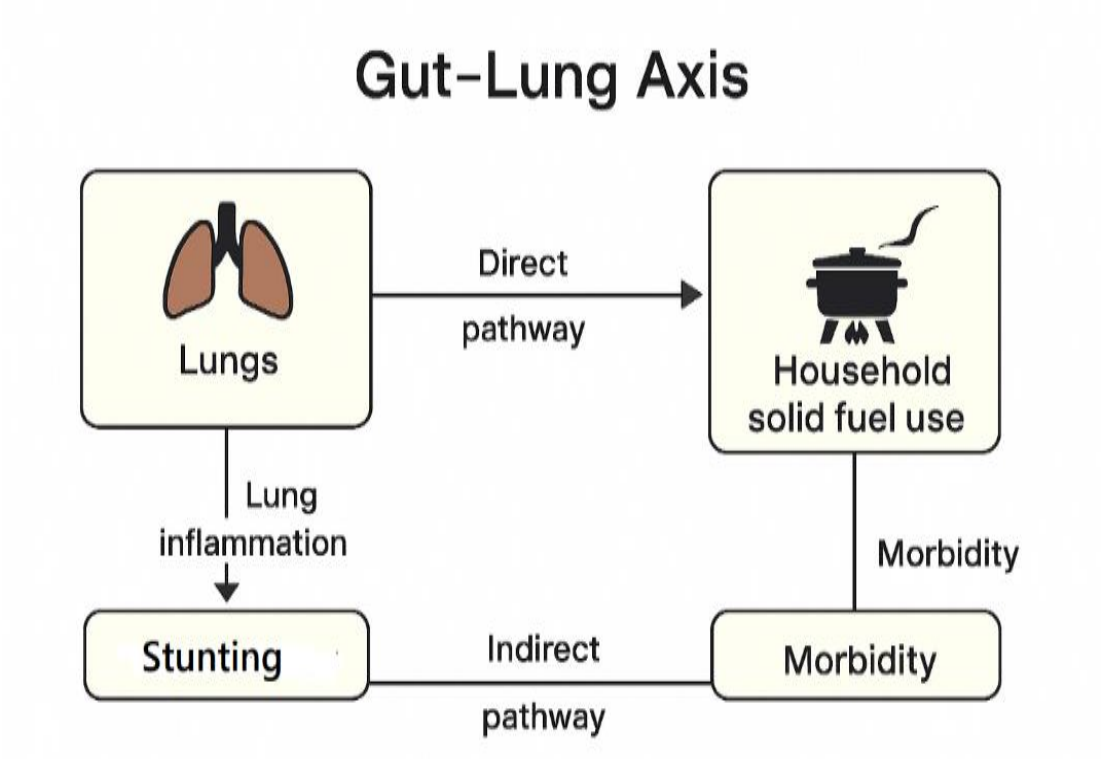

Fig. 2. Conceptual framework

Results and discussion

Table 1 indicated that the prevalence of stunting varied significantly across socio-demographic and health-related characteristics. Children from households using solid cooking fuel had a substantially higher prevalence of stunting (39.5%) compared with those using clean fuels (31.7%), and this difference was statistically significant (χ^2=854.3, p<0.001). Sex of the child showed a significant association, with male children more likely to be stunted (38.0%) than female children (35.8%) (χ^2=79.39, p<0.001). Significant differences in the prevalence of childhood stunting were observed across categories of child age, birth weight, and birth order.The prevalence of stunting increased from 25.5% among infants aged <1 year to 41.3% among one-year-old children, remaining high among children aged 2–4 years. Children with low birth weight (<2.5 kg) had the highest prevalence of stunting (44.0%), compared with those weighing 2.5 kg (39.7%) and >2.5 kg (33.0%) at birth. Similarly, the prevalence of stunting increased with birth order, from 33.1% among first-born children to 42.4% among children of birth order three or higher. Mother's age at birth displayed a strong gradient, where children born to mothers younger than 20 years had the highest stunting prevalence (41.0%). Children of mothers with no education had the highest stunting prevalence (45.2%), followed by those with primary education (42.3%). Scheduled Castes (40.2%) and Scheduled Tribes (39.3%) showed higher levels of stunting compared with OBC (36.8%) and particularly the 'Others' category (29.3%) (χ^2=819.6, p<0.001). Anemia status was positively associated with stunting, as anemic children were more likely to be stunted (38.3%) compared with non-anemic children (34.8%) (χ^2=202.8, p<0.001). Household wealth status exhibited the strongest gradient. Stunting was highest among children from the poorest households (44.9%), followed by poorer (38.5%), middle (33.7%), and richer (27.5%) households.

Table 1. Percentage distribution of Stunting by Background Characteristics

Background characteristics	Stunted	Otherwise	λ^2 (df)	*P*	Total N *(%)*
Cooking Fuel					
Solid	39.52	68.29	841.314	*<0.001*	101,071 (*66.80*)
Clean	31.87	68.18	(1)		50,244 (*33.20*)
Child Morbidity					
Yes	37.24	62.76	1.460		26,887 (*17.77)*
No	36.93	63.07	(1)	*0.227*	124,428 (82.23*)*
Sex of the Child					
Male	38.03	61.97	79.394		78,103 (*51.62)*
Female	35.86	64.14	(1)	*<0.001*	73,212 *(48.38*)
Child Age (year)					
0	25.45	74.55			29,785 *(19.68)*
1	41.30	58.70			29,606 *(19.57)*
2	39.70	60.30		*<0.001*	30,183 *(19.95)*
3	41.15	58.85	2300		30,564 *(20.20)*
4	37.16	62.84	(4)		31,177 *(20.60)*

Table 1. Continued

Background characteristics	Stunted	Otherwise	λ^2 (df)	*P*	Total N *(%)*
Birth weight					
<2.5 Kg	44.04	55.96	1100		*23,001 (16.94)*
2.5 Kg	39.72	60.28	(2)	*<0.001*	*27,388 (20.17)*
>2.5Kg	33.02	66.98			*85,386 (62.89)*
Birth Order					
1	33.13	66.87			55,844 *(36.91)*
2	36.25	63.75	950.25	*<0.001*	*49,227 (32.53)*
3 and above	42.40	57.60	(2)		*46,244 (30.56)*
Mother age at birth					
<20	41.04	58.96			51,432 *(33.79)*
20-24	35.95	64.05			76,662 *(50.66)*
25-29	31.19	68.81	845.310	*<0.001*	19,095 *(12.62)*
30-34	29.45	70.55	(4)		3,524 *(2.33)*
>35	31.52	68.48			602 *(0.40)*
Mother Education					
No education	45.40	54.60			36,721 *(24.27)*
Primary	42.34	57.66	2800	*<0.001*	20,752 *(13.71)*
Secondary	34.09	65.91	(3)		77,717 *(51.36)*
Higher and above	24.79	75.21			16,125 *(10.66)*
Social groups					
SC	40.20	59.80			33,513 *(22.15)*
ST	39.31	60.69	819.64	*<0.001*	35,344 *(23.36)*
OBC	36.76	63.24	(3)		60,162 *(39.76)*
Others	29.29	70.71			22,296 *(14.73)*
Religion					
Hindu	37.23	62.77			117,915 *(77.93)*
Muslim	37.25	62.50	230.55	*<0.001*	14,912 *(9.85)*
Christian	37.02	62.98	(3)		14,072 *(9.30)*
Others	26.42	73.58			4,416 *(2.92)*
Mother Anemia					
Yes	38.30	61.70	202.84	*<0.001*	91,435 *(60.43)*
No	34.81	65.19	(1)		59,880 *(39.57)*
Mother BMI					
Underweight	44.10	55.90			31,223 *(20.63)*
Normal	36.66	63.34	1400	*<0.001*	97,546 *(64.47)*
Total overweight	28.51	71.49	(2)		22,546 *(14.90)*

Table 1. Continued

Background characteristics	Stunted	Otherwise	λ^2 (df)	*P*	Total N *(%)*
Wealth status					
Poorest	44.87	55.13			48,260 *(31.89)*
Poorer	38.51	61.49	3700		40,606 *(26.84)*
Middle	33.69	66.31	(4)	*<0.001*	30,493 *(20.15)*
Richer	27.46	72.54			21,072 *(13.93)*
Richest	21.56	78.44			10,884 *(7.19)*

Notes: SC-Schedule Caste; ST-Schedule Tribe; OBC-Other Backward Class

Table 2. Logistic regression of child stunting on cooking fuel, morbidity, and covariates

Stunting	Coefficient	P	95% CI	
Cooking fuel	-0.026	*0.075*	-0.055	0.002
Morbidity	0.015	0.565	-0.035	0.065
Interaction (cooking fuel × morbidity)	0.012	0.707	-0.049	0.072
Sex of the child: female	-0.100	<0.001	-0.122	-0.079
Child Age	0.095	<0.001	0.087	0.102
Mother age at birth	-0.073	<0.001	-0.088	-0.059
Birth order 2	0.083	<0.001	0.057	0.109
Birth order 3	0.165	<0.001	0.137	0.193
Primary education of Mother	-0.052	0.004	-0.087	-0.017
Secondary education of Mother	-0.233	<0.001	-0.261	-0.204
Higher Secondary and above	-0.387	<0.001	-0.434	-0.340
Wealth status: Poorer	-0.154	<0.001	-0.182	-0.126
Wealth status: Middle	-0.278	<0.001	-0.312	-0.244
Wealth status: Richer	-0.484	<0.001	-0.525	-0.442
Wealth status: Richest	-0.696	<0.001	-0.753	-0.639
Mother have Anaemia	0.081	<0.001	0.059	0.103
Body Mass Index: Normal	-0.246	<0.001	-0.272	-0.219
Body Mass Index: Overweight	-0.451	<0.001	-0.489	-0.412
Caste	-0.060	<0.001	-0.071	-0.049
Religion	-0.039	<0.001	-0.053	-0.024

Table 2 presents the multivariable logistic regression estimates for childhood stunting. After adjustment for potential confounders, neither household cooking fuel (β = –0.026, p = 0.075), child morbidity (β = 0.015, p = 0.565), nor their interaction (β = 0.012, p = 0.707) was significantly associated with childhood stunting. Compared with male children, female children were significantly less likely to be stunted (β = –0.100, p < 0.001) (Fig. 3), whereas increasing child age (β = 0.095, p < 0.001) and higher birth order were associated with a greater likelihood of stunting. Higher maternal age at childbirth, maternal education, household wealth, and normal or overweight maternal BMI were significantly associated with lower odds of stunting, on other hand maternal anaemia was significantly associated with higher odds of childhood stunting (β = 0.081, p < 0.001).

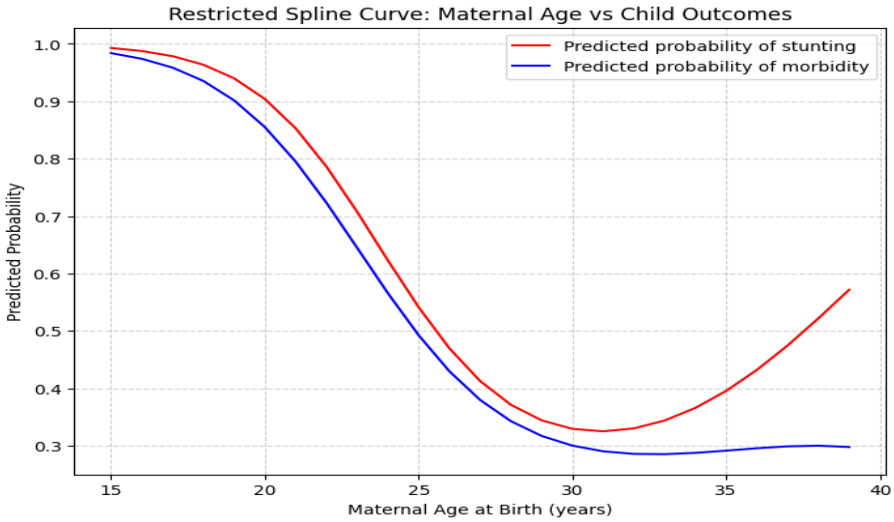

Fig. 3. Restricted cubic spline on maternal age birth and child outcome

Table 3. Logistic regression of morbidity on cooking fuel and covariates

Morbidity	Coefficient	*P*	95% CI	
Cooking fuel	-0.073	<0.001	-0.105	-0.042
Sex of the child: female	-0.106	<0.001	-0.132	-0.080
Child Age	-0.137	<0.001	-0.146	-0.128
Mother age at birth	-0.089	<0.001	-0.107	-0.071
Birth order 2	-0.007	0.650	-0.038	0.024
Birth order 3	0.007	0.695	-0.027	0.041
Primary education of Mother	0.181	<0.001	0.137	0.225
Secondary education of Mother	0.189	<0.001	0.153	0.225
Higher Secondary and above	0.166	<0.001	0.111	0.222
Wealth status: Poorer	-0.030	0.095	-0.065	0.005
Wealth status: Middle	-0.117	<0.001	-0.158	-0.075
Wealth status: Richer	-0.163	<0.001	-0.213	-0.114
Wealth status: Richest	-0.278	<0.001	-0.343	-0.212
Mother have Anaemia	0.048	<0.001	0.021	0.074
Body Mass Index: Normal	-0.077	<0.001	-0.109	-0.044
Body Mass Index: Overweight	-0.035	0.131	-0.081	0.010
Caste	0.014	0.037	0.001	0.028
Religion	0.045	<0.001	0.028	0.062

Table 4. Mediation analysis of the effect of solid fuel exposure on child stunting through child morbidity

Estimate	Coefficient	*P*	95% CI	
CDE	0.974	*0.082*	0.946	1.003
NDE	0.976	*0.086*	0.950	1.003
NIE	1.000	*0.150*	1.000	1.001
MTE	0.976	*0.082*	0.950	1.003

Notes: *CDE: Controlled Direct Effect, NDE: Natural Direct Effect, NIE: Natural Indirect Effect, MTE: Marginal Total Effect*

Table 3 presents the logistic regression estimates for child morbidity. The use of clean cooking fuel was significantly associated with a lower likelihood of child morbidity ($\beta = -0.073$, $p < 0.001$). Female children ($\beta = -0.106$, $p < 0.001$), older children ($\beta = -0.137$, $p < 0.001$), and children born to older mothers ($\beta = -0.089$, $p < 0.001$) were less likely to experience morbidity. Higher household wealth was associated with reduced odds of morbidity, whereas maternal anaemia ($\beta = 0.048$, $p < 0.001$), maternal education, caste, and religion were significantly associated with an increased likelihood of child morbidity. Birth order was not significantly associated with morbidity.

Table 4 displays the findings of the mediation analysis. The mediation analysis indicated that the controlled direct effect (CDE = 0.974, p = 0.086) and natural direct effect (NDE = 0.976, p = 0.086) of cooking fuel on stunting were statistically significant, whereas the natural indirect effect through morbidity was not significant (NIE = 1.000, p = 0.150). The marginal total effect (MTE = 0.976, p = 0.082) indicated a minor yet significant protective effect of clean cooking fuel on stunting. These findings suggest that the influence of clean cooking fuel on childhood stunting is mainly direct and not influenced by morbidity.

This study examined the relationship between household cooking fuel, child morbidity, and childhood stunting among children under five years of age using nationally representative data from India. The findings revealed that stunting was significantly more prevalent among children exposed to solid fuels compared with those in households using clean fuels. Stunting was also strongly associated with several socio-demographic and health-related factors, including the sex of child, mother age at birth, educational status of mother, caste, religion, anemia status, and household wealth. These patterns underscore the continuing impact of socio-economic and health disparities on nutritional outcomes among Indian children.

Previous research has shown that diarrhoeal disease contributes notably to linear growth faltering, with one study attributing approximately 25% of stunting to repeated diarrhoeal episodes [26]. Evidence from a pooled analysis of seven longitudinal cohorts in four low-income countries indicated that diarrhoeal illness during the first two years of life had only a limited impact on children's linear growth [27]. Similarly, a study in Gambian infants reported that although diarrhoeal episodes were common, their frequency did not fully explain the observed growth faltering [28]. These findings suggest that while diarrhoea contributes to poor growth, it is unlikely to be the sole driver of stunting.

The regression analysis conducted in this research verified that the use of solid fuels continued to be a notable predictor of stunting, even when

accounting for maternal, child, and household factors. Children living in homes that use solid fuels experienced an increased risk of growth delays, highlighting the harmful impacts of indoor air pollution. Maternal education and household wealth served as significant protective factors, aligning with findings that connect socio-economic status to better child nutrition [29, 30]. In addition to nutritional factors, environmental and infrastructural elements-especially Water, Sanitation, Hygiene (WASH), and air quality—significantly influence child development. Harttgen et al. (2019) showed that differences in stunting rates between South Asia and sub-Saharan Africa could not be solely attributed to mortality selection [31]. In India, consistently inadequate WASH conditions and elevated levels of open defecation continue to be significant factors in stunting disparities [32, 33].

Maternal age at childbirth also emerged as an important determinant of stunting, with younger maternal age linked to higher prevalence [34]. The findings indicate that while mothers' nutrition knowledge helps reduce stunting among children with lower height-for-age, formal education plays a more significant role in enhancing height-for-age in higher quantiles. Consistent with previous studies, stunting was more common among older children, those of higher birth order, and those with recent episodes of loose stools or poorly diversified diets.

The mediation analysis suggested that the association between household cooking fuel and childhood stunting was primarily explained by the direct pathway rather than indirectly through child morbidity. Nevertheless, these findings should be interpreted cautiously. Because the exposure, mediator, and outcome were measured simultaneously in the cross-sectional NFHS-5 survey, the temporal assumptions required for causal mediation analysis cannot be verified. Consequently, the estimated direct and indirect effects represent statistical associations rather than confirmed causal mechanisms. Longitudinal studies with repeated measurements are needed to establish the temporal sequence between household air pollution exposure, childhood morbidity, and linear growth and to validate the proposed mediation pathway. Comparable results a study in 2018 which was indicated that children aged 0–3 years in households using coal were notably shorter, displaying standardized z-score differences of −0.37 SD (approximately 1.3 cm less in height) [35]. These research findings together show that air pollution in households significantly affects the linear growth of children. Although nutrition, genetics, and physical activity are key factors influencing height, indoor air pollution might exacerbate growth deficiencies, especially as other protective elements decline with age [36]. A study from the UK observed that accounting for birth weight decreased the link between air pollution and the height of children by merely 15–20%, indicating that environmental factors play a separate role in growth results [37].

The breakdown of effects (CDE, NDE, NIE, and MTE) showed that the majority of the link between cooking fuel and stunting was accounted for by the direct pathway. The direct effects, both controlled and natural, continued to be statistically significant, whereas the indirect effect via morbidity was minimal. This result suggests that approaches concentrating only on decreasing child morbidity might not significantly diminish the nutritional impact associated with the use of solid fuels [38]. Rather, initiatives that enhance access to clean cooking fuels are expected to produce quicker and more lasting advancements in children's growth results [39]. While genetic influences account for a significant portion of height variability—approximately 80%—micronutrient interventions (such as iron or vitamin A supplementation) have demonstrated minimal effects on linear growth [40]. This indicates that growth stunting in developing nations might represent physiological adjustments to long-term shortages of calcium and

other nutrients that have low bioavailability [41].

Although the relationship between clean fuel and childhood stunting was modest, its potential public health importance should be interpreted within the broader population context. The findings of this study existing in India, where a substantial proportion of rural households continue to rely on solid fuels, even a small reduction in the risk of stunting could translate into a considerable number of children experiencing improved growth outcomes [38]. Nevertheless, the borderline statistical evidence observed in this study suggests that household cooking fuel alone is unlikely to be a major determinant of childhood stunting. Rather, exposure to household air pollution should be considered one of several interconnected environmental, socioeconomic, and maternal factors contributing to child growth [15]. Therefore, expanding access to clean cooking fuels should complement, rather than replace, established interventions aimed at improving maternal nutrition, child feeding practices, sanitation, healthcare access, and poverty reduction [40]. "The NFHS-5 report reported a higher prevalence of stunting among children from households that use solid fuels, mirroring the current results" [12]. A study by many researchers also recognized the negative impacts of indoor air pollution from biomass on child nutrition. Recent findings from WHO and UNICEF (2021) indicate that children subjected to household air pollution face an increased risk of developmental and growth issues [42]. In contrast to a study which was identified more pronounced indirect effects mediated by respiratory illness, this research did not observe notable mediation through sickness—possibly owing to contextual and definitional variations [43]. A study in 2021 discovered that the height-for-age disparity between Indian and sub-Saharan African children was noticeable at birth and continued during infancy, indicating prenatal factors influencing stunting [44]. They suggested that differences were primarily influenced by maternal health and prenatal investments instead of postnatal environmental factors [45]. Emerging evidence also suggests that maternal mental health plays an important role in child growth and development. Studies have reported that children of mothers with depressive symptoms are more likely to experience stunting, even after controlling for socioeconomic characteristics [46]. In addition, maternal depression has been associated with an increased risk of childhood diarrhoeal diseases and respiratory infections, indicating that its adverse effects on child health may operate through both biological and behavioural pathways, particularly in settings characterized by poverty and poor living conditions [47].

The results of this research support the complex causes of child stunting in India, influenced by environmental factors, maternal characteristics, and socio-economic disparities. While child morbidity was proposed as a mediator, evidence indicates that the direct effect of solid fuel exposure on stunting is more significant. Enhancing maternal education, postponing early childbearing, boosting child nutrition and anemia management, and tackling household poverty continue to be essential strategies [48]. At the same time, increasing access to clean cooking fuels provides a twofold advantage-lowering the risk of stunting while promoting India's wider goals in public health and sustainable development.

Limitation of the study

This study has some limitations. First, the cross-sectional design of the NFHS-5 does not permit causal inference or verification of the temporal ordering between household cooking fuel use, child morbidity, and childhood stunting. Second, although mediation analysis was undertaken to explore potential mechanisms, exposure, mediator, and outcome were measured simultaneously; therefore, the temporal assumptions underlying causal mediation analysis cannot be confirmed.

Therefore, the mediation findings should be interpreted as exploratory rather than evidence of a causal pathway. Third, household cooking fuel was used as a proxy for household air pollution and did not account for important determinants of exposure, such as ventilation, stove type, cooking location, fuel stacking, and cooking duration. Because these variables were not available in the NFHS-5, some degree of non-differential exposure misclassification is possible. Finally, the possibility of residual confounding due to unmeasured environmental and behavioural factors cannot be ruled out.

Conclusion

This study reveals that the use of solid cooking fuels is strongly linked to greater chances of childhood stunting in India, even when accounting for socioeconomic and demographic variables. Even though respiratory issues were seen as a possible mediator in the proposed gut–lung axis pathway, its impact was not statistically significant, suggesting that household air pollution primarily affects growth faltering directly instead of indirectly.

Beyond cooking fuel exposure, maternal education, age at childbirth, household wealth, caste, religion, and child anemia emerged as important predictors of stunting, highlighting the multidimensional drivers of poor growth outcomes. The findings suggest that increasing access to clean cooking fuels may make a modest contribution to reducing childhood stunting, particularly when implemented alongside broader interventions addressing maternal health, child nutrition, sanitation, and socioeconomic inequalities. First, large-scale promotion and sustained adoption of clean cooking fuels such as LPG and electricity should be prioritized under national programs like Pradhan Mantri Ujjwala Yojana to minimize children's exposure to household air pollution. Second, targeted nutrition and health interventions should be directed toward children of anemic mothers, socially disadvantaged groups, and households with lower wealth status, as these groups bear a disproportionate burden of stunting. Third, educational campaigns aimed at delaying early childbirth and encouraging female education can indirectly reduce stunting by improving maternal health and caregiving practices.

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Competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Ethical considerations

Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely observed by the authors.

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