

Household Tobacco Smoke Exposure as a Predictor of Child Stunting: A Multivariate Analysis in Indonesia

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ABSTRACT

Introduction: Stunting remains a significant public health concern in low- and middle-income countries, including Indonesia, with determinants that extend beyond nutrition and infection. Environmental exposures, particularly household tobacco smoke, have emerged as important contributors to impaired child growth. This study examined the association between household tobacco smoke exposure and stunting among children under five years of age, adjusting for selected biological and environmental factors. A cross-sectional analytical study was conducted among 420 children aged 6–59 months in urban and peri-urban areas of Makassar, Indonesia. Data collection involved caregiver interviews and standardized anthropometric measurements. Stunting was defined as a height-for-age z-score below -2 standard deviations according to World Health Organization (WHO) Child Growth Standards. Household tobacco smoke exposure was assessed through caregiver self-report. Multivariate logistic regression analysis was used to estimate adjusted odds ratios (AORs) and 95% confidence intervals (CIs). The overall prevalence of stunting was 36.7%. Children exposed to household tobacco smoke had a higher prevalence of stunting compared to those unexposed (45.7% vs. 26.6%). After adjustment for potential confounders, tobacco smoke exposure was significantly associated with increased odds of stunting (AOR = 2.30; 95% CI: 1.53–3.48; $p < 0.001$). Stunting was common among the study population, and the association between household tobacco smoke exposure and stunting persisted after adjustment for relevant factors. Child age was also significantly associated with stunting, while other variables were not statistically significant. In conclusion, household tobacco smoke exposure is significantly associated with stunting among children under five in Indonesia. These findings underscore the need to incorporate environmental and behavioral risk factors, including tobacco control, into comprehensive strategies for stunting prevention.

INTRODUCTION

Stunting, defined as impaired linear growth indicated by a height-for-age z-score below -2 standard deviations, remains a persistent indicator of chronic undernutrition and early-life adversity. In 2022, an estimated 148 million children under five years of age were affected by stunting globally, with the highest prevalence in low- and middle-income countries (1,2). In Indonesia, despite ongoing national initiatives to improve maternal and child nutrition, stunting continues to affect a significant proportion of children, making it a major public health and developmental concern (3). In addition to physical growth impairment, stunting is linked to long-term consequences such as reduced cognitive development, lower educational attainment, diminished economic productivity, and perpetuation of intergenerational disadvantage (4).

The etiology of stunting is complex and multifactorial, involving interactions among biological, environmental, and socioeconomic determinants. Established risk factors include inadequate dietary intake, recurrent infections, poor sanitation, suboptimal caregiving practices, and maternal undernutrition (2,5). These determinants function at multiple levels, ranging from immediate biological insults to broader structural conditions such as poverty and limited access to health services. Recently, research has expanded to consider environmental exposures that may affect child growth, including household air pollution and tobacco smoke exposure (6,7).

Household tobacco smoke exposure, particularly secondhand smoke (SHS), is a potentially modifiable environmental risk factor for impaired child growth. Biological mechanisms linking SHS exposure to growth restriction include hypoxia from carbon monoxide, systemic inflammation, impaired nutrient absorption, and

increased vulnerability to respiratory and infectious diseases (8,9). Prenatal exposure may further increase risk through placental dysfunction and intrauterine growth restriction. In Indonesia, where adult male smoking prevalence is among the highest globally and domestic smoking is common, young children are frequently exposed to tobacco smoke at home (10,11). However, tobacco smoke exposure is not consistently addressed in stunting prevention frameworks.

Empirical evidence on the relationship between SHS exposure and child growth is limited and inconsistent. Some studies have identified a significant association between household tobacco smoke exposure and stunting or impaired growth (6,9), while others report attenuated or non-significant associations after adjusting for socioeconomic and maternal factors. Many studies are limited by methodological issues, such as reliance on self-reported exposure, inadequate adjustment for confounders, and insufficient integration of multiple determinants within a single analytical framework (7). As a result, the independent contribution of household tobacco smoke exposure to stunting remains unclear, particularly in high-prevalence contexts like Indonesia.

This study addresses these gaps by examining the association between household tobacco smoke exposure and stunting among children under five years of age using a multivariate analytical approach. By adjusting for key biological, maternal, and environmental factors, the study aims to provide a robust estimate of the relationship between SHS exposure and child growth outcomes. Understanding this association is critical for informing comprehensive and context-sensitive stunting prevention strategies that extend beyond conventional nutrition-focused interventions.

METHODS

Study Design and Setting

A cross-sectional analytical design was used to examine the association between household tobacco smoke exposure and stunting among children under five years of age. The research was conducted in multiple community health service areas (Puskesmas catchment areas) within urban and peri-urban districts of Makassar, Indonesia, from October 2024 to March 2025. Study sites were selected based on a high reported prevalence of household smoking and the presence of routine child growth monitoring programs through integrated health posts (Posyandu).

Participants and Sampling

The study population comprised children aged 6–59 months and their primary caregivers. Eligibility required residence in the same household for at least six months prior to data collection. Children with congenital anomalies, chronic illnesses affecting growth, or recent hospitalization were excluded to minimize confounding unrelated to environmental exposure. A multistage cluster sampling technique was implemented. In the first stage, subdistricts were randomly selected from predefined health surveillance zones. In the second stage, households with eligible children were identified using Posyandu registries, and participants were recruited consecutively within selected clusters.

Sample size was estimated using G*Power version 3.1.9.7 (12) for logistic regression analysis. With a moderate effect size (odds ratio ≈ 1.5), a significance level (α) of 0.05, and statistical power of 80% ($1-\beta = 0.80$), the minimum required sample was 384 participants. This calculation was based on the national stunting prevalence of 21.6% (Kementerian Kesehatan Republik Indonesia, 2023). (3) To account for potential non-response and incomplete data, the final sample size was increased to 420 children.

Data Collection Procedures

Trained field enumerators collected data using standardized questionnaires and anthropometric measurement protocols. Caregivers were interviewed face-to-face in the local language with an adapted structured questionnaire based on WHO household survey instruments. The questionnaire captured sociodemographic characteristics, child feeding practices, and environmental conditions, including water, sanitation, and tobacco smoke exposure.

Anthropometric measurements followed WHO guidelines. Child height was measured with a calibrated portable stadiometer (accuracy ± 0.1 cm). Each measurement was taken twice, and the average was recorded to ensure reliability. Stunting was defined as a height-for-age z-score (HAZ) below -2 standard deviations, according to WHO Child Growth Standards. Household tobacco smoke exposure was assessed via caregiver self-report, including the number of smokers, frequency of smoking, and smoking location within the household. Households

were classified as “exposed” if daily smoking occurred inside the home. Although no biochemical validation (such as cotinine measurement) was performed, this operational definition is widely used in population-based studies to approximate secondhand smoke exposure.

Data Analysis

All data were checked for completeness and accuracy before analysis and entered into IBM SPSS Statistics version 29.0. Descriptive statistics summarized participant characteristics. Bivariate analyses, including chi-square tests for categorical variables and independent t-tests for continuous variables, explored initial associations. Multivariate logistic regression estimated adjusted odds ratios (AORs) and 95% confidence intervals (CIs) for the association between household tobacco smoke exposure and stunting, controlling for potential confounders. Variables for the multivariate model were selected based on theoretical relevance and bivariate analysis results. Model fit was evaluated using the Hosmer–Lemeshow goodness-of-fit test. Multicollinearity among independent variables was assessed using variance inflation factors (VIF), with values below 10 indicating acceptable levels. Statistical significance was set at $p < 0.05$.

Ethical Considerations

Ethical approval was granted by the Research Ethics Committee of STIKES Nani Hasanuddin Makassar (Ref. No. 674/2018). Written informed consent was obtained from all participating caregivers before data collection. Participant confidentiality and data privacy were maintained throughout the study in accordance with the principles of the Declaration of Helsinki.

RESULT AND DISCUSSION

RESULT

Characteristics of Study Participants

A total of 420 children aged 6–59 months were included in the analysis, with 210 (50%) A total of 420 children aged 6–59 months were included in the analysis, with equal distribution between households exposed to tobacco smoke ($n = 210, 50.0\%$) and those unexposed ($n = 210, 50.0\%$). The overall prevalence of stunting was 36.7% ($n = 154$). Stunting was substantially more common among children exposed to household tobacco smoke (45.7%) compared to those living in non-smoking households (26.6%). Children in smoke-exposed households were slightly older on average (mean 34.1 ± 14.9 months) than those in unexposed households (30.3 ± 16.1 months). Mean birth weight was comparable between groups (3.02 ± 0.50 kg vs. 2.98 ± 0.48 kg). (Table 1)

Table 1. Participant Characteristics by Household Tobacco Smoke Exposure (N = 420)

| Variable | Total (N = 420) | Exposed (n = 210) | Unexposed (n = 210) |
|----------------------------|-----------------|-------------------|---------------------|
| Stunting | | | |
| Yes | 154 (36.7%) | 96 (45.7%) | 58 (26.6%) |
| No | 266 (63.3%) | 114 (54.3%) | 152 (72.4%) |
| Mean Age (months) | 32.2 ± 15.6 | 34.1 ± 14.9 | 30.3 ± 16.1 |
| Mean Birth Weight (kg) | 3.00 ± 0.49 | 3.02 ± 0.50 | 2.98 ± 0.48 |
| Sex | | | |
| Male | 195 (46.4%) | 88 (41.9%) | 107 (50.9%) |
| Female | 225 (53.6%) | 114 (54.3%) | 111 (49.1%) |
| Maternal Education | | | |
| No Schooling | 48 (11.4%) | 22 (10.5%) | 26 (12.4%) |
| Primary | 165 (39.3%) | 87 (41.4%) | 78 (37.1%) |
| Secondary | 126 (30.0%) | 59 (28.1%) | 67 (31.9%) |
| Tertiary | 81 (19.3%) | 34 (16.2%) | 47 (22.4%) |
| Sanitation Facility | | | |
| Improved | 288 (68.6%) | 141 (67.1%) | 147 (69.5%) |
| Unimproved | 132 (31.4%) | 69 (32.9%) | 63 (30.0%) |

Bivariate and Multivariate Associations

Bivariate analysis showed a significant association between household tobacco smoke exposure and stunting ($\chi^2 = 18.87$, $p < 0.001$), indicating a higher prevalence of stunting among exposed children. Mean age also differed significantly between groups ($t = 2.52$, $p = 0.012$), whereas birth weight did not differ significantly ($p = 0.346$). No significant differences were observed for sex, maternal education, or sanitation status (all $p > 0.05$) (Table 2).

Table 2. Correlation Matrix of Study Variables (N = 420)

| Variable | 1 | 2 | 3 | 4 | 5 | 6 |
|---------------------------|---------|-------|-------|------|------|------|
| 1. Tobacco Smoke Exposure | — | | | | | |
| 2. Stunting | 0.21*** | — | | | | |
| 3. Age (months) | 0.12* | 0.10* | — | | | |
| 4. Birth Weight (kg) | 0.05 | -0.06 | -0.02 | — | | |
| 5. Sex | 0.05 | 0.03 | 0.01 | 0.02 | — | |
| 6. Maternal Education | -0.07 | -0.05 | -0.03 | 0.04 | 0.01 | — |
| 7. Sanitation Facility | -0.02 | -0.03 | 0.02 | 0.01 | 0.00 | 0.06 |

Note: Values represent approximate correlation coefficients derived from bivariate associations. $p < 0.05$; *** $p < 0.001$

Household tobacco smoke exposure was significantly associated with higher odds of stunting in both crude (OR = 2.31; 95% CI: 1.55–3.45; $p < 0.001$) and adjusted models (AOR = 2.30; 95% CI: 1.53–3.48; $p < 0.001$), demonstrating a robust relationship independent of measured confounders. Child age exhibited a small but significant association with stunting (AOR = 1.01; 95% CI: 1.00–1.02; $p = 0.011$), whereas birth weight, sex, maternal education, and sanitation were not statistically significant. The consistency between crude and adjusted estimates indicates minimal confounding. Model diagnostics showed acceptable fit (Hosmer–Lemeshow $p = 0.62$; Nagelkerke $R^2 = 0.18$) (Table 3).

Table 3. Multivariate Logistic Regression Models Predicting Odds of Stunting (N = 420)

| Variable | Model 1 (Crude OR) | 95% CI | p-value | Model 2 (Adjusted OR) | 95% CI | p-value |
|-------------------------|-----------------------|-------------|-----------|--------------------------|-------------|----------|
| Tobacco Smoke Exposure | 2.31 | 1.55 – 3.45 | <0.001*** | 2.30 | 1.53 – 3.48 | <0.001** |
| Age (months) | 1.01 | 1.00 – 1.02 | 0.018* | 1.01 | 1.00 – 1.02 | 0.011* |
| Birth Weight (kg) | 0.79 | 0.52 – 1.20 | 0.269 | 0.77 | 0.50 – 1.18 | 0.231 |
| Sex (Male vs Female) | 1.08 | 0.72 – 1.61 | 0.712 | 1.10 | 0.73 – 1.66 | 0.646 |
| Maternal Education | | | | | | |
| No schooling | 1.10 | 0.48 – 2.53 | 0.823 | 1.07 | 0.45 – 2.56 | 0.878 |
| Primary | 1.35 | 0.80 – 2.28 | 0.256 | 1.30 | 0.75 – 2.27 | 0.344 |
| Secondary | 1.05 | 0.61 – 1.80 | 0.861 | 1.04 | 0.60 – 1.82 | 0.883 |
| Sanitation (Unimproved) | 1.06 | 0.70 – 1.61 | 0.782 | 1.03 | 0.67 – 1.58 | 0.887 |

Note: Model 1 = Crude (unadjusted) analysis; Model 2 = Adjusted for all covariates; Model 2 Hosmer–Lemeshow test: $p = 0.62$; Nagelkerke R^2 : 0.18; Reference categories:

Female (sex), Tertiary education (maternal education), Improved sanitation; $p < 0.05$, *** $p < 0.001$.

The forest plot presents the adjusted associations between study variables and stunting. Household tobacco smoke exposure demonstrates a clear and statistically significant association, with an adjusted odds ratio above 1 and a confidence interval that does not cross the null value, indicating increased odds of stunting among exposed children. Child age also shows a small but statistically significant effect. In contrast, birth weight, sex, maternal education, and sanitation have confidence intervals that cross 1, suggesting no statistically significant association in the adjusted model. Overall, the plot identifies tobacco smoke exposure as the strongest and most consistent factor associated with stunting in this analysis (Figure 1).

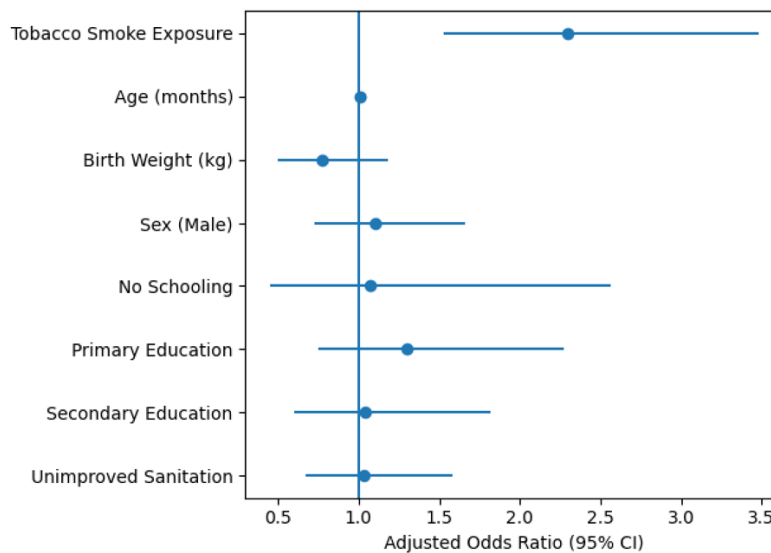


Figure 1. Forest plot of adjusted odds ratios (AOR) for factors associated with stunting among children under five. Error bars represent 95% confidence intervals. The vertical line indicates no association (OR = 1).

This study identified a significant association between household tobacco smoke exposure and stunting among children under five years of age. Children residing in smoke-exposed households exhibited more than twice the odds of being stunted compared to those in smoke-free environments, even after adjustment for key biological and environmental covariates. While not establishing a direct causal relationship, these findings indicate that secondhand smoke (SHS) exposure may be a critical environmental determinant that interacts with broader risk factors affecting child growth.

The observed association is biologically plausible and aligns with emerging evidence linking environmental tobacco exposure to impaired growth trajectories in early childhood. Tobacco smoke contains numerous toxic compounds, including carbon monoxide, nicotine, and fine particulate matter, which can contribute to hypoxia, systemic inflammation, and altered metabolic regulation (8,9). These mechanisms may impair nutrient utilization and increase susceptibility to infectious diseases, both established pathways that contribute to stunting. Furthermore, prenatal and early-life exposure to tobacco smoke has been associated with intrauterine growth restriction and compromised immune function, potentially increasing vulnerability to postnatal growth faltering (6,13).

The findings of this study are consistent with recent research in low- and middle-income settings that report significant associations between household air pollution, including SHS, and adverse child growth outcomes (6,9). Notably, the magnitude of association observed here is relatively stronger than in some previous reports. This difference may be attributable to contextual factors specific to Indonesia, where smoking prevalence among adult males is high and indoor smoking is socially normalized (11). In these environments, children may experience sustained and repeated exposure within confined household spaces, resulting in higher cumulative exposure levels compared to settings with stricter tobacco control norms.

A notable finding in this study is the absence of statistically significant associations between traditional determinants, such as maternal education and sanitation, and stunting in the adjusted model. Although these factors are consistently identified as key predictors in the literature (2,4), their lack of significance in this analysis may reflect complex interrelationships among household-level determinants. One possible explanation is that tobacco smoke exposure may function as a proximal environmental stressor that partially captures or mediates the effects of broader socioeconomic and behavioral conditions. Alternatively, shared underlying factors, including household behavioral patterns, risk perception, and health literacy, may simultaneously influence both smoking practices and child care behaviors, thereby reducing the independent effects of conventional predictors when modeled together. In addition to its epidemiological relevance, this study contributes conceptually by identifying household tobacco smoke exposure as a modifiable environmental factor within the broader framework of stunting determinants. Traditional stunting interventions have primarily focused on nutrition, infection control, and sanitation, while environmental exposures related to household behavior remain underrepresented in prevention strategies. The

findings suggest that integrating tobacco control into child health interventions may provide an additional pathway to reduce growth-related inequalities, particularly in settings with high exposure. ure settings.

Implications for Practice and Policy

These findings have significant implications for public health practice and policy development. Current stunting reduction strategies in many low- and middle-income countries, including Indonesia, primarily emphasize nutrition, breastfeeding, and hygiene interventions. However, the consistent association observed in this study underscores the need to broaden these strategies to address household-level environmental risk factors, particularly tobacco smoke exposure. At the community level, incorporating tobacco exposure screening into routine child health services, such as Posyandu-based growth monitoring, could facilitate early identification of at-risk households. Health promotion initiatives should include targeted counseling for caregivers about the risks of indoor smoking and its potential impact on child growth. At the policy level, strengthening smoke-free home campaigns and integrating tobacco control messages into maternal and child health programs may enhance the effectiveness of existing stunting-reduction efforts. Given the high prevalence of smoking among adult males in Indonesia, interventions that engage fathers and other household members are particularly important.

Strengths and Limitations

This study demonstrates several strengths. It employs a multivariate analytical approach that accounts for multiple biological and environmental determinants simultaneously, enabling a more robust estimation of the association between tobacco smoke exposure and stunting. Furthermore, the study provides context-specific evidence from a setting with a high prevalence of household smoking, which is underrepresented in the current literature. Nonetheless, several limitations should be acknowledged. First, tobacco smoke exposure was assessed through caregiver self-report, which may be subject to recall and social desirability biases. The lack of objective biomarkers, such as cotinine levels, may have resulted in exposure misclassification. Second, the cross-sectional design limits the ability to establish temporal or causal relationships between exposure and outcome. Third, residual confounding from unmeasured variables, including detailed dietary intake, indoor air pollution from other sources, or caregiving practices, cannot be entirely excluded. Finally, the findings may not be generalizable to rural or culturally distinct settings with different smoking patterns.

Future research should utilize longitudinal or prospective designs to clarify the temporal relationship between tobacco smoke exposure and child growth outcomes. Incorporating objective exposure measurements, such as biomarkers or environmental monitoring, would enhance the accuracy of exposure assessment. Additionally, studies examining behavioral and sociocultural determinants of household smoking practices may yield valuable insights for developing more effective, context-sensitive interventions. Investigating potential interactions between tobacco exposure and nutritional or infectious factors may further elucidate underlying mechanisms.

CONCLUSION

This study demonstrates a significant association between household tobacco smoke exposure and stunting among children under five in Indonesia. These findings highlight the importance of considering environmental and behavioral risk factors alongside traditional determinants of child growth. Expanding stunting prevention strategies to incorporate tobacco control measures at the household level may provide an additional, potentially impactful approach to improving child health outcomes.

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AUTHOR'S CONTRIBUTION STATEMENT

M : Conceptualization, Methodology, Supervision, Writing – Original Draft Preparation, KN: Data Curation, Investigation, Writing – Review & Editing. MTT: Formal Analysis, Resources, Project Administration.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

The authors acknowledge the use of AI to support language improvement, improve clarity, and improve the overall readability and structure of the manuscript.

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REFERENCES

1. World Health Organization. Improving early childhood development: WHO guideline: Summary. World Health Organization; 2023.
2. UNICEF. Improving young children's diets during the complementary feeding period. UNICEF programming guidance. 2020;76:118–22.
3. Kemenkes RI. Profil Kesehatan Indonesia tahun 2023. 2023. Report.
4. Victora CG, Christian P, Vdaletti LP, Gatica-Domínguez G, Menon P, Black RE. Revisiting maternal and child undernutrition in low-income and middle-income countries: variable progress towards an unfinished agenda. *The Lancet*. 2021;397(10282):1388–99.
5. Danaei G, Andrews KG, Sudfeld CR, Fink G, McCoy DC, Peet E, et al. Risk factors for childhood stunting in 137 developing countries: a comparative risk assessment analysis at global, regional, and country levels. *PLoS Med*. 2016;13(11):e1002164.
6. Yao M, Li L, Yang M, Wu Y, Cheng F. Household air pollution and childhood stunting in China: A prospective cohort study. *Front Public Health*. 2022;10:985786.
7. Islam S, Rana MJ, Mohanty SK. Cooking, smoking, and stunting: Effects of household air pollution sources on childhood growth in India. *Indoor Air*. 2021;31(1):229–49.
8. Merianos AL, Mahabee-Gittens EM, Stone TM, Jandarov RA, Wang L, Bhandari D, et al. Distinguishing exposure to Secondhand and Thirdhand Tobacco smoke among US children using machine learning: NHANES 2013–2016. *Environ Sci Technol*. 2023;57(5):2042–53.
9. Cao S, Xie M, Jia C, Zhang Y, Gong J, Wang B, et al. Household second-hand smoke exposure and stunted growth among Chinese school-age children. *Environ Technol Innov*. 2022;27:102521.
10. Organization WH. Suicide worldwide in 2019: global health estimates. 2021.
11. Muchlis N, Yusuf RA, Rusydi AR, Mahmud NU, Hikmah N, Qanitha A, et al. Cigarette smoke exposure and stunting among under-five children in rural and poor families in Indonesia. *Environ Health Insights*. 2023;17:11786302231185210.
12. Faul F, Erdfelder E, Buchner A, Lang AG. Statistical power analyses using G* Power 3.1: Tests for correlation and regression analyses. *Behav Res Methods*. 2009;41(4):1149–60.
13. Wake SK, Zewotir T, Lulu K, Fissuh YH. Longitudinal trends and determinants of stunting among children aged 1–15 years. *Archives of Public Health*. 2023;81(1):60.