Ethnic majority-minority disparities: Differential effects of exposure to secondhand smoke on child development*

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Abstract

This study empirically examines the effects of exposure to paternal smoking on child growth, utilizing comprehensive data from the Indonesian Family Life Survey. We use doubly robust estimations to compare the health outcomes of children whose fathers began smoking before pregnancy with children whose fathers abstained from smoking during the same time. We present separate point estimates for children from ethnic minorities and the ethnic majority group to analyze whether smoking is a channel through which the majority-minority gradient in health outcomes is transmitted from one generation to the next. Our findings reveal a significant reduction in the anthropometric height-for-age z-score among children from ethnic minority groups but not from the ethnic majority group, suggesting that smoking is an important conduit for these disparities. We present suggestive evidence that ethnic segregation contributes to the effect heterogeneity of secondhand smoke exposure between the ethnic majority and ethnic minorities. These findings underscore the importance of strengthening tobacco control regulations, such as increasing tobacco taxes and enforcing smoking bans, and point to the need to address ethnic segregation to mitigate the disproportionate impact of second-hand smoke exposure on ethnic minority children.

Keywords: *secondhand smoke, birth weight, height-for-age z-scores, ethnic disparities, development*

JEL Classification: *I14, J13, J15*

*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 article. The data that support the findings of this study are available from the corresponding author upon reasonable request.
We thank Alfredo R. Paloyo, Alina Sowa, and Ansgar Wübker for their constructive comments.

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1 Introduction

Studying health disparities across race and ethnicity has a long tradition (Berkman et al., 2014).[1] Differences in health behaviors and health status are frequently reported to be as varied as the number of ethnic groups within a geographic area. It is also often noted that ethnic majority groups experience significant advantages over minorities in these domains (e.g., Victora et al., 2010, Axelson et al., 2012). Moreover, empirical evidence suggests that the same health behavior is differently associated with health outcomes between the ethnic majority and ethnic minorities (e.g., Krueger and Chang, 2008, Pampel et al., 2010). For instance, tobacco smoking is not only more prevalent among ethnic minorities but also reported to be disproportionately associated with reduced health status in these groups (e.g., Haiman et al., 2006, Krueger et al., 2011).[2] One explanation is that economically disadvantaged ethnic minority groups may face greater economic barriers to accessing medical advice and treatment when suffering from a weakened immune and respiratory system caused by smoking-related inflammations in the airways. Further explanations that have been prominently proposed concern entitlements, individual preferences, and discrimination in connection with health care utilization (e.g., Bollini and Siem, 1995, Smaje and Le Grand, 1997, Morris et al., 2005, Hicken et al., 2018, Williams et al., 2019, Islam et al., 2021).[3]

In this paper, we assess whether and to what extent smoking serves as a conduit through which ethnic majority-minority disparities are transmitted from one generation to the next. We estimate the effects of secondhand smoke (SHS) on child development both for the aggregate sample and differentiated by the ethnic majority and minority groups. To achieve rigorous estimation results, we introduce an empirical approach designed to capture lasting impacts of in-utero exposure and apply it to estimating the differential effects on children from ethnic majority and minority groups. This effect heterogeneity has not been previously studied even though it might mask overall effects. For instance, this could occur if one group, which makes

up a substantial share of the study population–such as ethnic majority children–does not experience any effects of SHS exposure due to timely access to health interventions, thereby obscuring the effects experienced by another group–such as children from ethnic minority groups who lack the same access. We present this approach within the context of developing countries, where roughly eighty percent of the world's tobacco users live (World Health Organization, 2023). Considering that smoking rates among women are very low in most developing countries (World Health Organization, 2018a), we focus on a child's exposure to paternal tobacco smoke. It is, however, straightforward to follow the same analytical steps to estimate the effect of exposure to the mother's tobacco smoke.

SHS contains many toxic chemicals that are harmful to children. For instance, nitrogen dioxide is known to harm the mucous membranes of the airways. Carbon monoxide, even in small quantities, impairs oxygen intake, which is crucial for the development and functionality of all organs. Nicotine suppresses the immune system and acrolein causes inflammations (Sopori, 2002, Kawakita et al., 2008, Coneus and Spiess, 2012, Raghuveer et al., 2016). Importantly, SHS can even affect a child before birth, i.e., the fetus, via the mother's blood, as pollutants can pass through the placental barrier and thereby impair the development of fetal organs (e.g., Jauniaux and Burton, 2007). It can therefore result in various diseases in children and cause irreversible height deficits (e.g., Cogill, 2003, Coneus and Spiess, 2012). Especially for children and their mothers from ethnic minorities facing barriers to accessing medical treatment, these health risks can lead to pronounced disparities compared to families from the ethnic majority.

Our study considers the (lasting) effects of in-utero exposure by focusing on children of fathers who were initially non-smokers but started smoking three years before procreation. As a comparison group, we use children whose fathers abstained from smoking, i.e., remained non-smokers, during the same time frame. We use up to three years before birth as the reference

period as this time frame yields enough statistical power for our empirical analyses while limiting the period during which potentially confounding shocks might occur that could affect both the treatment status and the outcome of interest. To further increase our ability to create a meaningful counterfactual, we use parental, household, and geographic baseline information in doubly robust estimation that combines inverse probability weighting with regression adjustment in a difference-in-differences framework.

We use comprehensive data from the Indonesian Family Life Survey 2014/2015, covering over 3,000 children, to empirically analyze the effects of tobacco smoking initiation before procreation on child growth in Indonesia. We employ common indicators of child growth (e.g., Schott et al., 2013, Hoff et al., 1985) that are associated with morbidity in the long run (Cogill, 2003, Dewey and Begum, 2011, Victora et al., 2015, Gatica-Dominguez et al., 2020), specifically birth weight and the height-for-age z-score (HAZ). Details on the data can be found in Appendix A. We find relatively moderate differences in parental and household baseline characteristics between the treatment and control groups before applying the inverse probability weighting. After re-weighting, virtually no differences remain, i.e., balancedness is achieved. By considering both information before and after the treatment concerning the households of treated and non-treated children, we follow a similar approach as Ronchetti and Terriau (2021) who rely on a method that combines propensity score matching with the difference-in-differences estimator in their study of the role of electronic cigarettes for smoking cessation. As our paper aims to explore the ethnic majority-minority heterogeneity in the effects, we use an extension of our doubly-robust regression model, in which we compare the estimated effects of paternal smoking initiation among the ethnic majority group (Javanese) to those of the ethnic minorities. These separate estimates allow us to uncover relevant spillover effects on the next generation among ethnic minorities. A detailed explanation of our empirical approach, including step-by-step descriptions of each component and rationale, can be found in Appendix B.

Indonesia is an interesting setting to study this effect heterogeneity for several reasons (for a comprehensive background description, see Appendix C). First, Indonesia's male smoking rate is among the highest in the world (World Health Organization, 2018b) while female smoking is negligible. At the same time, there is a high prevalence of growth failure in children and a large ethnic majority group. This ethnic majority displays significantly higher child growth and lower smoking rates relative to ethnic minorities. Their advantage in child health outcomes is accompanied by a rate of health service consumption roughly the same as for ethnic minorities, which implies a higher conditional health-seeking propensity compared to ethnic minorities. Second, unlike differences in health outcomes and healthcare utilization, both groups are relatively similar concerning socioeconomic characteristics. It is, thus, rather unlikely that lower socioeconomic status and associated barriers to the treatment of inflammations and respiratory diseases, such as lack of entitlement (Sudano and Baker, 2006), explain the effect heterogeneity in our study context. In this case, our findings of substantial effect heterogeneity would point us to other commonly mentioned explanations for ethnic majority-minority heterogeneity. These include differences in individual preferences for health services as well as discrimination by health service providers and associated mistrust. Third, degrees of ethnic segregation vary substantially within the country. This variation allows us to explore whether the effect heterogeneity between children from the ethnic majority group and those from ethnic minorities may point towards institutional racism, discrimination, and associated medical mistrust as relevant channels.[4]

As our first and main contribution, we provide the first estimates of the differential effects of SHS exposure on child development across ethnic majority and minority groups. This adds more broadly to the still very limited understanding of what causes persistent health disadvantages of ethnic minority groups (Bhopal, 1997, Kawachi et al., 2005, Hicken et al., 2018). Several previous studies have investigated the short-term effects of SHS on the general child population, particularly focusing on health outcomes at birth (e.g., Evans and Ringel,

1999, Lien and Evans, 2005, Abrevaya, 2006, Abrevaya and Dahl, 2008, Wehby et al., 2011, Bharadwaj et al., 2014, Costi et al., 2024). The available empirical findings tend to suggest that maternal smoking during pregnancy increases the likelihood of infants being born with low birth weight. However, to the best of our knowledge, evidence examining heterogeneity across ethnic groups in the effects of SHS exposure, or other forms of air pollution, is currently missing in the context of both developing and developed countries in the literature.

Additionally, we provide evidence on the *long*-term effects of SHS exposure on indicators of chronic health issues, such as low HAZ, and focus on the context of developing countries. In contrast to that, most existing empirical studies concentrate either on health outcomes of newborns, and thereby short-term effects (for systematic reviews, see Simon, 2016, Nadhiroh et al., 2020), or the effects of smoking among the adult population (Moayeri et al., 2022, Pieroni et al., 2015). This existing evidence base is predominantly concentrated on developed countries (see e.g., Rosales-Rueda and Triyana, 2019).[5] A few studies estimate the correlation between paternal smoking and child growth in Indonesia. For instance, paternal smoking was found to be positively correlated with chronic child malnutrition (Barber et al., 2008). Also, a disproportionate number of non-smoking mothers exposed to SHS reported low-birth-weight children relative to non-smoking mothers without SHS inside the home (Andriani et al., 2023).

Moreover, our setting allows us to study the isolated effects of paternal smoking initiation, given the negligible share of women who smoke in Indonesia. The existing SHS literature has primarily focused on the effects of maternal smoking on child health (e.g., Evans and Ringel, 1999, Lien and Evans, 2005, Abrevaya, 2006, Abrevaya and Dahl, 2008, Costi et al., 2024, Wehby et al., 2011, Bharadwaj et al., 2014). We, therefore, contribute to a smaller literature that provides evidence on the effects of smoking stemming from both parents or solely the father (e.g., Klonoff-Cohen et al., 1995, Markowitz, 2008, Costi et al., 2024).

Finally, we make a methodological contribution that helps to better understand and incorporate

the lasting impacts of *in-utero* exposure by introducing an empirical strategy that captures these impacts. This is relevant because the lasting effects of in-utero exposure can, in theory, have important implications for the validity of the methods that are increasingly becoming the standard in the literature on the effects of SHS on child development, i.e., longitudinal studies relying on individual-level fixed effects (Nadhiroh et al., 2020). In particular, omitting past treatment, such as in-utero SHS exposure, from a regression model when historical effects are relevant, can lead to biased and inconsistent estimates (Nickel, 1981), which potentially explains the mixed evidence regarding the effects of SHS on child growth (Nadhiroh et al., 2020).

We find paternal smoking initiation to cause a significant reduction in HAZ among children from ethnic minorities. In contrast, no significant effects are observed among the ethnic majority group or the overall sample. This underscores the importance of considering the heterogeneous effects between the ethnic majority and minority groups. Contrary to previous evidence on the negative impacts of maternal smoking, we additionally find no significant effects of paternal smoking initiation on birth weight. This is plausible, as the women in our sample are primarily exposed to men's tobacco smoke and do not smoke themselves. The impact pathway from paternal smoking on birth outcomes is, thus, rather indirect. This may suggest that the threat of the dynamic omitted variable bias for the current standard method of estimating the effects of SHS may be lower than anticipated, particularly when focusing on paternal smoking in the context of developing countries. However, our results for the HAZ scores among ethnic-minority children align with in-utero exposure effects that accumulate over time in absolute terms. As these effects may unfold in a measurable way only later on during childhood, we recommend incorporating this aspect into future estimations.

Given that socioeconomic differences unlikely explain the observed ethnic majority-minority disparity in the effects in our study context, we explore the lower likelihood of ethnic minority

groups encountering ethnically concordant medical staff when they require care as an alternative channel. Ethnic concordance increases the acceptance and utilization of health care services (see e.g., Alsan and Wanamaker, 2017, Laksono et al., 2020). Employing a commonly used segregation measure (Mehra et al., 2017) to approximate the likelihood of ethnically concordant medical health service supply, we find that the heterogeneity in the effects of SHS exposure is driven by children living in highly segregated areas.

We conclude the paper providing brief guidance to policymakers aimed at mitigating the undesirable effects of secondhand smoke exposure among ethnic minority groups on child development. Additionally, we lay out relevant avenues for future research, including the need to validate and further rationalize the effect channel of ethnic segregation.

2 Results

In this section, we present estimates of the aggregate effects of SHS exposure and results for the effect heterogeneity between children belonging to the ethnic majority group and ethnic minorities, respectively.

2.1 Aggregate effects of SHS exposure

Table 1 presents the effects of paternal smoking initiation on birth weight (Panel A) and HAZ (Panel B) for the average child in the sample (Columns 1-4). While the first column presents the results from the OLS model after reweighting but without control variables, Columns 2-4 show the results when successively adding the different sets of control variables. The results in Panel A indicate a small negative coefficient of paternal smoking initiation before conception on the average child's body weight at birth (Columns 1 to 4). It is, however, far from any statistical significance. According to the results shown in Panel B, SHS exposure seems to negatively affect children's height, resulting in a reduction of around 0.14 standard deviations.

The negative point estimate is, however, again statistically insignificant (Column 4).

2.2 Effects of SHS exposure by ethnic majority/ minority groups

Table 2 presents the effects of SHS on children's birth weight (Panel A) and HAZ (Panel B), differentiated by ethnic majority group. Again, we observe no significant effects of paternal smoking on birth weight, irrespective of belonging to the majority or minority groups.

Concerning the HAZ measure, we observe substantial heterogeneity in the effects between the Javanese children and children from ethnic minorities. Specifically, the point estimates of paternal smoking are negative and statistically significant, indicating a large reduction in the HAZ among children from ethnic minorities. In quantitative terms, the effect of exposure to paternal smoking corresponds to roughly 20 percent $\left(-\frac{0.264}{1.34}\right)$ of the sample average of children from ethnic minorities or 19 percent $\left(-\frac{0.264}{1.34}\right)$ of a standard deviation. In contrast, the coefficient of the interaction term between the Javanese and paternal smoking initiation points to a positive effect on HAZ of similar magnitude that is statistically significant at the ten-percent level. The sum of the coefficients of FSS and the interaction term yields a statistically insignificant effect among Javanese children. This implies that, unlike children from ethnic minority groups, ethnic majority children do not experience a deterioration in the respective anthropometric health measures. Hence, SHS exposure only seems to adversely affect children from ethnic minorities.

In an additional exploratory step, we show that these heterogeneous effects are not driven by particular minorities. For this purpose, we analyze the effect heterogeneity across different ethnic minority groups that each make up at least five percent of our study sample. More precisely, we replace the *Javanese* dummy in regression model (3) (see Appendix B) for *Minority*, which indicates the respective ethnic minority group. The results are provided in

Table D.1 of the Appendix D.[6]

2.3 Discussion of findings

Our findings of a strong negative effect of paternal smoking initiation on the HAZ of children among ethnic minority groups are consistent with results reported by previous literature for the direct health effects of smoking in the black population in the US (Haiman et al., 2006, Krueger et al., 2011). However, relative to the black-white gap in the US, socioeconomic differences between the ethnic majority and ethnic minorities are less pronounced in Indonesia and, thus, unlikely to be the main channel for the ethnic majority-minority disparity in the effect of SHS exposure.

A more plausible explanation in the context of Indonesia refers to significant differences in healthcare utilization patterns between the ethnic majority group and ethnic minorities. Minority groups tend to be more likely to encounter non-ethnically concordant medical staff, which reduces the probability of utilizing health care services (see e.g., Alsan and Wanamaker, 2017, Laksono et al., 2020). This occurs when ethnic minority group members perceive non-ethnically concordant providers as medically less trustworthy, possibly restricting service utilization to acute cases only (Alsan and Wanamaker, 2017, Kinlock et al., 2017).[7]

To shed light on this channel, we analyze whether the observed ethnic majority-minority variation in the effects on HAZ is stronger, in absolute terms, in areas with a very low concentration of the child's ethnic group. In these areas, the likelihood of encountering ethnically concordant providers is arguably lower. For this purpose, we proxy segregation levels with binary variables that indicate different minimum district-level shares of individuals who belong to the child's ethnic group (following Mehra et al., 2017). These shares include below 10 percent, 20 percent, 30 percent, and 40 percent (which is roughly the same population

fraction at the district level as the ethnic majority share at the country level), respectively. For each dummy variable corresponding to a minimum district share of the child's ethnic group, Equation (2) (see Appendix B) is then augmented by this segregation proxy as well as an interaction term of this variable and paternal smoking initiation.

The results are displayed in Table 3. The respective point estimate is statistically significant only when using the lowest threshold of the district-level population share of the same ethnic group, i.e., 10 percent (Column 1). The estimated treatment effect on its own becomes statistically insignificant after including the segregation proxy and its interaction with paternal smoking initiation. All in all, this suggests that the negative effect of paternal smoking initiation is driven by areas with a very low population share of the child's ethnic group. However, the small number of ethnic minority children from these areas in our sample implies that this finding should be further investigated in future research.[8]

Even though we do not find any significant effects of paternal smoking on birth weight overall and by ethnic majority/ minority groups, we present the results for this outcome by ethnic segregation as well. Table D.2 in the Appendix shows that the null effects remain intact even when accounting for different levels of segregation and the potential interaction effects with paternal smoking initiation. This solidifies the robustness of the null effects further (e.g., Salm and Wübker, 2023).

3 Conclusion

In this study, we aim to shed light on whether tobacco smoking is a channel through which the majority-minority gradient in health outcomes is transmitted from one generation to the next in a developing country context. For this purpose, we examine the effects of exposure to paternal smoking on various anthropometric child development measures, using

comprehensive data from the IFLS. We introduce a doubly robust estimation approach for the SHS effects on child growth that combines inverse probability weighting and multivariate regression techniques. Importantly, we compare the growth of children aged up to nine years whose fathers initiated smoking within three years before their birth, rather than any time after, to that of children whose fathers remained abstinent during the same period.

We observe strong effect heterogeneity in the effects of paternal smoking initiation on HAZ of children. While children from the ethnic majority group do not seem to be significantly affected by SHS exposure, we observe a large reduction in the HAZ due to paternal smoking among children from ethnic minorities. Our aggregate results yield an insignificant average effect. This points to the importance of going beyond the estimation of mean effects of health-related behavior when interested in uncovering potential causes of persistent ethnic majority-minority health disparities.

Moreover, we find all estimates of the effects on birth weight to be statistically insignificant, including when differentiating between ethnic majority and minority groups. This finding tends to be consistent with the plausibility argument of the impact pathway from paternal smoking on pregnancy outcomes being too indirect to cause substantial adverse effects.[9]

Regarding potential explanations of our results concerning the HAZ outcome, there is very limited descriptive support that they are driven by a socioeconomic ethnic majority-minority gradient in Indonesia. In a further exploratory analysis, we shed light on the relevance of ethnic segregation for our results, which has been used to explain ethnic health disparities in the direct associations between smoking and health. In particular, in areas with a very low population density of their own ethnicity, children and their parents may be more likely to encounter institutional racism, discrimination, and a shortage of ethnically concordant health service providers. Our estimates of the effects of paternal smoking are significantly more pronounced, in absolute terms, in districts with a very low population share of the child's ethnic group.

Additionally, the negative impact of SHS on minority children turns statistically insignificant in areas with a very low share of the child's ethnicity. However, the results from this exploratory analysis need to be validated by future research due to the relatively small sample size of minority children in these areas. We consider assessing the magnitude of effect dynamics of in-utero exposure and the underlying channels of the effects of SHS exposure among children from ethnic minorities a promising avenue for future research.

Our findings of paternal smoking behavior contributing to the health gradient between ethnic majority and minority groups are important from a policy perspective. They underscore both the general relevance of employing policies to protect vulnerable population groups, especially children, from SHS exposure as well as the necessity to consider, and potentially target, specific ethnic groups among which negative health effects are most pronounced.

Policymakers aiming to reduce second-hand smoke exposure among ethnic minorities may consider multiple layers of action across three key domains. First, classical policy tools such as tax and price increases, strongly enforced smoking bans, and anti-smoking campaigns have consistently been among the most effective strategies for reducing smoking prevalence (e.g., Akter et al., 2024; De Cicca et al., 2022). In contrast, changes to insurance policies, such as surcharges for smokers, have shown limited promise and may inadvertently exacerbate inequities (Friedman et al., 2016; Ku et al., 2016; Jarlenski et al., 2014). Second, given the insignificant differences in socioeconomic and insurance coverage between ethnic majority and minority groups, the focus of action should arguably lie on the removal of healthcare barriers unrelated to costs. Existing evidence on the importance of ethnic concordance between patients and providers and our own suggestive evidence of a high relevance of ethnic segregation underscores the potential of policies aimed at increasing ethnic minority representation among medical staff (e.g., Alsan and Eichmeyer, 2024). Additional strategies include providing cultural competency training and implementing community health outreach programs and

awareness campaigns to foster trust and engagement (e.g., Handtke et al., 2019). Finally, there is a pressing need for rigorous research to better understand the causes of the ethnic majority-minority divide in the effects of second-hand smoke exposure. Developing effective programs will hinge on a deeper, evidence-based understanding of these disparities, enabling the design of tailored and optimal interventions.

List of Tables (Main Text)

Table 1 – Effect of SHS exposure on main outcomes

	(1)	(2)	(3)	(4)
Panel A - Outcome: Birth weight				
Father started smoking	-0.017	-0.017	-0.021	-0.029
	(0.054)	(0.052)	(0.052)	(0.051)
Observations total	1566	1566	1566	1566
Observations treated	192	192	192	192
Panel B - Outcome: Height-for-age z-score				
Father started smoking	-0.091	-0.115	-0.124	-0.140
	(0.103)	(0.096)	(0.094)	(0.091)
Observations total	2606	2605	2605	2605
Observations treated	362	362	362	362
IPW variables	No	Yes	Yes	Yes
Family member controls	No	No	Yes	Yes
Child controls	No	No	No	Yes

Notes: Birth weight reported by the child's mother in kilograms. Height-for-age z-score relates the child's height (or length for children under two) to the height of a comparable reference population of the same sex and age. Father started smoking is equal to 1 if a father started smoking zero to three years before the child's birth and 0 if the father remained a non-smoker during the same period. Inverse probability weight (IPW) variables include father's log. income, father's age at birth, urban residence, Java, Kilmatan, Lesser Sunda Islands, and Sulawesi. Family member controls consist of female household head, household size, father Muslim, and mother having at least secondary education. Child controls include age in years, and female. Further details on the variables are presented in Appendix A. Standard errors are clustered at the father-level and shown in parentheses. Significance levels show $*p < 0.10$, $**$ $p < 0.05$, $***$ $p < 0.01$.
Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Table 2 – Effect of SHS exposure on main outcomes by ethnicity

	(1)	(2)	(3)	(4)
Panel A - Outcome: Birth weight				
Father started smoking	-0.005	0.005	-0.007	-0.014
	(0.074)	(0.069)	(0.069)	(0.068)
Javanese*Father started smoking	-0.026	-0.041	-0.020	-0.020
	(0.102)	(0.099)	(0.100)	(0.101)
Javanese	-0.022	-0.070	-0.087	-0.096
	(0.073)	(0.067)	(0.068)	(0.067)
Observations total	1566	1566	1566	1566
Observations treated	192	192	192	192
Panel B - Outcome: Height-for-age z-score				
Father started smoking	-0.192	-0.234*	-0.257**	-0.264**
	(0.142)	(0.123)	(0.122)	(0.121)
Javanese*Father started smoking	0.241	0.307*	0.350*	0.330*
	(0.199)	(0.179)	(0.180)	(0.179)
Javanese	0.096	-0.089	-0.164	-0.170
	(0.154)	(0.165)	(0.163)	(0.164)
Observations total	2606	2605	2605	2605
Observations treated	362	362	362	362
IPW variables	No	Yes	Yes	Yes
Family member controls	No	No	Yes	Yes
Child controls	No	No	No	Yes

Notes: Birth weight reported by the child's mother in kilograms. Height-for-age z-score relates the child's height (or length for children under two) to the height of a comparable reference population of the same sex and age. Father started smoking is equal to 1 if a father started smoking zero to three years before the child's birth and 0 if the father remained a non-smoker during the same period. Inverse probability weight (IPW) variables include father's log. income, father's age at birth, urban residence, Java, Kilmatan, Lesser Sunda Islands, and Sulawesi. Family member controls consist of female household head, household size, father Muslim, and mother having at least secondary education. Child controls include age in years, and female. Further details on the variables are presented in Appendix A. Standard errors are clustered at the father-level and shown in parentheses. Significance levels show $*p < 0.10$, $**p < 0.05$, $***p < 0.01$

Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Table 3 – Effects of SHS exposure on height-for-age z-scores by share of child's ethnicity within district

	below 10 p.	below 20 p.	below 30 p.	below 40 p.
	(1)	(2)	(3)	(4)
Father started smoking	-0.091	-0.112	-0.110	-0.098
	(0.092)	(0.095)	(0.099)	(0.101)
Segregation	0.672*	0.204	0.075	-0.045
	(0.361)	(0.209)	(0.160)	(0.152)
Segregation*Father started smoking	-1.171**	-0.317	-0.214	-0.210
	(0.455)	(0.308)	(0.246)	(0.230)
IPW variables	Yes	Yes	Yes	Yes
Family member controls	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Observations total	2605	2605	2605	2605
Observations treated	362	362	362	362

Notes: Height-for-age z-score relates the child's height (or length for children under two) to the height of a comparable reference population of the same sex and age. Father started smoking is equal to 1 if a father started smoking zero to three years before the child's birth and 0 if the father remained a non-smoker during the same period. Inverse probability weight (IPW) variables include father's log. income, father's age at birth, urban residence, Java, Kilmatan, Lesser Sunda Islands, and Sulawesi. Family member controls consist of female household head, household size, father Muslim, and mother having at least secondary education. Child controls include age in years, and female. "Below X p." indicates the district share of a child being below X percent. Further details on the variables are presented in Appendix B. Standard errors are clustered at the father-level and shown in parentheses. Significance levels show * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$
Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Appendix A – Description of the Data and Main Variables

This study uses data from the Indonesian Family Life Survey (IFLS), a continuing socioeconomic and health panel survey. It was first collected in 1993 and represents approximately 83 percent of the Indonesian population, living in 13 of the 27 provinces in the country.[10] For our study, we use IFLS 5, the most recently available survey wave from 2014 and 2015 (Strauss et al., 2016), which constitutes the sole survey year that reports the ethnicity of the respondents. Besides ethnicity, the dataset provides information on self-reported individual- and household-level socioeconomic characteristics, smoking behavior (e.g., smoking status, age started/ quit smoking, number of cigarettes smoked per day), health outcomes (e.g., birth weight as reported by the mother), as well as anthropometric measures (e.g., height).

To study the effects of SHS on child health, following the definition of the WHO, we define subjects aged zero to nine as children (Singh et al., 2019). This allows us to rule out the influence of own smoking behavior, which may mask the effects of the exposure to SHS among older non-adult subjects given the relatively high smoking prevalence among them. With only one percent of smokers reporting having started smoking below the age of ten in the IFLS 5 data, it is very unlikely that children in our sample already started smoking themselves.

Health outcomes

We use two main outcome variables in our study. First, we employ the birth weight reported by the child's mother in kilograms to capture the short-term effects of SHS exposure during pregnancy. Second, we use the child's measured height to study the long-term effects of SHS exposure. For each child, we calculate the HAZ, which is a continuous variable with a distribution close to normal and relates the child's height (or length for children under two) to the height of a comparable reference population of the same sex and age. It can thereby indicate

past growth failure (e.g., due to frequent infections, Cogill, 2003).[11] We obtain the z-scores using the WHO reference standard for the growth of children below the age of five (World Health Organization and Tobacco Free Initiative, 2007). For children aged six to nine, we use the WHO growth reference for school-aged children and adolescents (World Health Organization, 2009). To exclude biologically implausible values, we exclude children with a HAZ outside the range of minus six to plus six standard deviations as recommended by the World Health Organization (2007, 2009).

Secondhand smoke exposure

The IFLS does not provide a direct measure of SHS exposure. We, therefore, use information on the father's smoking history as a proxy for SHS exposure in the home.[12] The treatment variable indicates a substantially increased probability of being exposed to SHS but not actual SHS exposure.[13]

Descriptive statistics by ethnic majority/ minority groups

In Table D.3 of the Appendix, we display descriptive statistics of the ethnic majority population and ethnic minorities. We observe smoking rates of roughly seventy percent, which are higher among ethnic minority men. While we observe similar means in birth weight, the ethnic majority group has a larger average HAZ. In terms of socioeconomic household characteristics, we do not observe a clear pattern of ethnic majority-minorities disparity in our data. For instance, while rates of asset ownership and employment are higher among the ethnic majority group, the annual income (median and mean) is, on average, higher among households belonging to ethnic minorities. In Table D.4 of the Appendix, we additionally provide similar descriptive statistics of our estimation sample.

Appendix B - Technical Details on Estimation Strategy

To estimate the effect of SHS, we would ideally compare the outcome Y_1 of a child with exposure to tobacco smoke of the father ($D = 1$) with the outcome Y_0 of the same child without respective exposure ($D = 0$) at the same time. The difference between these potential outcomes, i.e., $Y_1 - Y_0$, would then represent the causal point estimate. However, since we can only observe either children with or without SHS exposure, information on one of those two outcomes is missing in reality. One solution to this missing information problem is comparing different children with some being exposed to SHS and others not. However, since smoking is a choice variable of the father, the two groups of children likely differ in many ways from each other. For example, if the father's smoking status is related to his income and so is the dietary provision to his children, it is difficult to attribute the difference in the outcomes between these groups to SHS. In this example, the father's income represents a confounding variable.

Difference-in-differences

One common solution to reduce selection bias represents the difference-in-differences approach. In our case, this implies comparing the change in the actually observed outcome variables between children who were all not exposed to paternal smoking in some initial state t, one group of these children experiencing exposure at a later period $t+1$ while the other group does not. The estimator is then provided by $\left(Y_{i,t+1}^{1} - Y_{i,t}^{1}\right) - \left(Y_{i,t+1}^{0} - Y_{i,t}^{0}\right)$. This approach reduces the sources of potential bias to changes in the confounding variables over time. Aimed at covering the exposure to SHS during pregnancy, which is essential for a child's development and long-term health (e.g., Alati et al., 2006), we define t as the stage of conception. Assuming the baseline outcome at conception to be negligible, the difference-in-differences estimator simplifies to $\left(Y_{i,t+1}^{1} - 0\right) - \left(Y_{i,t+1}^{0} - 0\right) = Y_{i,t+1}^{1} - Y_{i,t+1}^{0}$.

To implement this difference-in-differences approach, we define the treatment variable *father*

started smoking, abbreviated by *FSS*, using the father's self-reported smoking status as well as the age at which he reports having started smoking. The variable *FSS* takes on the value one if the father reports having started to smoke between zero to three years before birth, i.e., before the initial stage. Hence, the event of smoking initiation occurs during a period that covers the time of conception. The variable is equal to zero, in contrast, if a child's father did not start smoking during the three years before birth. We restrict our sample to children whose fathers were non-smokers three years before birth and exclude children from the sample whose fathers started smoking but then stopped during this time frame. We use a reference period of three years before birth as this best balances the trade-off between a large enough sample to ensure statistical power for our analyses as well as the smallest possible time frame during which potentially confounding shocks might occur that could both affect the treatment status and the outcome of interest.

Even though restricting the sample to children with non-smoking fathers at $t - 1$ improves comparability across groups, differences between children with fathers who started smoking in the three years before the child's birth (treatment group) and children of non-smoking fathers who did not start during that period (control group) are still present (see Table D.5). These differences might grow over time and influence a child's health, potentially biasing our estimation results.

Inverse probability weighting

Aimed at removing these remaining differences, we combine our difference-in-differences approach with inverse probability weighting. The inverse probability weighting approach creates a pseudo-population in which the treatment does not depend on the measured potential confounders. It thereby mimics randomized treatment assignment to minimize the bias that selection into treatment might cause. This is possible by assigning weights to children in the treated and control group based on the inverse of the estimated probability of treatment

(Rosenbaum and Rubin, 1983) – here the children's fathers' smoking initiation. While we cannot observe the actual propensity scores, we can proxy them by predicting each child's probability of being treated based on a set of potential confounders. We can then take the difference between the weighted averages of the difference in the outcomes for both children with paternal smoking initiation ($FSS = 1$) and children without paternal smoking initiation ($FSS = 0$). The propensity score, $p(X)$, is a function of all variables that simultaneously affect FSS and changes in Y. The inverse probability weighting estimator relies on the assumption that the assignment of treatment is unconfounded, conditional on the propensity score, $i.e., Y1, Y0 \perp FSS \vee p(X)$. This assumption requires observing and incorporating all variables that influence the probability of receiving treatment when estimating the propensity scores. We then use the propensity scores to individually re-weight children from fathers who did not start smoking during the three years before birth such that they jointly become similar to children from fathers who initiated smoking during the same period. Given the unconfoundedness assumption, this difference then yields the true causal effect of fathers' smoking initiation on the child's development.

Following Brunell and Dinardo (2004), we set the weights of the treated children $w^{FSS=1}$ equal to one. We compute the weights of children from the control group $w^{FSS=0}$with the following formula:

$$w_i^{FSS=0} = \frac{P(FSS=1|X_h)}{1-P(FSS=1|X_h)} \cdot \frac{p^{FSS=0}}{p^{FSS=1}} \quad (1)$$

where P is the probability of having a father who started smoking in the three years before birth, $p^{FSS=1}$ and $p^{FSS=0}$ are the fractions of children in the treatment and control group, respectively. The vector X_h includes different household and household member covariates, including control variables concerning the father that are unaffected by the treatment itself but are largely imbalanced at baseline (see Table D.5). Specifically, the vector includes several geographic

variables, namely an urban dummy, as well as region dummies for Java, Kalimantan, Lesser Sunda Islands, Sulawesi, and Sumatra. These geographic variables likely affect SHS exposure because smoking patterns differ between regions and smoking is more common in rural than in urban areas (Rokx et al., 2018). Moreover, the father's log. income is included in Xh as a proxy for the socioeconomic status of the family, which likely affects SHS exposure because poorer and less educated individuals are more likely to smoke (e.g., Raghuveer et al., 2016). Finally, the father's age at birth substantially differs between treated and control children and is therefore included on its own and in squared form. The propensity scores are estimated using the *teffects aipw* command in Stata version 18.

Covariate balance of the sample before and after reweighting

Table D.5 displays the means and standard deviations of the variables that enter the propensity score estimations (upper panel) and the linear regression (lower panel) for the treatment (Column 1) as well as the control group. The values of the latter group are shown both before (Column 4) and after applying the inverse probability weights (Column 7). As to the former variables, we observe statistically significant differences without the weights (Column 10) that fully disappear after reweighting (Column 11). The same general pattern is observed for the latter variables, with only the child's age in years and the presence of a female household head remaining unbalanced in the weighted comparisons of the two groups.

In the weighted sample, roughly 50 percent live in urban areas and 40 percent are from the Java region. The average age of the father at the child's birth and that of the child at the interview are roughly 25 years and five years, respectively. Households consist of, on average, five members, and almost all (around 94 percent) are headed by a male person. The share of fathers with secondary education amounts to 82 percent and exceeds that of the average mother by around three percentage points in the weighted sample.

A prominent variable that remains unbalanced after reweighting is the age of the child. We, however, do not include it when calculating the propensity weights as this imbalance unlikely introduces a bias when estimating the relationship between the father's smoking initiation and the child's physical development. In particular, our main outcome variables are by definition independent of the child's age at the time of the interview. The information on birth weight refers to the same period, irrespective of the child's age later on. The HAZ is a standardized measure that relates the child's height at the time of the interview to the median height and standard deviation of the height of the reference population (same sex and age group). In addition, including the child's age as a control variable in our empirical specification does not change the results (see last column of Tables 1 and 2 of the main text). This is reassuring that the imbalance in children's age is unlikely a threat to our analyses.

As an additional measure of covariate balance, we use the standardized bias as suggested by Rosenbaum and Rubin (1985) that quantifies the difference between the mean of the treated and the reweighted control group for each variable as a percentage of the square root of the average variance in both groups. Results for this measure are displayed in Figures D.1 and D.2 of the Appendix E.[14] The balance substantially improves for all covariates that were used in the inverse probability weighting and for most of the other covariates. More precisely, the standardized bias reduced from initially up to 30 percent (urban) - or even 150 percent (father's age at childbirth, see Figure E.2) to less than twenty percent after reweighting. This bolsters confidence in the ability of the inverse probability weighting to make children with and without paternal smoke exposure more comparable.

Doubly-robust estimation

To further reduce the threat of estimation bias due to selection into treatment, Robins and Rotnitzky (1995) propose combining inverse probability weighting with regression adjustment, which consists of incorporating both the treatment indicator as well as factors that might affect

the outcome as covariates in the regression analysis (see also Hirano and Imbens, 2001). This combination is often referred to as doubly-robust estimation (Funk et al., 2011) and followed in the present paper. Employing the weights from Equation (1), we run weighted least squares estimation of the effects of SHS exposure on child development outcomes with the following regression model:

$$Y_i = \alpha + \beta \cdot FSS_i + \gamma' Z_h + \delta' X_i + \varepsilon_i \quad (2)$$

in which Y_i is the respective outcome variable, i.e., birth weight or HAZ, of child i in household h. *FSS* is attached to the coefficient of interest that estimates the effect of being exposed to SHS due to the father's smoking initiation zero to three years before the child's birth. We provide the results of four different specifications that successively add sets of control variables. The vector Z_h includes different covariates for the household and its members. The vector X_i comprises variables concerning the child.

In the first specification, vectors Z_h and X_i are both empty. In specification two, the subset X_h of the vector Z_h is added, i.e., the inverse probability weighting variables (urban, log. father's income, father's age at birth, and father's age at birth squared, as well as the regional dummy variables). The third specification includes the entire vector Z_h. In this specification, we control for additional family characteristics, including the household size as well as dummies for a female household head, a Muslim father, and a mother having completed secondary education. In the fourth and final specification, we add vector X_i with controls for the child's age at the interview and a dummy variable indicating whether the child is a girl. Standard errors are clustered at the level of treatment, i.e. at the father's level.[15] The number of observations is 2606 (1566) children for the height-for-age z-score (birth weight) outcome, of which 362 (192) were treated.

The key objective of our paper is to explore the heterogeneity in the effects of exposure to

paternal smoking on anthropometric health outcomes by children's ethnicity. More precisely, we differentiate between belonging to the ethnic majority group, Javanese, and ethnic minority groups.[16] For this purpose, we expand the weighted least squares regression model that we introduced above in the following way:

$$Y_i = \alpha + \beta_1 \cdot FSS_i + \beta_2 \cdot Javanese \cdot FSS_i + \beta_3 \cdot Javanese + \gamma' Z_h + \delta' X_i + \varepsilon_i \quad (3)$$

in which *Javanese* indicates that the child belongs to the ethnic majority group. The coefficient β_2, which is connected to the interaction term of *Javanese* and *FSS*, captures the differences in the effects of SHS exposure between children belonging to the ethnic majority group and minority groups. All other components of the regression model remain as described before. The number of observations is 2606 (1566) children for the height-for-age z-score (birth weight) outcome, of which 362 (192) were treated.

Appendix C – Background Information on Indonesia

Indonesia is a lower middle-income country with around 264 million inhabitants (World Health Organization, 2018b). It is home to over 1000 ethnic groups (Suryadinata et al., 2003), the largest one being the Javanese which account for approximately 40 percent of the population.

Among the Indonesian population, tobacco consumption, and, in particular, cigarette smoking, is very popular. Roughly 40 percent of the population aged 15 years and older reported smoking tobacco in the 2016 Global Adult Tobacco Survey (World Health Organization, 2018b). Most Indonesian smokers report daily cigarette smoking (World Health Organization, 2018b). While tobacco smoking is highly accepted among men and associated with masculinity in the Indonesian culture (Ayuningtyas et al., 2021), it is still often considered inappropriate for Indonesian women (Ng et al., 2006). Consistently, only 2.6 percent of all Indonesian women reported smoking tobacco in 2016, with a decreasing trend since 2000. In contrast, the smoking prevalence of male Indonesians has continuously increased from 59 percent in 2000 to 76 percent in 2016 and has become the highest rate worldwide (World Health Organization, 2018b).

There is very limited literature concerning differences in smoking patterns across ethnic groups available in Indonesia. Martini and Sulistyowati (2005) report that, among male students in the East Java province, 18 percent, 20 percent, and 21 percent of those identifying as Pandulung, Javanese, and Madurese are smokers, respectively. Effendi et al. (2021) show that among Indonesian men, Madurese identity was positively associated with tobacco consumption. Non-communicable diseases, for which active smoking and SHS exposure are important risk factors, have become a major public health problem in Indonesia.

The five currently leading causes of death in Indonesia are all tobacco-related and include ischemic heart disease, cerebrovascular disease, tuberculosis, diabetes, and chronic respiratory

diseases (World Health Organization, 2018b). When it comes to chronic malnutrition and past growth failure, Indonesia is among the five countries with the highest number of cases worldwide. It is a severe problem in nearly all Indonesian provinces and for all income levels despite Indonesia's thriving economy and decline in poverty. Ethnicity is significantly associated with food consumption patterns in Indonesia. Specifically, Javanese children score high in dietary diversity relative to ethnic minorities, particularly in comparison to the Minangkabau. The same ethnic group also displays the most favorable HAZ among children (Kunto and Bras, 2019). Ethnicity continues to predict anthropometric measures among elderly Indonesian women (Hartono, 2018, Jain and Ma, 2020).[17] Despite the high smoking prevalence and the associated threats including non- communicable diseases, the Indonesian government has to date not signed the World Health Organization's (WHO) Framework Convention on Tobacco Control (World Health Organization and Tobacco Free Initiative, 2007, World Health Organization, 2021) and has implemented very limited policy measures for tobacco control. For instance, while cigarette excise taxes have successively been increased to up to 59 percent of the retail price, the affordability of cigarettes has not decreased and instead stabilized over time (Barber et al., 2008). Additionally, smoking bans for healthcare and educational facilities and public transport, which were introduced in 2018, lack enforcement (e.g., Wahyuti et al., 2019).

Appendix D - Tables

Table D.1 – Effect of SHS exposure on height-for-age z-scores by minority ethnicities

	(1)	(2)	(3)	(4)
Panel A - Sundanese (8%)				
Father started smoking	-0.103	-0.107	-0.119	-0.133
	(0.104)	(0.096)	(0.095)	(0.093)
Minority*Father started smoking	0.129	-0.084	-0.055	-0.074
	(0.418)	(0.425)	(0.399)	(0.410)
Minority	0.298	0.295	0.269	0.287
	(0.329)	(0.357)	(0.328)	(0.335)
Observations minority (treated)	34	34	34	34
Observations minority (control)	186	186	186	186
Panel B - Balinese (6%)				
Father started smoking	-0.092	-0.109	-0.106	-0.123
	(0.105)	(0.099)	(0.096)	(0.094)
Minority*Father started smoking	-0.008	-0.077	-0.219	-0.194
	(0.463)	(0.373)	(0.346)	(0.331)
Minority	-0.214	0.178	0.571	0.583
	(0.424)	(0.370)	(0.414)	(0.402)
Observations minority (treated)	22	22	22	22
Observations minority (control)	137	137	137	137
Panel C - Minang (6%)				
Father started smoking	-0.099	-0.126	-0.137	-0.150
	(0.106)	(0.099)	(0.097)	(0.094)
Minority*Father started smoking	0.308	0.260	0.286	0.242
	(0.462)	(0.462)	(0.450)	(0.458)
Minority	0.226	-0.006	-0.029	-0.002
	(0.242)	(0.257)	(0.255)	(0.254)
Observations minority (treated)	15	15	15	15
Observations minority (control)	147	147	147	147
Panel D - Banjar (5%)				
Father started smoking	-0.096	-0.124	-0.139	-0.157*
	(0.107)	(0.099)	(0.098)	(0.094)
Minority*Father started smoking	0.071	0.178	0.291	0.329
	(0.381)	(0.372)	(0.389)	(0.390)
Minority	-0.369	0.123	0.071	0.092
	(0.315)	(0.424)	(0.434)	(0.444)
Observations minority (treated)	18	18	18	18
Observations minority (control)	110	110	110	110
Panel E - Betawi (5%)				
Father started smoking	-0.098	-0.124	-0.133	-0.148
	(0.106)	(0.098)	(0.095)	(0.092)
Minority*Father started smoking	0.160	0.150	0.124	0.104
	(0.412)	(0.405)	(0.406)	(0.393)
Minority	-0.133	-0.553*	-0.498*	-0.503*
	(0.311)	(0.311)	(0.296)	(0.281)
Observations minority (treated)	15	15	15	15
Observations minority (control)	118	118	118	118

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Table D.1 - (Continued from previous page)

	(1)	(2)	(3)	(4)
Panel F - Sasak (5%)				
Father started smoking	-0.053	-0.081	-0.090	-0.106
	(0.105)	(0.097)	(0.095)	(0.092)
Minority*Father started smoking	-0.905*	-0.847	-0.820	-0.823
	(0.503)	(0.516)	(0.519)	(0.522)
Minority	-0.048	0.141	-0.031	0.005
	(0.348)	(0.400)	(0.401)	(0.402)
Observations minority (treated)	15	15	15	15
Observations minority (control)	108	108	108	108
Panel G - Other minority groups (21%)				
Father started smoking	-0.021	-0.038	-0.032	-0.055
	(0.117)	(0.115)	(0.111)	(0.105)
Other minorities*Father started smoking	-0.267	-0.280	-0.339	-0.309
	(0.239)	(0.218)	(0.214)	(0.208)
Other minorities	-0.113	0.036	0.126	0.112
	(0.180)	(0.162)	(0.148)	(0.142)
Observations minority (treated)	98	98	98	98
Observations minority (control)	466	466	466	466
Panel H - Javanese & special minority groups (Banjar, Betawi, Minang)				
Father started smoking	-0.257	-0.332**	-0.375***	-0.377***
	(0.171)	(0.145)	(0.143)	(0.142)
Javanese*Father started smoking	0.307	0.409**	0.472**	0.447**
	(0.220)	(0.194)	(0.195)	(0.194)
Javanese	0.086	-0.181	-0.278	-0.280
	(0.174)	(0.184)	(0.183)	(0.185)
Special min. groups*Father started smoking	0.289	0.383	0.454*	0.432
	(0.293)	(0.277)	(0.275)	(0.277)
Special min. groups	-0.036	-0.325	-0.381*	-0.371*
	(0.212)	(0.219)	(0.216)	(0.216)
Observations Javanese (treated)	145	145	145	145
Observations Javanese (control)	977	977	977	977
Observations special minorities (treated)	48	48	48	48
Observations special minorities (control)	375	375	375	375
IPW variables	No	Yes	Yes	Yes
Family member controls	No	No	Yes	Yes
Child controls	No	No	No	Yes
Observations (total)	2605	2605	2605	2605
Observations (treated)	362	362	362	362

Notes: Height-for-age z-score relates the child's height (or length for children under two) to the height of a comparable reference population of the same sex and age. Father started smoking is equal to 1 if a father started smoking zero to three years before the child's birth and 0 if the father remained a non-smoker during the same period. Inverse probability weight (IPW) variables include father's log. income, father's age at birth, urban residence, Java, Kilmatan, Lesser Sunda Islands, and Sulawesi. Family member controls consist of female household head, household size, father Muslim, and mother having at least secondary education. Child controls include age in years, and female. The variable" Minority" represents the minority group that is mentioned in the title of the respective panel. "Other minority groups" (Panel I) includes all minority groups with a share of less than 4% of the sample. "Special minority groups" (Panel H) refers to the groups Banjar, Betani, and Minang. Further details on the variables are presented in Appendix A. Standard errors are clustered at the father-level and shown in parentheses. Significance levels show $*p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Table D.2 – Effects of SHS exposure on birth weight by share of child's ethnicity within district

	below 10 p.	below 20 p.	below 30 p.	below 40 p.
	(1)	(2)	(3)	(4)
Father started smoking	-0.020	-0.030	-0.039	-0.015
	(0.052)	(0.054)	(0.057)	(0.060)
Segregation	0.299*	0.103	0.045	0.085
	(0.167)	(0.117)	(0.085)	(0.076)
Segregation*Father started smoking	-0.195	-0.007	0.051	-0.067
	(0.227)	(0.153)	(0.120)	(0.110)
IPW variables	Yes	Yes	Yes	Yes
Family member controls	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Observations total	1566	1566	1566	1566
Observations treated	192	192	192	192

Notes: Birth weight reported by the child's mother in kilograms. Father started smoking is equal to 1 if a father started smoking zero to three years before the child's birth and 0 if the father remained a non-smoker during the same period. Inverse probability weight (IPW) variables include father's log. income, father's age at birth, urban residence, Java, Kilmatan, Lesser Sunda Islands, and Sulawesi. Family member controls consist of female household head, household size, father Muslim, and mother having at least secondary education. Child controls include age in years, and female. "Below X p." indicates the district share of a child being below X percent. Further details on the variables are presented in Appendix A. Standard errors are clustered at the father-level and shown in parentheses. Significance levels show * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.
Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Table D.3 – Descriptive statistics by ethnic groups (all observations from IFLS 2014/2015)

	Javanese		Minority Groups					
			All		Special		Others	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Outcome:								
Birth weight of child (in kg)	3.14	(0.46)	3.15	(0.48)	3.14	(0.46)	3.16	(0.49)
Height for age z-score	-1.29	(1.17)	-1.48	(1.18)	-1.35	(1.17)	-1.52	(1.19)
Parental smoking patterns:								
Ever smoked cigarette	0.34	(0.47)	0.36	(0.48)	0.36	(0.48)	0.36	(0.48)
Ever smoked cigarette (women)	0.02	(0.12)	0.03	(0.18)	0.04	(0.20)	0.03	(0.17)
Ever smoked cigarette (men)	0.69	(0.46)	0.72	(0.45)	0.71	(0.45)	0.72	(0.45)
Age started smoking	19.46	(5.95)	19.43	(6.12)	19.11	(5.79)	19.52	(6.20)
Cigarettes smoked per day (smokers)	11.02	(6.94)	12.43	(7.73)	13.37	(7.83)	12.17	(7.68)
Cigarettes smoked per day (smokers + nonsmokers)	2.00	(5.17)	2.18	(5.73)	2.33	(6.03)	2.14	(5.64)
Education:								
Any secondary education	0.52	(0.50)	0.52	(0.50)	0.54	(0.50)	0.51	(0.50)
Completed secondary education	0.24	(0.43)	0.28	(0.45)	0.29	(0.45)	0.27	(0.45)
Years of education	6.87	(4.90)	6.94	(5.14)	7.03	(5.17)	6.91	(5.13)
Completed Bachelor's degree	0.07	(0.25)	0.07	(0.26)	0.07	(0.26)	0.07	(0.26)
Completed Master's degree	0.01	(0.08)	0.01	(0.08)	0.01	(0.09)	0.01	(0.08)
Any secondary education (men)	0.54	(0.50)	0.54	(0.50)	0.56	(0.50)	0.53	(0.50)
Completed secondary education (men)	0.24	(0.43)	0.28	(0.45)	0.29	(0.45)	0.27	(0.45)
Any secondary education (women)	0.51	(0.50)	0.50	(0.50)	0.52	(0.50)	0.49	(0.50)
Completed secondary education (women)	0.24	(0.43)	0.28	(0.45)	0.29	(0.45)	0.27	(0.45)
Income & wealth:								
Worked during last 12 months	0.61	(0.49)	0.59	(0.49)	0.58	(0.49)	0.59	(0.49)

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Table D.3 - (Continued from previous page)

Household annual income (IDR)	31.42	(40.39)	35.90	(42.31)	42.33	(45.03)	34.06	(41.32)
Household annual income per capita (IDR)	6.62	(8.40)	6.81	(7.99)	7.92	(8.04)	6.49	(7.95)
Household annual income per adult member (IDR)	9.66	(11.81)	10.35	(11.88)	12.39	(12.51)	9.77	(11.63)
Annual salary (mil. IDR)	18.38	(19.79)	20.45	(20.90)	22.86	(20.81)	19.74	(20.87)
Annual salary (women; mil. IDR)	14.65	(17.63)	17.52	(20.16)	19.11	(21.10)	17.05	(19.87)
Annual salary (men; mil. IDR)	20.56	(20.64)	22.15	(21.11)	25.01	(20.35)	21.30	(21.25)
Employed in social services	0.19	(0.40)	0.21	(0.41)	0.24	(0.43)	0.21	(0.40)
Household owns house	0.77	(0.42)	0.74	(0.44)	0.67	(0.47)	0.76	(0.43)
Household size	5.15	(2.48)	5.68	(2.63)	5.73	(2.69)	5.67	(2.61)
Access to healthcare:								
Has any health insurance	0.47	(0.50)	0.50	(0.50)	0.53	(0.50)	0.50	(0.50)
General medical checkup performed (past 5 years)	0.09	(0.28)	0.09	(0.28)	0.11	(0.32)	0.08	(0.27)
Sought outpatient care (past 4 weeks)	0.19	(0.39)	0.18	(0.38)	0.15	(0.35)	0.19	(0.39)
- Type of last visited outpatient care provider:								
... Public Hospital	0.07	(0.26)	0.09	(0.28)	0.09	(0.28)	0.09	(0.28)
... Public Health Center*	0.23	(0.42)	0.32	(0.46)	0.34	(0.47)	0.31	(0.46)
... Private Hospital	0.07	(0.25)	0.05	(0.21)	0.06	(0.24)	0.04	(0.21)
... Polyclinic, Private Clinic, Medical Center	0.10	(0.30)	0.09	(0.29)	0.13	(0.33)	0.09	(0.28)
... Private Physician	0.20	(0.40)	0.19	(0.39)	0.14	(0.35)	0.20	(0.40)
... Nurse, Paramedic, Midwife practitioner	0.30	(0.46)	0.28	(0.45)	0.27	(0.44)	0.28	(0.45)
... Traditional practitioner	0.24	(0.43)	0.18	(0.38)	0.16	(0.37)	0.18	(0.39)
Distance to last visited outpatient care provider (in km)	6.70	(38.01)	7.88	(38.51)	6.44	(30.35)	8.24	(40.26)
Sought inpatient care (12 months)	0.05	(0.21)	0.05	(0.22)	0.05	(0.21)	0.05	(0.22)

Notes: Inverse probability weight (IPW) variables include father's log. income, father's age at birth, urban residence, Java, Kilmatan, Lesser Sunda Islands, and Sulawesi. Family member controls consist of female household head, household size, father Muslim, and mother having at least secondary education. Child controls include age in years, and female. Standard errors are clustered at the father-level and shown in parentheses. Significance levels show * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Table D.4 – Descriptive statistics by ethnic groups (estimation sample only)

	Javanese		Minority Groups					
			All		Special		Others	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Outcome:								
Birth weight of child (in kg)	3.12	(0.45)	3.16	(0.48)	3.13	(0.48)	3.17	(0.48)
Height for age z-score	-1.16	(1.36)	-1.34	(1.38)	-1.26	(1.30)	-1.37	(1.41)
Parental smoking patterns:								
Father ever smoked	0.35	(0.48)	0.37	(0.48)	0.34	(0.48)	0.38	(0.48)
Mom ever smoked	0.01	(0.08)	0.01	(0.11)	0.01	(0.12)	0.01	(0.11)
Father age started smoking	20.44	(4.85)	20.65	(4.89)	20.79	(4.53)	20.60	(5.02)
Mom age started smoking	20.14	(8.38)	24.65	(5.58)	21.17	(3.06)	25.88	(5.81)
Father number of cigarettes smoked daily	12.04	(7.36)	13.30	(8.53)	13.55	(8.54)	13.20	(8.53)
Mom number of cigarettes smoked daily	11.43	(1.51)	5.11	(3.86)	4.00	(1.26)	5.62	(4.56)
Education:								
Father's years of education	10.92	(3.91)	10.95	(4.20)	11.09	(3.97)	10.90	(4.29)
Mom's years of education	10.75	(3.83)	10.56	(4.17)	10.93	(4.27)	10.42	(4.13)
Father any secondary education	0.83	(0.38)	0.81	(0.39)	0.84	(0.37)	0.80	(0.40)
Mom any secondary education	0.83	(0.38)	0.79	(0.41)	0.81	(0.39)	0.79	(0.41)
Father completed secondary education	0.53	(0.50)	0.60	(0.49)	0.61	(0.49)	0.59	(0.49)
Mom completed secondary education	0.50	(0.50)	0.51	(0.50)	0.56	(0.50)	0.49	(0.50)
Father completed Bachelor's degree	0.20	(0.40)	0.21	(0.41)	0.20	(0.40)	0.21	(0.41)
Mom completed Bachelor's degree	0.19	(0.39)	0.18	(0.39)	0.23	(0.42)	0.17	(0.38)
Father completed Master's degree	0.02	(0.15)	0.01	(0.09)	0.00	(0.00)	0.01	(0.11)
Mom completed Master's degree	0.01	(0.10)	0.00	(0.06)	0.01	(0.11)	0.00	(0.03)

continued on next page

Table D.4 - (Continued from previous page)

Income & wealth:								
Father worked during last 12 months	0.99	(0.10)	0.99	(0.11)	0.99	(0.08)	0.99	(0.11)
Mom worked during last 12 months	0.55	(0.50)	0.56	(0.50)	0.58	(0.49)	0.55	(0.50)
Father's annual income (Mil. IDR)	25.84	(22.51)	29.36	(24.19)	33.97	(24.68)	27.50	(23.75)
Mom's annual income (Mil. IDR)	16.97	(19.68)	20.52	(20.75)	22.39	(21.19)	19.73	(20.54)
Household annual income (IDR)	35.01	(36.89)	41.88	(41.96)	48.42	(41.82)	39.29	(41.75)
Household annual income per capita (IDR)	7.44	(7.80)	8.37	(7.73)	9.56	(7.56)	7.90	(7.75)
Household annual income per adult member (IDR)	13.22	(13.89)	15.43	(14.97)	18.10	(14.68)	14.37	(14.97)
Father employed in social services	0.24	(0.43)	0.27	(0.44)	0.24	(0.43)	0.28	(0.45)
Mom employed in social services	0.29	(0.45)	0.35	(0.48)	0.38	(0.49)	0.33	(0.47)
Any HH member employed in social services	0.37	(0.48)	0.39	(0.49)	0.38	(0.48)	0.39	(0.49)
Household owns house	0.73	(0.44)	0.70	(0.46)	0.66	(0.48)	0.72	(0.45)
Household size	4.91	(1.75)	5.30	(2.19)	5.33	(2.13)	5.28	(2.21)
Access to healthcare:								
Any HH member had general med. checkup performed (past 5 years)	0.21	(0.41)	0.21	(0.41)	0.26	(0.44)	0.19	(0.40)
Any HH member sought outpatient care (past 4 weeks)	0.39	(0.49)	0.37	(0.48)	0.31	(0.46)	0.39	(0.49)
- Type of last visited outpatient care provider:								
... Public hospital past 4 weeks	0.06	(0.23)	0.13	(0.33)	0.09	(0.29)	0.14	(0.35)
... Public Health Center* past 4 weeks	0.24	(0.43)	0.31	(0.46)	0.36	(0.48)	0.29	(0.46)
... Private hospital past 4 weeks	0.10	(0.30)	0.08	(0.27)	0.12	(0.33)	0.07	(0.25)
... Polyclinic, Private Clinic, Medical Center past 4 weeks	0.15	(0.36)	0.15	(0.36)	0.23	(0.43)	0.13	(0.34)
... Private Physician past 4 weeks	0.16	(0.37)	0.19	(0.39)	0.13	(0.34)	0.21	(0.40)
... Nurse, Paramedic, Midwife practitioner past 4 weeks	0.31	(0.46)	0.31	(0.46)	0.28	(0.45)	0.32	(0.47)
... Traditional practitioner past 4 weeks	0.31	(0.46)	0.16	(0.36)	0.11	(0.32)	0.17	(0.38)
HH mean distance to last visited outpatient care provider (in km)	6.40	(19.64)	6.08	(16.05)	5.80	(11.68)	6.17	(17.29)
Any HH member sought inpatient care (12 months)	0.15	(0.35)	0.15	(0.35)	0.14	(0.35)	0.15	(0.36)

Notes: The table shows the means, standard deviations (SD), and number of observations (n) for the treatment group as well as the control group before and after reweighting, separately. Additionally, the difference in means between treatment (TG) and control group (CG), before and after reweighting, are shown separately. Asterisks indicate the significance level of the differences in means between the treatment and the control group by t-tests. *** 1% level of significance, ** 5% level of significance, * 10% level of significance.
Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Table D.5 – Balancedness of treatment status

	Treatment group			Control group						Difference	
				No weights			Weights			No weights	Weights
	Mean	SD	n	Mean	SD	n	Mean	SD	n		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
Outcome:											
Height-for-age z-score	-1.44	1.18	362	-1.23	1.40	2249	-1.35	1.37	2244	-0.210***	-0.091
IPW variables:											
Urban	0.52	0.50	362	0.66	0.47	2249	0.49	0.50	2244	-0.137***	0.032
Log. Income Father	18.97	5.59	362	18.45	4.67	2249	18.92	5.68	2244	0.523*	0.052
Father's age at child's birth	24.81	5.19	362	32.55	6.55	2249	24.99	5.27	2244	-7.742***	-0.182
Father's age at child's birth squared	642.45	293.54	362	1102.64	465.37	2249	652.48	327.26	2244	-460.187***	-10.031
Java	0.43	0.50	361	0.50	0.50	2244	0.44	0.50	2244	-0.076***	-0.012
Kalimantan	0.06	0.23	361	0.06	0.24	2244	0.06	0.24	2244	-0.007	-0.004
Lesser Sunda Islands	0.13	0.33	361	0.14	0.34	2244	0.12	0.32	2244	-0.010	0.009
Sulawesi	0.05	0.21	361	0.06	0.23	2244	0.06	0.24	2244	-0.011	-0.012
Sumatra	0.34	0.47	361	0.24	0.43	2244	0.32	0.47	2244	0.104***	0.019
Additional Covariates:											
Age (in years)	5.03	2.66	362	4.73	2.74	2249	4.57	2.86	2244	0.299*	0.464***
Female	0.49	0.50	362	0.47	0.50	2249	0.47	0.50	2244	0.023	0.016
Household size	4.63	1.70	362	4.72	1.51	2249	4.73	1.81	2244	-0.086	-0.097
Household head female	0.03	0.16	362	0.03	0.17	2249	0.06	0.23	2244	-0.000	-0.028**
Mother at least secondary education	0.78	0.41	362	0.80	0.40	2249	0.79	0.41	2244	-0.015	-0.011
Father Muslim	0.88	0.32	362	0.88	0.32	2249	0.88	0.32	2244	0.004	-0.000
Father at least secondary education	0.80	0.40	362	0.82	0.39	2249	0.82	0.38	2244	-0.012	-0.021

Notes: The table shows the means, standard deviations (SD), and number of observations (n) for the treatment group (father started smoking zero to three years before the child's birth) as well as the control group (father did not start smoking zero to three years before the child's birth) before and after reweighting, separately. Additionally, the difference in means between treatment (TG) and control group (CG), before and after reweighting, are shown separately. Height-for-age z-score relates the child's height (or length for children under two) to the height of a comparable reference population of the same sex and age. Asterisks indicate the significance level of the differences in means between the treatment and the control group by t-tests. *** 1% level of significance, ** 5% level of significance, * 10% level of significance.

Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Table D.6 – Effects of SHS exposure on height-for-age z-scores by share of child's ethnicity within district (interaction with Javanese)

	below 10 p.	below 20 p.	below 30 p.	below 40 p.
	(1)	(2)	(3)	(4)
Father started smoking	-0.198	-0.220*	-0.234*	-0.223*
	(0.122)	(0.124)	(0.127)	(0.129)
Javanese*Father started smoking	0.276	0.282	0.321*	0.317*
	(0.179)	(0.181)	(0.183)	(0.183)
Javanese*Father started smoking*Segregation	0.268	0.326	0.031	0.065
	(0.649)	(0.421)	(0.367)	(0.350)
Segregation	0.640*	0.176	0.047	-0.082
	(0.362)	(0.212)	(0.163)	(0.156)
Segregation*Father started smoking	-1.178**	-0.405	-0.200	-0.195
	(0.483)	(0.362)	(0.297)	(0.274)
Javanese	-0.145	-0.159	-0.165	-0.185
	(0.165)	(0.167)	(0.168)	(0.169)
IPW variables	Yes	Yes	Yes	Yes
Family member controls	Yes	Yes	Yes	Yes
Child controls	Yes	Yes	Yes	Yes
Observations total	2605	2605	2605	2605
Observations treated	362	362	362	362

Notes: Height-for-age z-score relates the child's height (or length for children under two) to the height of a comparable reference population of the same sex and age. Father started smoking is equal to 1 if a father started smoking zero to three years before the child's birth and 0 if the father remained a non-smoker during the same period. Inverse probability weight (IPW) variables include father's log. income, father's age at birth, urban residence, Java, Kilmatan, Lesser Sunda Islands, and Sulawesi. Family member controls consist of female household head, household size, father Muslim, and mother having at least secondary education. Child controls include age in years, and female. "Below X p." indicates the district share of a child being below X percent. Further details on the variables are presented in Appendix A. Standard errors are clustered at the father-level and shown in parentheses. Significance levels show $^{*}\ p < 0.10$, $^{**}\ p < 0.05$, $^{***}\ p < 0.01$.
Source: Own calculations based on the Indonesian Family Life Survey (IFLS) 2014/2015.

Appendix E - Figures

Figure E.1 – Balancedness of covariates

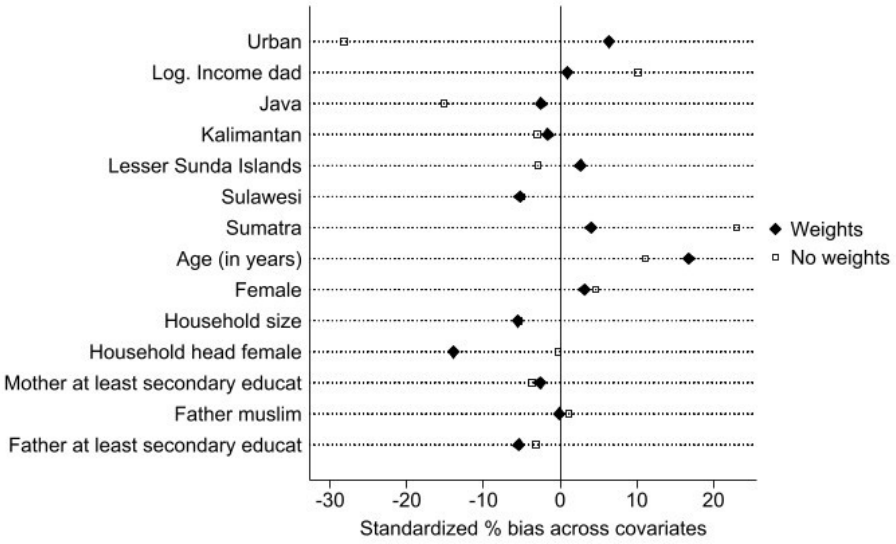
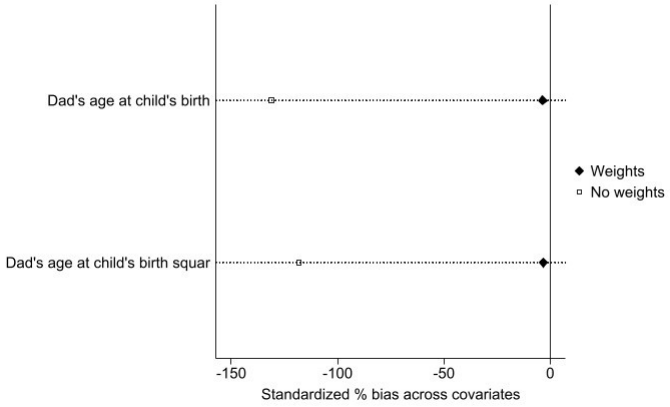


Figure E.2 – Balancedness of additional covariates



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Endnotes

[1] In this article, we use the term ethnicity rather than race as the data underlying our analyses works with the former term. However, similar to race, we regard ethnicity as akin to caste. As such, it is a separate construct from class and not a biologically meaningful category (Kawachi et al., 2005)

[2] Health disparities across race and ethnicity tended to grow during the COVID-19 pandemic (Alsan et al., 2021, Cheung et al., 2022, Irizar et al., 2023, Amele et al., 2023). Recent data from the US suggest that, possibly due to disproportionate pandemic-induced stress and anxiety (Chen-Sankey et al., 2020), smoking rates of the black minority group is for the first time larger than that of non-Hispanic white adults (Arrazola et al., 2023). Causal evidence suggests that ethnic minorities engage less in health prevention due to mistrust in the medical system due to institutional racism and discrimination (e.g., Anderson, 2023, Alsan and Eichmeyer, 2024). This, in turn, contributes to increased mortality rates (Alsan and Wanamaker, 2017, Friedman et al., 2021).

[3] Studies have started to provide causal associations between social determinants and health outcomes (e.g., Alsan and Wanamaker, 2017, Kawachi and Subramanian, 2018). However, today knowledge of the causes of persistent health disadvantages of ethnic minority groups remains incomplete (Bhopal, 1997, Kawachi et al., 2005, Hicken et al., 2018).

[4] Many studies argue that the biological pathway is unlikely to be of high significance for ethnic health disparities (e.g., Bliss, 2011, Kaufman et al., 2015).

[5] Bella et al. (2023) present estimates of the stunting effects of shifts in parental smoking status that occur after the birth of the child rather than procreation in Indonesia. Hence, it does not cover SHS exposure during the pregnancy period, which is of high general relevance for child development (Alati et al., 2006). Paraje and Valdes (2021) report effect estimates of current parental smoking status and transitions in parental smoking status on parents' self-report of the current health of their children in Chile. A larger body of literature is concerned with the effects of other forms of air pollution, which has paid relatively more attention to the context of developing countries (e.g., Rosales-Rueda and Triyana, 2019).

[6] Remarkably, there are a few ethnic minority groups (we call them Special minority groups) for which we find the effects to go in the opposite direction compared to most minorities, i.e., results for the special minority groups are in line with the effects observed for the Javanese ethnic group.

[7] Same-ethnic density can have a protective effect but also intensify low birth weight outcomes among ethnic minorities (Shaw et al., 2010, Mehra et al., 2017, Karlsen et al., 2002). Different ethnic groups can also have different concepts of illness that potentially explain the differential propensity of seeking care (Laksono et al., 2020).

[8] In Table D.6 of the Appendix D, we additionally display the results when analogously augmenting Equation (3). We observe the same results pattern. When using binary variables indicating higher population shares, the results look quite similar to our main results, i.e., a concentration of the adverse child growth effects of paternal smoking initiation among ethnic minority groups. This is further support for the relevance of a very low population share of the child's own population in the district for our findings.

[9] This is, for instance, in line with findings from a systematic review and meta-analysis by Jones et al. (2011) who conclude that exposure to maternal smoking before birth has a weaker effect compared to maternal smoking exposure after birth.

[10] Free access to the data is available at https://www.rand.org/labor/FLS/IFLS.html.

[11] The HAZ is estimated by subtracting the reference population's median height from the height of the child and then dividing it by the reference population's standard deviation of the height.

[12] To maximize sample size, we use smoking history information from the IFLS waves 1997 and 2000 whenever the smoking status is missing in the IFLS 5 even though the data from the previous waves tend to be less accurate (Strauss et al., 2016).

[13] We show that our results are robust to defining the treatment in an alternative way, namely that the father not only initiated smoking but also reported that his cigarette consumption exceeds that of the sample's median number of cigarettes (results available upon request).

[14] To enhance readability, the standardized biases of father's age and father's age squared are shown in a separate graph as their initial values exceed that of all other covariates at least threefold.

[15] It implies in our case that $Y_{i,t+1}^{1}, Y_{i,t+1}^{0} \perp FSS|p(X)$.

[16] The findings are robust when using robust standard errors instead (results available upon request). Our results are also robust to including different indicators for the father's mental health as covariates (results available upon request). A low mental health status might increase the probability of smoking initiation while negatively affecting child development.

[17] Across the globe, ethnic minorities have been documented to display worse anthropometric measures. While their rates of stunting are larger in Southeast Asia, the obesity rates are worse among ethnic minority groups in the UK (e.g., Sutaria et al., 2019, Harris et al., 2021).