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Pusat Kajian Jaminan Sosial Universitas Indonesia

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Through thick and thin: Do parental smoking behaviors affect the children's stunting, thinness, and overweight status?

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ABSTRACT

The anticipated financial and health impacts of smoking exposure on children's status of malnutrition have been of global concern. Albeit the emerging double burden of malnutrition along with the remarkably high prevalence of smokers in Indonesia, few studies have examined the impacts of parental smoking on child nutritional status. Using a balanced panel data of the Indonesia Family Life Survey (IFLS), we analyzed the extent of parental smoking effects on the likelihood of stunting, thinness, and overweight on children. We employed a Probit Random Effect Model with Mundlak correction to remove the endogeneity issue and estimate the impact of parental smoking (smoking status and smoking intensity) on child malnutrition status. The finding showed that paternal smoking status could increase child's stunting by 3.73 percentage points and that one extra cigarette stick consumed by a father per day (paternal smoking intensity) could raise children's probability of thinness, stunting, and overweight by 0.16, 0.24, and 0.09 percentage points, respectively. Overall, exposure to paternal smoking increases the risk of stunting, thinness, and overweight in children. Key policies in tobacco-control should be encouraged to reduce the potential long-term effects of paternal smoking on the future human capital and economic growth in the country.

JEL classification: I12
Key words: parental smoking, child malnutrition, stunting, Indonesia

1. INTRODUCTION

The major issue of public health nutrition has transitioned from undernutrition to overweight and obesity on a global scale (Popkin, 2006). During the process, most developing countries may not fully proceed from the former to the latter phase, resulting in a condition referred to as the double burden of malnutrition – hunger, stunting, and wasting coexist with an increasing prevalence of overweight and obesity. One of the most affected groups of double burden of malnutrition in developing countries is children (Stevens et al., 2012; Uauy, Garmendia, & Corvalán, 2014). Evidence has shown that stunting and wasting among children are more prevalent in developing countries than developed countries (UNICEF., WHO., & The World Bank, 2012). Interestingly, while the prevalence of child overweight and obesity remains higher in advanced nations, many developing countries are now facing an increased incidence rate (De Onis, Blössner, & Borghi, 2012; Wabitsch, Moss, & Kromeyer-Hauschild, 2014).

As a developing country, Indonesia is no exception. While addressing the high rates of stunting and wasting among children for decades, Indonesia witnessed a gradual increase in childhood overweight and obesity. Despite a significant decrease in stunting prevalence by around 16 percentage points since 1995, the proportion of stunted children in Indonesia remained high at 30.8% in 2018, far exceeding the world's child stunting rate at 22.2% in 2017 (Gani & Budiharsana, 2018; Shrimpton & Rokx, 2015). The undernutrition issue was also deteriorated by a relatively high proportion of wasted children (10.2%) in 2018, placing Indonesia in the first- and second-highest rate of wasting and stunting, respectively, in Southeast Asia (ASEAN/UNICEF/WHO, 2016; Gani & Budiharsana, 2018). Additionally, the last three decades have seen a simultaneous incidence of overweight children that nearly doubled from 5.1% to 9.2% between 1993 and 2018 (Indonesian Ministry of Health, 2018; Rachmi, Li, & Alison Baur, 2017). The upshot is that the country has recently started facing the double burden of malnutrition (Gani & Budiharsana, 2018; Shrimpton & Rokx, 2015).

On the other hand, Indonesia is one of the prominent homes for tobacco smokers in the world; around 68% of Indonesian adult males and 32.8% of people were active smokers in 2016 (Reitsma et al., 2017; World Bank Group, 2018).
Whilst the rest of the world see a declining prevalence in smoking, Indonesia experienced a significant increase in the proportion of smokers with an annual growth of 0.2% and 1.8% in male and female smokers, respectively, from 1990 to 2015 (Reitsma et al., 2017). As a result, smoking is one of the leading causes of preventable diseases and disease-related healthcare costs in Indonesia. Tobacco smoking was responsible for approximately 21% of all chronic illnesses, 9.5% of total Disability-Adjusted Life Years (DALYs), and the fourth risk factor to most death and disability in Indonesia in 2017 (Kristina, Endarti, Wiedyaningsih, Fahamsya, & Faizah, 2018; Mboi et al., 2018; World Bank Group, 2018). Those smoking-induced diseases could have cost the country's economy approximately USD 45.9 billion in 2015 due to smoking-attributable healthcare expenditure, unavailing cigarette spending, DALYs loss, and premature death (Kosen, Thabrany, Kusumawardani, & Martini, 2017). Despite the appalling condition of smoking nationwide, Indonesia delayed its participation in the ratification of WHO Framework Convention on Tobacco Control (FCTC) and remained the lowest cigarette excise in Southeast Asia (Lian & Dorotheo, 2018).

[1] The FCTC, which was first opened for signature in 2003, was built as a response to the global tobacco epidemic, which aims to minimize the devastating impacts of tobacco consumption and exposure to tobacco smoke on health, social, environmental and the economy.

Considering the growing evidence of the relationship between parental smoking and child malnutrition in the literature (Best et al., 2007; Chowdhury et al., 2011; Davis et al., 2016; Koshy, Delpisheh, & Brabin, 2011; Nadhiroh, Djokosujono, & Utari, 2020; Yang, Decker, & Kramer, 2013), the concurrent problems of smoking and dual burden of malnutrition in Indonesia may raise a question as to whether these two indicators are significantly linked. However, only a few studies (Best et al., 2007; Block & Webb, 2009; Semba et al., 2007) have worked on parental tobacco exposure and child nutritional status in Indonesia. Due to the potential long-term impacts of nutritional problems on children as well as on the economic growth of the country (Haas, Glymour, & Berkman, 2011; Johnson & Schoeni, 2011; Martorell, Melgar, Maluccio, Stein, & Rivera, 2010), it is vital to have a clearer picture of these interlinked issues in Indonesia. Previous studies by Best et al. (2007), Semba et al. (2007), Block and Webb (2009) examined the association between parents' smoking behavior and child undernutrition while exempting children overweight and engaging limited indicators (stunting) in a short range of children's age. Additionally, most previous studies used cross-sectional data which generally cannot capture causal inference of parental smoking behavior on children's nutritional status.

We aim to fill the gap in the literature by analyzing the significance, magnitude, and direction of parental smoking on child stunting, thinness, and overweight using a longitudinal dataset to gain a clear observation of the long-term effect of parental smoking behaviors on their children's nutritional status. Since the Achilles' heel of a causal inference study is endogeneity issues, we employ a probit random effect model with Mundlak correction to address the issue. This study expects to contribute evidence for establishing policies in tobacco control and stunting or malnutrition in Indonesia and other developing countries.

This study proceeds as follows: Section 1 explains the motivation study. Section 2 provides a theoretical framework on the relationship between parental smoking behaviors and children nutritional status. Section 3 elaborates data and descriptive statistics, while section 4 presents econometric estimation procedures that discuss the random effect model with Mundlak correction. Section 5 and 6 elaborate the results and discuss the extent of parental smoking behaviors on the likelihood of stunting, thinness, and overweight on children. Lastly, the concluding section of the paper summarizes the key findings and discusses policy implications and limitations.

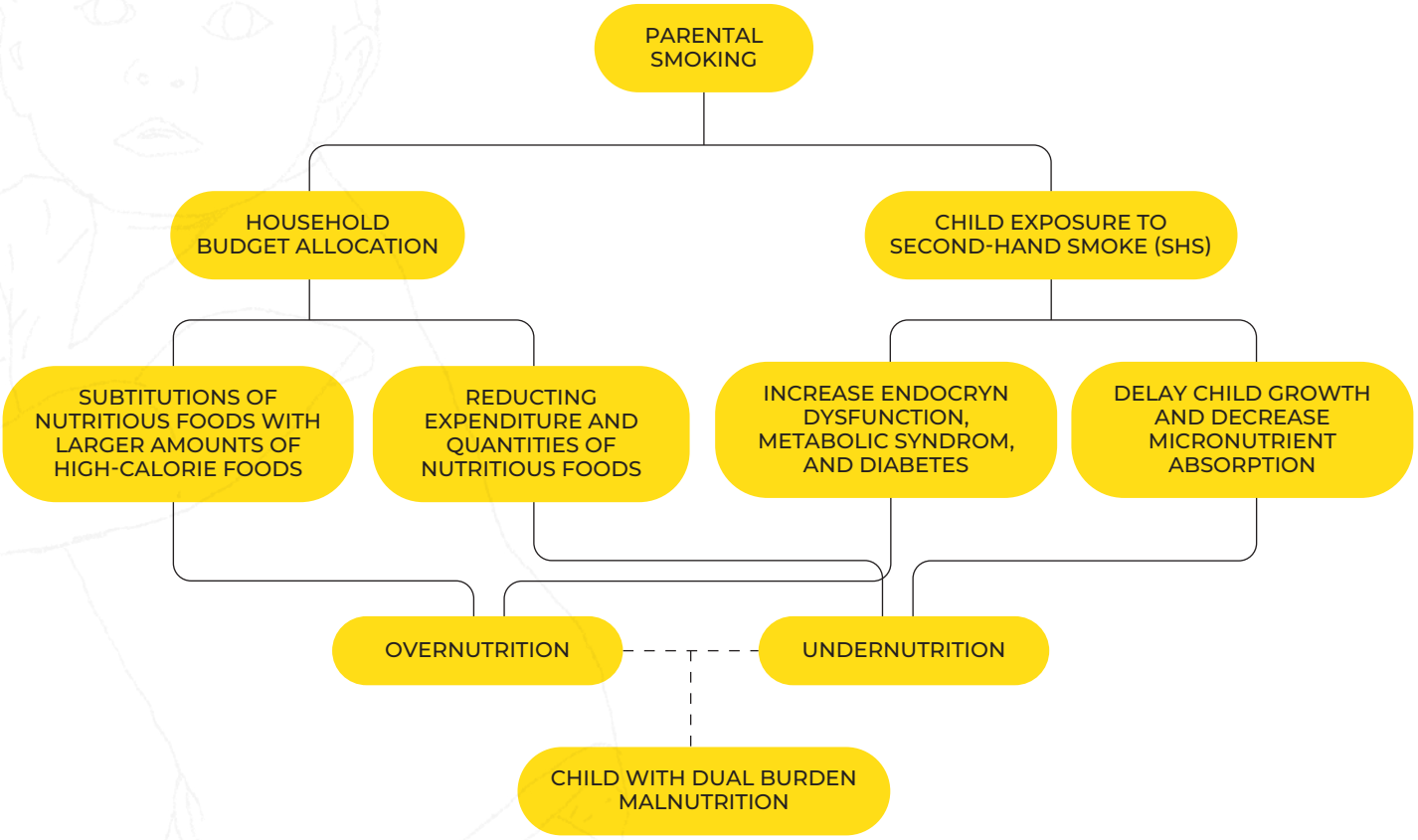
2. THEORETICAL FRAMEWORK

There has been a growing literature discussing the link between parental smoking exposure and child undernutrition (Best et al., 2007; Best et al., 2008; Block & Webb, 2009; Kyu, Georgiades, & Boyle, 2009; Muraro et al., 2014; Nonnemaker & Sur, 2007; Semba et al., 2007), as well as overweight and obesity of the offspring (Davis et al., 2016; Florath et al., 2014; Raum et al., 2011; Yang et al., 2013). This study proposed two possible mechanisms mediating parental smoking behavior on child malnutrition: crowding-out effects of tobacco expenditure and exposure to secondhand smoke. The possible link from parental smoking to children undernutrition and overweight/obesity is depicted in Figure 1.

Firstly, parental smoking may increase the likelihood of child malnutrition by diverting the household budget for foods into tobacco expenditure, namely the crowding-out effect of tobacco spending (Wang, Sindelar, & Busch, 2006). Consequently, children consume food of lower quantity and quality and eventually suffer from undernourishment, such as stunting and underweight (Block & Webb, 2009; John, 2008; Nonnemaker & Sur, 2007). It is estimated that if only a smoker stopped smoking, he/she could cater around 500 calories to one to two child(ren) a day (Efroymson et al., 2001; Nonnemaker & Sur, 2007). On the other hand, the crowding-out effect of tobacco could induce household food scarcity that subsequently leads to overweight and obesity among household members (Ghattas, 2014; Semba et al., 2011; Tanumihardjo et al., 2007). The food scarcity may affect overweight by substituting high-quality foods, such as meat, fruits, and vegetables, with the ones that contain lower nutrients and higher calories, like a number of staple foods (Bratanova, Loughnan, Klein, Claassen, & Wood, 2016; Tanumihardjo et al., 2007).

Secondly, parental smoking could increase child's exposure to secondhand smoke, leading to child undernourishment (Baheiraei et al., 2015; Ikeda, Irie, & Shibuya, 2013; Kyu et al., 2009; Muraro et al., 2014) and overweight and obesity (Durmuş et al., 2011; Florath et al., 2014; Raum et al., 2011; Yang et al., 2013). In terms of stunting and undernourishment, exposure to second-hand smoking in children may impede child growth by delaying child skeletal growth and diminishing micronutrient absorption in children (Caulfield, Richard, Rivera, Musgrove, & Black, 2006; Kawakita et al., 2008; K. M. Wilson, Finkelstein, Blumkin, Best, & Klein, 2011). Additionally, passive smoking contributes to overweight and obesity by inducing endocrine dysfunction, metabolic syndrome, and diabetes to the exposed children (Behl et al., 2013; Lisboa, Oliveira, & Moura, 2012; Weitzman et al., 2005).

Figure 1. Possible channels from parental smoking to child malnutrition



Source: Authors

3. DATA, DEFINITIONS, AND DESCRIPTIVE STATISTICS

3.1 Data

This research used panel data from the Indonesia Family Life Survey (IFLS), which were established by the RAND Corporation to collect social and economic indicators at individual, household, and community levels. The survey spanned to 21 years in five waves: in 1993/94 (IFLS1), 1997 (IFLS2), 2000 (IFLS3), 2007/08 (IFLS4), and 2014/15 (IFLS5). The first wave of IFLS, which interviewed over 22,000 individuals in 13 out of 26 Indonesia's provinces, contained a representative sample of 83% of Indonesian people (Frankenberg & Thomas, 2000).

This study used balanced-panel data by following a group of children aged 19 or younger who have at least one parent living under the same roof in all periods (see section 3.2.2). The age restriction is set to comply with WHO's definition of children (WHO, 2013). To ensure that the children followed in the first dataset may still be in the child age range in the last dataset, this study only used three latest waves of the IFLS (IFLS 3-5) and restricted the sample in IFLS 3 for children aged 0-4 years. Therefore, the children in our sample may still be categorized as children in the last wave (19 years or younger). The final sample—before accounting the effects of missing variables—contains 7,320 children-year.

3.2 Variables

3.2.1 Child-malnutrition variables

We obtained three malnutrition indicators to describe undernutrition and overnutrition problems in children – stunting, thinness, and overweight. Stunting is defined as having height-for-age z-scores < -2 standard deviation (SD) (WHO, 2010), while thinness means having weight-for-height z-scores < -2 SD for those aged below 5 years and having BMI-for-age z-scores < -2 SD for those aged 5-19 years. The term thinness was proposed by Cole, Flegal, Nicholls, and Jackson (2007) to avoid the confusion with 'underweight' or 'wasting' in undernutrition while providing a continuous indicator in children from birth to adolescence. We use dual indicators for thinness according to the WHO definitions of thinness using BMI-for-age (Riley, 2020) for children aged 5-19 years, and a study by Flegal, Wei, and Ogden (2002) that stated weight-for-height as a comparable estimate of BMI-for-age. Meanwhile, overweight is an overnutrition indicator, defined as having BMI-for-age z-scores > +1 (WHO, 2010). The overweight status, which covers obesity staus, is not splitted into obese and pre-obese status because the proportion of obese children is exceptionally low across the three waves of IFLS (1.85% of all children across all waves).

3.2.2 Definition of parents and parental-smoking variables

This study defines parents as the biological mother, father, or both who lived under the same roof with their children during the study undertaking. A parent is categorized as a current smoker if she/he regularly smoked either self-rolled cigarettes, commercial cigarettes, or cigars. Smoking a pipe and chewing tobacco was excluded from the study because both have incomparable indicators of addiction compared to cigarettes or cigars, and the prevalence of parents with these habits was also extremely low (0.12% of all parents). The measures of parental smoking included: 1) parental smoking status (dummy) and 2) parental smoking intensity (number of cigarettes or cigars consumed per day).

3.2.3 Other explanatory variables

This study incorporated additional variables to estimate child malnutrition, namely child characteristics, parental characteristics, and household characteristics. The child characteristics are the frequency of protein intake (total number of days a child eats either eggs or meat), while the parental characteristics are the father's and mother's height and BMI. The parental and household variables include father's and mother's education, household poverty status, household size, and some binary variables – access to clean water, electricity availability, province in Java Island, and urban area.

4 METHOD

This study employs the probit estimation to estimate the impact of parental smoking indicators on child nutritional status:

$$NS_{it} = \beta_0 + \beta_1 FS_{it} + \beta_2 MS_{it} + \sum_{j=1}^{n} \gamma_j X_{jit} + \alpha_i + \varepsilon_{it} \qquad (1)$$

where NS_{it} is a binary variable of child nutritional status (stunting, thinness, or overweight), $\boldsymbol{F\ ather\_Smoke}_{it}$ and $\boldsymbol{M\ other\_Smoke}_{it}$ are father's and mother's smoking-related variables, respectively, $\boldsymbol{X}_{jit}$ is a set of control variables, and $\boldsymbol{\alpha}_i$ and $\boldsymbol{\varepsilon}_{it}$ are time-invariant and time-variant errors, respectively. There are two smoking-related variables which are run separately for each dependent variable[2]: i) smoking status of a mother or a father living in the same household with the child, and ii) the number of cigarettes smoked per day by a mother or a father living in the same household with the child.

Estimating equation (1) with a strong assumption of zero correlation between the time-invariant error i and the covariates may be impractical. In this case, parents' rate of time preference may affect their decisions to smoke and to invest in their children's health (Agee & Crocker, 1996; Brown & Van der Pol, 2015; Khwaja, Silverman, & Sloan, 2007), so that the strict exogeneity assumption may not hold. In analyzing models with binary dependent variables, some well-known methods to overcome the violation of the strong exogeneity assumption may include conditional logit fixed effects (FE) and probit random effects (RE) with Mundlak correction. However, the former method may suffer from the elimination of subjects which lacks within-individual variations of the dependent variable and the exclusion of stable characteristics, such as sex and race, and the problem of lower efficiency (compared to random effects) (J. R. Wilson & Lorenz, 2015). Therefore, this paper addresses the strong-exogeneity issues by employing the latter method.

The idea of Mundlak (1978) was established to provide a consistent, unbiased, and more efficient estimate of panel regression by removing time-invariant unobserved heterogeneity from the model. The approach suggests that the time-invariant error i is a linear function of the time averages of all the time-varying explanatory variables, as written below:

$$\alpha_i = \theta_0 + \theta_1 \overline{FS}_{it} + \theta_1 \overline{MS}_{it} + \sum_{j=1}^{n} \delta_j \overline{X}_{jit} + \delta_i \qquad (2)$$

[2]The independent variables are run separately for each dependent variable to avoid the effect of multicollinearity among the parents' smoking-related variables.

where $\underline{\boldsymbol{ES}}_{it}$, $\underline{\boldsymbol{MS}}_{it}$, and $\underline{\boldsymbol{X}}_{it}$ are the time averages of $\boldsymbol{Father\_Smoke}_{it}$, $\boldsymbol{Mother\_Smoke}_{it}$, and other time-varying explanatory variables respectively, and δ_i is individual effects. After substituting equation (2) to equation (1), we can draw the model of random effects with Mundlak correction in the following estimation:

$$NS_{it} = \beta_0 + \beta_1 FS_{it} + \beta_2 MS_{it} + \sum_{j=1}^{n} \gamma_j X_{jit} + \theta_1 \overline{FS}_{it} + \theta_1 \overline{MS}_{it} + \sum_{j=1}^{n} \delta_j \overline{X}_{jit} + \delta_i + \varepsilon_{it} \quad (3)$$

where δ_i follows $\delta_i \tilde{} N(0,\sigma_2)$ and is independent of $\underline{\boldsymbol{ES}}_{it}$, $\underline{\boldsymbol{MS}}_{it}$, $\underline{\boldsymbol{X}}_{it}$, and ε_{it} for all i and t. In this paper, we argued that the source of omitted variable bias was limited to only unobserved heterogeneity, so we could draw the causal effects of parental smoking on child malnutrition status.

5. RESULT

5.1 Descriptive Analysis

Descriptive statistics of our data for children experiencing malnutrition status are depicted in Table 1. Around one-third of the children were stunted, and only about 10% and 5% of them experienced thinness and overweight, respectively. It is evident that approximately 40% of all children with various malnutrition statuses were exposed to their father's smoking, while only a tiny portion was exposed to their mother's. A deeper probe into the parents' smoking intensity across various malnutrition statuses found that on average, fathers smoked 10 times as many cigarettes as mothers per day (5.25-5.94 vs 0.01-0.05).

In households with children with various malnutrition statuses, on average, mothers had a slightly higher BMI and lower height than fathers. Children with malnutrition consumed meat/chicken and eggs around once and twice a week and, respectively. Turning to household characteristics, children who were overweight had parents with a slightly higher education level than children who were thin or stunted. In addition, 60% of children who were overweight lived in urban areas, whereas around 50-60% of children who were stunted or thin lived in rural areas. Around 57% of children who were stunted came from poor households, while around 53-58% of children who were thin or overweight came from wealthy households. However, almost all children had access to clean water and electricity.

5.2 Pooled probit and probit RE estimations of child malnutrition status

The marginal effects of pooled probit with cluster-robust standard errors and probit RE *without* Mundlak correction are set out in Appendix A. The pooled probit estimates indicated that none of the parental smoking behavior significantly affected child malnutrition status at least at a 5% significance level. The effect of father's smoking intensity on child's overweight status was negligible because it was only significant at a 10% significance level. However, these insignificant results might suffer omitted variable bias and inconsistent estimations. Furthermore, the pooled probit models ignored the substantial serial correlation in the composite error.

The other half of the table indicates the results of the probit RE model, which assumes a strict exogeneity of the correlation between individual heterogeneity and explanatory variables. Departing from the pooled probit models, the coefficient significance of the father's smoking intensity on child's overweight status increased to 5% significance level in the probit RE models, while the other indicators remained quite similar. Although the probit RE model appears to provide more efficient estimation results than the pooled one, the strict exogeneity assumption may not be fulfilled.

5.3 Probit RE with Mundlak correction: Impacts of parental smoking behaviors

We then explored the effect of parental smoking behaviors on child-malnutrition conditions by applying Mundlak correction to overcome the heterogeneity problems that cannot be solved by pooled probit or probit RE models. We also checked the robustness of the results by running three separate regressions with three different sets of control variables for each regression: household characteristics, household and parental characteristics, and household, parental, and child characteristics.

As illustrated in Table 2, a father's smoking status may increase the likelihood of child stunting by around 3-4 percentage points, which is consistent at 5% and 1% significance levels. Father's smoking intensity appeared to have small positive significant impacts on the risk of child's thinness, stunting, and overweight at 5% significance level in the fully controlled regressions. An additional cigarette consumed by a father per day may increase the risk of their child's thinness, stunting, and overweight by 0.16, 0.24, and 0.09 percentage points, respectively. Taken together, after removing the problem of unobserved heterogeneity, some coefficients of the parents' smoking indicators corrections become significant in the probit RE with Mundlak estimation across various dependent variables, and that the magnitudes tend to be higher than those estimated using the pooled probit and probit RE models.

5.4 Sensitivity analysis

This study performed the sensitivity analysis using the logit FE method to check the robustness of the probit RE estimate (see Appendix B). Engaging the same indicators and sample of the Probit RE with Mundlak correction model, the logit FE method provided the same direction of coefficient of the main estimation (see Model (1) on Appendix B). However, only the number of cigarettes consumed by the father significantly impacted the risk of stunting in children in the logit FE model. The result was anticipated since the logit FE method resulted in a drastic reduction in the number of samples of about 90% from the primary model, potentially leading to inconsistent estimation (Katz, 2001).

In addition, we estimated the logit FE model using the sample of parents regardless of their cohabitation status with the children (see Model (2) on Appendix B). Although parental smoking behavior did not significantly impact child malnutrition indicators, the coefficient seemed smaller than that in parents who lived with the children. The second model may indicate greater passive smoking and financial impacts of parent's cigarette consumption on cohabiting children.

Table 1. Descriptive statistics

Variable names	Variable definition	Thinness		Stunting		Overweight	
		Mean or %	Std. Dev.	Mean or %	Std. Dev.	Mean or %	Std. Dev.
Malnutrition							
Percentage of children with related malnutrition status	Percentage of children with thinness, stunting, and overweight	9.98%	30%	29.11%	45%	4.56%	21%
Parental smoking behaviors							
Father's smoking status	(1) if father was a current smoker and lived in the same house with the child; (0) otherwise	45.21%	50%	48.92%	50%	46.25%	50%
Mother's smoking status	(1) if mother was a current smoker and lived in the same house with the child; (0) otherwise	0.28%	5%	0.94%	10%	0.90%	9%
Father's smoking intensity	The number of cigarettes/cigars consumed per day by a father who lived in the same house with the child	5.25	7.61	5.61	7.35	5.94	8.53
Mother's smoking intensity	The number of cigarettes/cigars consumed per day by a mother who lived in the same house with the child	0.01	0.22	0.04	0.50	0.05	0.54
Parental characteristics							
Father's BMI	Father's BMI	24.90	85.95	23.99	69.61	24.64	11.64
Mother's BMI	Mother's BMI	25.98	90.12	24.39	52.44	26.13	12.31
Father's height	Father's body height in cm	161.34	8.70	159.40	8.01	162.6053	8.48815
Mother's height	Mother's body height in cm	150.61	7.70	148.69	6.69	151.2029	8.020426
Child characteristics							
Child's frequency of eating meat	Value of a child who consumed meat or chicken in the last seven days	1.25	1.69	1.19	1.60	1.62	1.92
Child's frequency of eating egg	Value of a child who consumed eggs in the last seven days	2.61 150.61	2.15	2.60	2.24	2.68	2.14
Household characteristics							
Highest educational attainment of father	Father's years of schooling	7.94	4.10	7.42	4.16	9.06	4.24
Highest educational attainment of mother	Mother's years of schooling	7.50	3.98	6.92	3.83	8.53	4.27
Java	(1) if living in Java Island; (0) otherwise	58.23%	49%	49.23%	50%	57.49%	50%
Urban	(1) if living in an urban area; (0) otherwise	49.93%	50%	40.97%	49%	61.08%	49%
Clean water source	(1) if have access to clean water; (0) otherwise	88.61%	32%	83.81%	37%	88.92%	31%
Electricity availability	(1) if have access to electricity; (0) otherwise	93.95%	24%	91.46%	28%	95.21%	21%
Household poverty status	(1) if categorized as poor household; (0) otherwise	46.55%	50%	57.25%	49%	41.92%	49%
Household size	Number of household members	5.09	1.81	5.15	2.02	4.95	1.86
Observations		**711**		**2131**		**334**	

Source: Authors' estimation

Table 2. Marginal effects of the impacts of parental smoking behaviors using Probit RE with Mundlak corrections

Explanatory Variables	Dependent Variables								
	(1) Thinness	(2) Thinness	(3) Thinness	(1) Stunting	(2) Stunting	(3) Stunting	(1) Overweight	(2) Overweight	(3) Overweight
Father's smoking status	0.00394 (0.00973)	0.0129 (0.0107)	0.0153 (0.0108)	0.0303** (0.0121)	0.0368** (0.0143)	0.0373*** (0.0144)	0.00693 (0.00634)	0.0107 (0.00727)	0.0114 (0.00735)
Mother's smoking status	-0.0902 (0.0615)	-0.0975 (0.0692)	-0.0932 (0.0691)	0.0416 (0.0629)	0.0156 (0.0768)	0.0186 (0.0769)	-0.0186 (0.0294)	0.0195 (0.0361)	0.0183 (0.0364)
Observation	7113	5747	5724	7311	5926	5903	7311	5926	5903
Father's smoking intensity	0.00112* (0.000617)	0.00143** (0.000697)	0.00161** (0.000701)	0.00182** (0.000805)	0.00236** (0.000934)	0.00238** (0.000942)	0.000581 (0.000399)	0.000796* (0.000446)	0.000893** (0.000454)
Mother's smoking intensity	-0.0143 (0.0149)	-0.0205 (0.0164)	-0.0199 (0.0166)	0.00281 (0.0116)	-0.00384 (0.0152)	-0.00324 (0.0152)	-0.00244 (0.00477)	0.0113 (0.00879)	0.0110 (0.00879)
Observation	7113	5747	5724	7311	5926	5903	7311	5926	5903
Control variables:									
Household characteristics	YES	YES	YES	YES	YES	YES	YES	YES	YES
Parental characteristics	NO	YES	YES	NO	YES	YES	NO	YES	YES
Child characteristics	NO	NO	YES	NO	NO	YES	NO	NO	YES

Standard errors in parentheses
* p < 0.1, ** p < 0.05, *** p < 0.01

Household characteristics: residence in Java Island, residence in urban area, safe water source, use of electricity, household poverty status, household size; parental characteristics: parents' BMI, parents' height, parents' years of schooling; child characteristics: frequency of eating meat and eggs in a week.

Source: Authors' estimation

6. DISCUSSION

This study has observed the extent of the parental smoking effect on children's status of undernutrition and overweight using balanced panel data from the third, fourth, and fifth waves of IFLS. By removing the potential endogeneity issues with a probit RE model with Mundlak correction, we revealed that paternal smoking could increase the likelihood of child malnutrition, particularly stunting, by 3.73 percentage points. Regarding the effects of father's smoking intensity, each additional cigarette stick consumed by a father per day could increase their child's probability of thinness, stunting, and overweight by 0.16, 0.24, and 0.09 percentage points, respectively.

The mechanism through which child nutritional status is affected by parental smoking has been well documented in previous studies (Davis et al., 2016; Harris, Willett, & Michels, 2013; Jeong, Jang, Kang, Joo, & Park, 2021; Koshy et al., 2011; Nadhiroh et al., 2020) as being mediated through passive smoking effect (PSE). Either through secondhand smoking (SHS) or third-hand smoking (THS), the exposure of more than 250 carcinogenic and toxic substances from cigarette smoke (Talhout et al., 2011) can have an extensive harmful impact on children (Anderson & Cook, 1997; Davis et al., 2016; Diver, Jacobs, & Gapstur, 2018), including their growth and development (Peterson & Hecht, 2017; Yang et al., 2013). A recent study by Jeong et al. (2021) has demonstrated that the exposure to harmful substances from SHS and THS in children significantly increased with the number of cigarettes smoked by the parents. In terms of overweight, the close association of insulin resistance and obesity has been proposed as the possible pathway of how the exposure of cigarette smoke can predispose children to become overweight, which in turn leads to even further metabolic diseases (Harris et al., 2013; Weitzman et al., 2005). The result of this study supports other recent studies (Davis et al., 2016; Harris et al., 2013; Yang et al., 2013) reporting that children exposed to paternal smoking are more likely to develop obesity.

With regards to undernutrition, although the causal path of PSE altering children's growth remains inconclusive (Vilcins, Sly, & Jagals, 2018), Kawakita et al. (2008) suggested that the exposure to nicotine contained in cigarette smoking is associated with delayed human skeletal growth. In our study, stunted children were linked to both paternal smoking status and intensity, thus confirming the significant correlation reported in the previous study (Astuti, Handayani, & Astuti, 2020). Concerning the effect of smoking exposure on thinness in children, there has been a growing body of evidence that simultaneously explored the incidence of both underweight and wasting in children in correlation with parental smoking (Best et al., 2007; Semba et al., 2007; Tielsch et al., 2009). However, most of these studies (Best et al., 2007; Semba et al., 2007) still used paternal smoking status as the exposure indicator. Accordingly, the present study extends the existing evidence by highlighting that the more cigarettes smoked by fathers, the higher the risk of both stunting and thinness in children.

Besides the possible biological pathway of smoking exposure on child growth and development, financial impact is another attributable factor that has been well established as related to the likelihood of both undernutrition and overweight in children due to cigarette smoking exposure. Based on the National Socio-Economic Household Survey 2019 (Central Bureau of Statistics, 2019), on a weekly basis, households in Indonesia consumed approximately 98 cigarette sticks and spent almost a hundred thousand Rupiah (approximately USD 6-7) on cigarettes, which takes up about 20% of household food expenditure. As reported previously, the large share of cigarette spending in household expenditure suggests a crowding-out effect of tobacco smoking on other -

- household necessities, especially food (Efroymson et al., 2001; Jumrani & Birthal, 2017; Sreeramareddy & Ramakrishnareddy, 2017; Wang et al., 2006). A related study in Indonesia has revealed that an increased budget share for cigarettes would eventually reduce budget share for almost all food commodities, including calorie and protein intake (Djutaharta, Nachrowi, Ananta, & Martianto, 2021). It reflects the pivotal role of cigarette consumption in diverting the fund supposedly dedicated for food, thus negatively impacting nutrient intake in the children.

On the contrary, the financial impact of cigarette consumption may lead to household insecurity, which is then linked to child overweight. Preceding papers have discovered that the presence of adult smokers, especially men and fathers, would increase the likelihood of food insecurity in the households (Cutler-Triggs, Fryer, Miyoshi, & Weitzman, 2008; Semba et al., 2011; Sreeramareddy & Ramakrishnareddy, 2017). Consequently, the children have no access to healthier options but a poor-quality or less nutritious diet, leaving them vulnerable to being overweight (Au et al., 2019; Kral, Chittams, & Moore, 2017). Children from food-insecure families tend to have a higher BMI and develop obesity due to the substitution of nutritious foods with a relatively higher intake of staple foods, excessive sugar, and snacks (Au et al., 2019; Bratanova et al., 2016; Kral et al., 2017).

The limitation of this study is the self-stated data of parental smoking variables, which may suffer the problem of recall and social desirability bias. Moreover, due to the adoption of balanced panel data, the attrition problem that resulted in a lower number of observations is inevitable. Notwithstanding the limitation, to the best of our knowledge, this study is among the limited research (Koshy et al., 2011) exploring both undernutrition and overweight simultaneously and the first study exploring such an issue in Indonesia. While previous related studies in Indonesia employed only binary variables of parental smoking (Best et al., 2007; Semba et al., 2007), the present study also uses a continuous smoking measure as an arguably better smoking indicator. The use of the number of cigarettes consumed per day in this study averts the lumped smoking addiction level of parents as found in the dichotomous smoking variable (Emery, White, & Pierce, 2001; Fang, Ali, & Rizzo, 2009). Unlike the preceding studies in Indonesia probing into undernutrition issues in children under five years, this paper examines the effects of parental smoking on children's undernutrition and overweight by following the same children from zero to 19 years of age. Having removed unobserved heterogeneity by employing probit random effect with Mundlak correction, this study draws a causal relationship between parental smoking and child malnutrition.

7. CONCLUSION

Our findings reinforce the idea that paternal smoking may increase the risk of stunting, thinness, and overweight among their children. The effect of paternal smoking on child malnutrition that ranges from undernutrition to overweight provides a vital implication that paternal smoking could have a higher range of deteriorating impacts on child development than what preceding evidence has provided. Considering the emergence of double burden malnutrition in Indonesia and its potential adverse effects on human capital in the long term, tobacco control policies may unintendedly provide dual benefits on tackling nutrition problems in Indonesia. We further suggest planning a key policy in tobacco control to reduce paternal cigarette consumption and child exposure to smoking.

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APPENDICES

Appendix A. Marginal effects of the pooled probit and probit RE regressions

Explanatory Variables	Dependent Variables					
	Thinness		Stunting		Overweight	
	Pooled Probit	Probit RE	Pooled Probit	Probit RE	Pooled Probit	Probit RE
Father's smoking status	0.00241 (0.00821)	0.0047 (0.00838)	0.012 (0.012)	0.0165 (0.0118)	0.00711 (0.00561)	0.00804 (0.00574)
Mother's smoking status	-0.0846 (0.0569)	-0.0877 (0.0587)	0.0603 (0.0568)	0.0467 (0.0618)	0.0166 (0.0272)	0.0184 (0.0263)
Observation	5724	5724	5903	5903	5903	5903
Father's smoking intensity	0.000177 (0.000527)	0.000416 (0.000519)	0.000333 (0.000758)	0.000729 (0.000742)	0.000684* (0.000355)	0.000713** (0.000332)
Mother's smoking intensity	-0.0175 (0.0135)	-0.0183 (0.0143)	0.00533 (0.0097)	0.00249 (0.012)	0.00364 (0.00431)	0.00458 (0.00443)
Observation	5724	5724	5903	5903	5903	5903

Standard errors in parentheses
* p < 0.1, ** p < 0.05, *** p < 0.01

Regressions were controlled by parents' BMI, parents' height, parents' years of schooling, frequency of eating meat and eggs in a week, residence in Java Island, residence in urban area, safe water source, use of electricity, household poverty status, and household size.

Appendix B. Sensitivity analysis

Explanatory Variables	Dependent Variables					
	(1) Thinness	(2) Thinness	(1) Stunting	(2) Stunting	(1) Overweight	(2) Overweight
Father's smoking status	0.00115 (0.623)	0.00140 (0.770)	0.0000451 (0.000196)	0.00000793 (0.0000472)	0.000376 (0.191)	0.0348 (0.026)
Mother's smoking status	-0.0103 (5.585)	0.00167 (0.916)	0.000000952 (0.000154)	-0.000108 (0.000472)	-0.000559 (0.284)	0.0309 (0.0929)
Observation	1150	1152	2071	2074	543	541
Father's smoking intensity	0.00011 (0.0869)	0.0000693 (0.0555)	0.00388** (0.00182)	0.000000147 (0.00000103)	0.000126 (0.00486)	0.00242 (0.00161)
Mother's smoking intensity	-0.00197 (1.558)	-0.0000313 (0.0251)	-0.00322 (0.0246)	-0.00000658 (0.0000291)	0.00113 (0.0435)	0.042 (0.0417)
Observation	1150	1152	2071	2074	543	541

Standard errors in parentheses
* p < 0.1, ** p < 0.05, *** p < 0.01

Regressions were controlled by parents' BMI, parents' height, parents' years of schooling, frequency of eating meat and eggs in a week, residence in Java Island, residence in urban area, safe water source, use of electricity, household poverty status, and household size.

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