



# Association Between Parents' Smoking Status and Tobacco Exposure in School-age Children: Assessment of Major Urine Biomarkers

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## INTRODUCTION

- With secondhand smoke (SHS), individuals can be exposed to more than 250 carcinogens and toxic chemicals. Exposure to SHS is as harmful as the act of smoking itself because it can also cause various diseases such as lung cancer, cardiovascular disorders, and chronic diseases among non-smokers. Children are especially vulnerable to SHS. Exposure to SHS in children leads to early death and increases the risk of sudden infant mortality syndrome (SIDS), acute respiratory infections, and severe asthma symptoms. SHS also has severe adverse health effects, such as the slowing of lung growth in children.
- At the global level, around 40% of children are still exposed to SHS at home or other places frequently visited by children, and most exposures are related to parental smoking. While most parents tend to refrain from smoking in the company of children, some parents consider it acceptable to smoke nearby, on the balcony or near the window. but This may lead to entry of smoke into the room or its spread nearby, thus exposing children to SHS.
- Many studies have been conducted on the association between parents' smoking status and SHS exposure biomarkers in children. However, most previous studies have focused on adolescents in terms of smoking probability or on children with certain diseases.
- Therefore, we aimed to investigate the association of parental smoking with NNAL and cotinine concentrations, biomarkers of SHS exposure, in children.

## MATERIALS AND METHODS

- Data source:** This study used data from the 2018 Korea National Health and Nutrition Examination Survey (KNHANES VII) 2016-2018 for analysis.
- Study population:** The total number of respondents for the 2016–2018 KNHANES was 24,269. Participants who were not matched based on parent-child relationships (n=1,384), those without data on age (n=21,019) for those aged between 6-12 years, and those with no data on NNAL levels, cotinine levels, or other independent variables (n=992) were excluded. Finally, a total of 847 participants were included in the study.
- Outcome variables:** We used outcome variables were NNAL and cotinine concentrations, which are biomarkers of indirect smoking exposure and were used to quantify the children's degree of exposure to SHS.
- Interesting variables:** The main independent variable was the parents' smoking status, classified as "smoker" if any one of the parents replied "yes" or as "non-smoker" if both parents replied "no" to the question "Do you currently identify yourself as a smoker?". Furthermore, as a subgroup analysis, the amount of smoking (none, <10, 10–19, and ≥20) and the smoking patterns of parents (non-smoking parents, father only, mother only, both parents) were classified.
- Covariates:** Data on the participants' sociodemographic, economic, and health-related characteristics, as well as the survey year were assessed. Sociodemographic characteristics included children's sex, age, parents' age, education level, type of housing, and region. Economic characteristics included the parental household income and ownership of private health insurance. Health-related characteristics included parents' drinking status and children's exposure to SHS at home and in public.
- Statistical analysis:** Univariate linear regression was used to assess the relationship between children's NNAL and cotinine concentrations and parents' smoking status; Prior to the multiple logistic regression analysis, we performed a log-transformation of the NNAL and cotinine values to ensure normality. Multiple regression analysis was performed while controlling for covariates to analyze the association between parental smoking status, amount of cigarettes smoked, smoking patterns and log-transformed NNAL and cotinine concentrations in children. All statistical analyses were carried out using SAS software, version 9.4 (SAS Institute, Inc.). Statistical results were considered significant at a p-value <.05.

## RESULTS

Table 1. General characteristics of the study population

Variables	N	%	NNAL			Cotinine		
			MEDIAN	IQR	P value	MEDIAN	IQR	P value
Parents' smoking status					<.0001			<.0001
Smoker	392	44.9	1.4	1.9		0.4	0.4	
Non-smoker	482	55.1	0.8	0.9		0.2	0.3	

Table 2. Association between the concentrations of urinary NNAL and cotinine in children and parents' smoking status

Variables	Log-transformed model*					
	NNAL			Cotinine		
	β	S.E	P value	β	S.E	P value
Parents' smoking stauts						
Smoker	0.482	0.065	<.0001	0.472	0.06	<.0001
Non-smoker	Ref.		Ref.			

\* Adjusted for children's sex, age, BMI, secondhand smoke exposure (house), and secondhand smoke exposure (public) and for parents' household income, type of housing, region, age, education level, private health insurance, drinking status, and year of evaluation.

- Of the 874 children, 392 (44.9%) and 482 (55.1%) were identified as smoking parents and non-smoking parents, respectively. The median NNAL and cotinine concentrations were 1.4 (interquartile range [IQR]: 1.9) and 0.4 (IQR: 0.4) in children with parents who smoked, respectively; the corresponding values were 0.8 (IQR: 0.9) and 0.2 (IQR: 0.3) in children with parents who did not smoke, respectively(Table 1).
- The association between children's NNAL and cotinine concentrations and parents' smoking status after adjusting for all confounding variables. There was a significant association between parents' smoking status and children's NNAL ( $\beta=0.482$ , standard error [SE]=0.065,  $p<.0001$ ) and cotinine ( $\beta=0.472$ , SE=0.06,  $p<.0001$ ) concentrations; the association was stronger for smoking parents than for non-smoking parents(Table 2).

## RESULTS

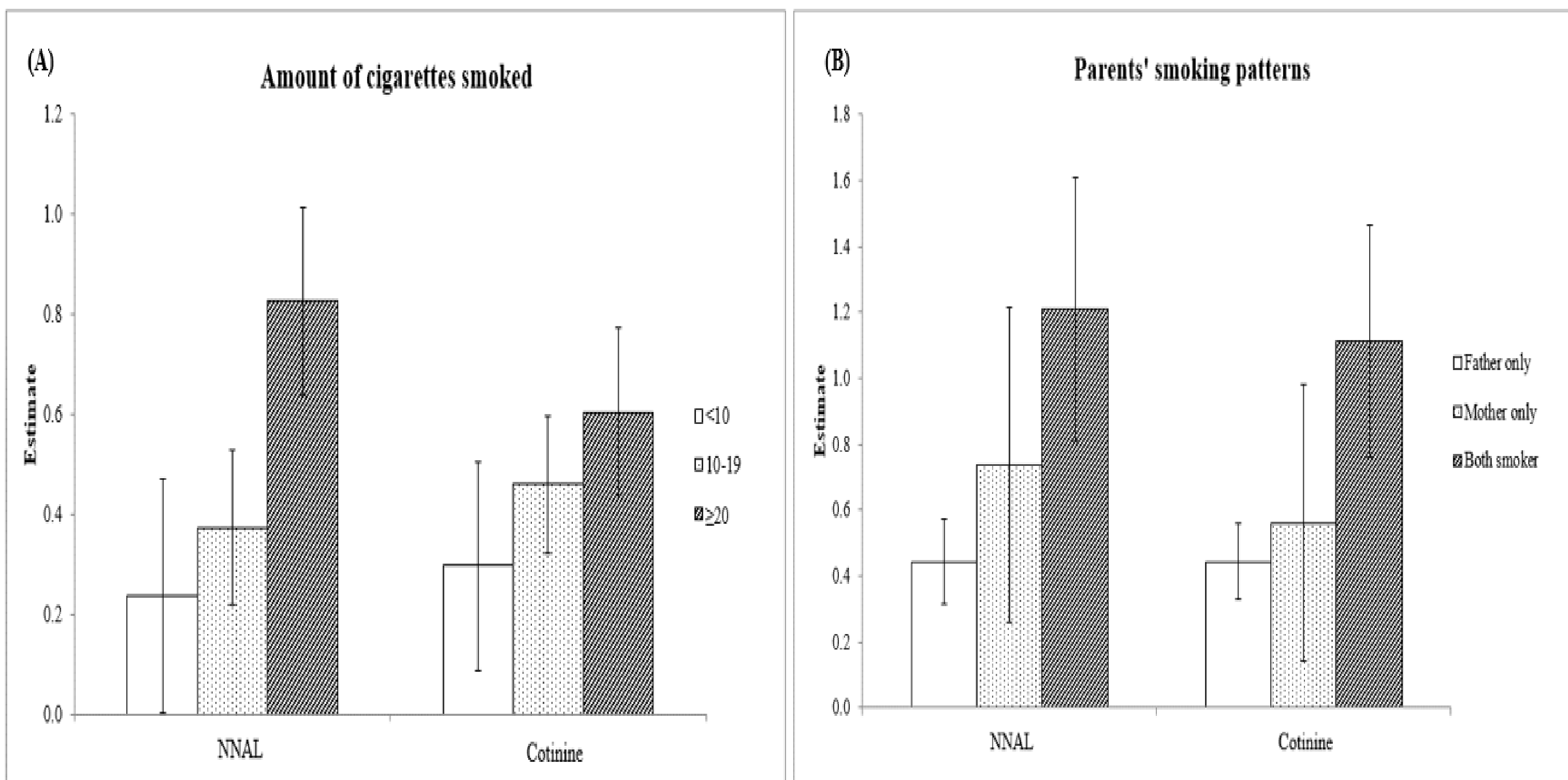


Fig 1. Association of the children's NNAL and cotinine concentrations with the parental amount of cigarettes smoked (A) and smoking pattern (B)

Adjusted for the children's sex, age, body mass index, secondhand smoke exposure (house), and secondhand smoke exposure (public) and for the parents' household income, type of housing, region, age, education level, private health insurance, drinking status, and year of evaluation. The reference group is the group of parents who are 'non-smokers.'

NNAL, N-(methylnitrosamino)-1-(3-pyridyl)-1-butanol

- The association between the amount of cigarettes smoked and smoking patterns and children's NNAL and cotinine concentrations. The higher the amount of cigarettes smoked, the higher were the children's NNAL and cotinine concentrations. Children of parents who smoked more than 20 cigarettes had the highest NNAL ( $\beta=0.825$ , SE=0.096,  $p<.0001$ ) and cotinine ( $\beta=0.604$ , SE=0.085,  $p<.0001$ ) concentrations. In addition, the children's NNAL and cotinine concentrations were also high when only the father (NNAL:  $\beta=0.444$ , SE=0.066,  $p<.0001$ ; cotinine:  $\beta=0.443$ , SE=0.058,  $p<.0001$ ) or mother (NNAL:  $\beta=0.738$ , SE=0.244,  $p=.0026$ ; cotinine:  $\beta=0.561$ , SE=0.241,  $p<.0090$ ) smoked. When both parents smoked, the NNAL ( $\beta=1.209$ , SE=0.204,  $p<.0001$ ) and cotinine ( $\beta=1.111$ , SE=0.179,  $p<.0001$ ) concentrations were the highest (Figure 1).

## DISCUSSION

- This study showed that at least one child out of two was living with a parent who smoked, and the NNAL and cotinine concentrations were higher in children of parents who were smokers. Through further subgroup analysis, we confirmed that the higher the amount of cigarettes smoked by both parents, the higher was the degree of SHS exposure in children.
- Additionally, although direct comparison is difficult, children's NNAL and cotinine concentrations were higher when only the mother smoked than when only the father smoked, compared to when both the parents did not smoke. In fact, in East Asian countries influenced by Confucianism, the smoking rate in men is higher than that in women. Thus, smoking abstinence by paternal figures is often chosen as the first strategy to reduce children's exposure to indirect smoking. However, for school-age children, time spent with the mother tends to be more than 2-fold longer than the time spent with the father; therefore, there is a need to consider smoking abstinence in mothers.
- The higher the amount of cigarettes smoked, the higher were the children's NNAL and cotinine concentrations. This is because the higher the amount of cigarettes smoked by the parents, the greater is the amount of harmful substances adhering to their clothes and skin; this indirectly affects the children.
- While adolescents tend to spend more time outside the home, school-age children spend a lot of time at home and stay close to their parents, suggesting that living with parents who smoke can be a strong predictor of increased exposure to substances included in cigarettes.
- Our research has showed that even after controlling SHS exposure at home and in public, many children still faced serious threats from their parents' smoking habits. This indicates that while the prohibition of smoking at home and in public show a highly negative correlation with children's exposure to SHS, these policies alone cannot fully protect their health from the adverse effects of SHS exposure due to parental smoking.
- Our study had several limitations to this study. First, we used cross-sectional data. Therefore, the cause and effect and the direction of relationships observed cannot be determined. Second, the results of this study were based on self-reported data. Therefore, the amount of cigarettes smoked may have been underestimated or overestimated, and some survey questions may be subject to recall bias. Third, despite our efforts to control for confounding factors, all covariates affecting NNAL and cotinine concentrations may not have been considered.
- However, despite these limitations, our research has important implications. The research results can be generalized because the research was conducted using the nationally representative and highly reliable KNHANES data, collected by national institutions. Our research findings also support the evidence presented in previous studies. This is because we targeted school-age children to minimize the bias related to smoking status and controlled for both SHS exposure in public and at home; these factors were not well considered in previous studies. Furthermore, while analyses based on cotinine measurements were commonly performed in previous studies, our research is meaningful in that we additionally analyzed the concentration of NNAL, which has a longer half-life.

## CONCLUSION

- In conclusion, children of parents who smoked are at a higher risk of exposure to SHS, implying that individual efforts to avoid smoking in the presence of children may be an insufficient alternative.
- Therefore, comprehensive national anti-smoking policies are required, such as the provision of anti-smoking services. furthermore, price and non-price-related policies are needed that can persuade parents to quit smoking, the provision of cessation treatment and counseling and an increase in cigarette prices and taxes are also required.