Pediatric Pulmonology 47:338–345 (2012)

Original Articles

Association Between Parental Smoking Behavior and Children's Respiratory Morbidity: 5-Year Study in an Urban City of South Korea

Jae Woo Jung, MD, PhD,[1,2] Young Su Ju, MD, PhD,[3] and Hye Ryun Kang, MD, PhD[1,2]***

Summary. Introduction: After intensive tobacco control efforts in recent decades, the prevalence of active smoking has decreased. However, the hazardous effect of indirect exposure to cigarette smoke is often underestimated, especially in children. We aimed to investigate the effect of parental smoking on the respiratory morbidity of the children of parents who smoke by evaluating the relationship between parental smoking behavior and children's respiratory symptoms. Methods: We conducted a cross-sectional follow-up study of 31,584 children aged 6–11 in an urban community in Anyang City, Korea. The children's parents were asked about their smoking status and completed questionnaires regarding their children's symptoms related to asthma and other upper or lower respiratory illnesses. Our analysis focused on a comparison of the frequency of respiratory and ocular symptoms according to parental smoking status, whether it was non-smoking (Non-S), indirect passive smoking (third-hand smoking, THS) or direct passive smoking (second-hand smoking, SHS). Results: The children with Non-S patients were 40.9%, THS group 40.6%, and SHS group 18.5%. THS group showed lower ORs for most respiratory symptoms when compared with those of SHS group, however, THS group revealed increased ORs compared with Non-S in cough-related symptoms. There was a linear trend in frequencies of cough and sputum-related symptoms according to the degree of exposure to cigarette smoke ($P < 0.05$). Conclusion: The prevalence of respiratory symptoms increased in children exposed to parental smoking including SHS and THS. To avoid the risk of respiratory and allergic disease by environmental tobacco smoke, absolute smoking cessation by parents is strongly recommended. **Pediatr Pulmonol. 2012; 47:338–345.**

Key words: asthma; children; smoking; questionnaires.

Funding source: Korean Healthcare Technology R&D Project, Ministry of Health and Welfare, Republic of Korea, Number: A102065.

[1]Department of Internal Medicine, Seoul National University College of Medicine, Seoul, Korea.

[2]Institute of Allergy and Clinical Immunology, Seoul National University Medical Research Center, Seoul, Korea.

[3]Department of Occupational and Environmental Medicine, Hallym University College of Medicine, Anyang, Korea.

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

*Correspondence to: Hye Ryun Kang, MD, PhD, Assistant Professor, Department of Internal Medicine, Seoul National University Hospital, 101 Daehangno, Chongno-gu, Seoul 110-744, Korea.
E-mail: helenmed@snu.ac.kr

**Correspondence to: Young Su Ju, MD, PhD, Professor, Hallym University College of Medicine; Director, Department of Occupational & Environmental Medicine, Hallym University Sacred Heart Hospital, 896 Pyongchon-Dong, Dongan-Gu, Anyang 431-070, Republic of Korea.
E-mail: zorro@hallym.ac.kr

Received 13 March 2011; Accepted 31 August 2011.

DOI 10.1002/ppul.21556
Published online 17 October 2011 in Wiley Online Library (wileyonlinelibrary.com).

INTRODUCTION

Environmental tobacco smoke (ETS) or passive smoking is one of the most frequent and commonly encountered types of indoor air pollutant. ETS produces harmful effects on the health of non-smokers, such as acute and chronic respiratory diseases, lung cancer, and cardiovascular diseases.[1] According to the 2006 Surgeon General's report, 126 million people were exposed to ETS and 50,000 people died of diseases related to ETS in the US per year.

ETS had been limited to second-hand smoke (SHS) in the past, but recently third-hand smoke (THS) has been classified as a type of ETS after numerous examples of supporting evidence. SHS is the direct inhalation of tobacco smoke, but THS refers a type of tobacco smoke which remains in the environment after the cigarette has been extinguished.[2] Owing to its invisibility, people are often unaware of its existence. However, THS occurs every day via the inhalation of noxious gases or minute particles from air contaminated by either previously smoked cigarettes or objects such as carpets, upholstery, fabrics, and other porous materials that absorb cigarette smoke.[2] THS also includes a mixture of semi-volatile compounds which adsorb or settle onto surfaces in indoor spaces and are later re-emitted into the air.[2]

Generally, children are more vulnerable to the deleterious effects of ETS than adults, and damage sustained in childhood can persist throughout one's lifetime, as children's respiratory and immunologic organ systems are still developing and their metabolic capacity is immature.[3,4] Infants and young children usually breathe more air relative to their body weight compared to adults and tend to put their hands, toys, and often non-food items possibly contaminated with THS into their mouths.[3] Therefore, children are not safe from, even at higher risk of, exposure to toxic air pollutants related to THS.[3]

There are numerous reports that support the deleterious effect of parental smoking on the health of their children. However, there are few studies that evaluate whether parental smoking in the THS form as well as the SHS form affect children's respiratory morbidity. We analyzed the association between parental smoking patterns and the prevalence of respiratory and ocular symptoms in children to determine the effect of both SHS and THS at home.

MATERIALS AND METHODS

Study Population

A cross-sectional and follow-up study was carried out on children aged 6–11 attending eight different regional elementary schools in Anyang, South Korea, a satellite city in the Seoul metropolitan area. Anyang is a part of a new planned town which is composed of relatively homogeneous young neighborhoods of similar socio-economic statuses. All eight schools included in the study were located within 3 km from Hallym University Hospital, the main regional medical center in this area.

Study subjects were 31,983 children whose parents responded to a questionnaire. Among them, 399 children whose parents (1.25%) did not complete the questionnaire were excluded from the analysis. Data collection took place over 5 years from 1999 to 2004. Informed consent was obtained from the parents of the study subjects.

Classification of Children by Parental Smoking Pattern

Patterns of passive smoke exposure were determined from questions about the parents' smoking behavior at home. Parents were first asked whether they smoked or not and if they smoked, whether or not they did so in the presence of their children. According to the answers to the above questions, children were classified into three different groups: the non-smoking (Non-S) group, the THS group, and the SHS group.

Contents of the Questionnaire

The questionnaire included the International Study of Asthma and Allergies in Childhood (ISAAC) core questionnaire on asthma[5] and additional questions related to cough, sputum, eye, and nose symptoms. The parents also completed questions on the basic demographic data and past medical history of children.

Statistical Analyses

All statistical analyses were conducted using SPSS (version 17.0, SPSS Inc., Chicago, IL). Continuous variables were depicted as the mean and standard deviation (SD). To adjust confounders, a multivariate logistic regression analysis involving age and sex was conducted to define the effect of smoking exposure, and the odds ratio (OR) and 95% confidence interval (CI) were calculated to evaluate the risk of each smoking exposure pattern. A chi-squared test for a linear trend was used to analyze the trends in the variables according to the degree of exposure to ETS. Additionally, a log-linear model was used to examine the linear association between serial changes in smoking patterns and respiratory symptom rates. $P < 0.05$ was considered as statistically significant in all statistical analyses.

RESULTS

Demographics of Study Subjects

The analyses were done for 31,584 children attending local elementary schools (Table 1). The mean age was 9.27 and proportion of males was 52.1%. The average body mass index was 17.64 ± 2.89 kg/m^2. A past history of pulmonary tuberculosis was noted in 0.2% of the study subjects. 14.9% had atopic dermatitis and 33.7% had nasal disease including sinusitis and rhinitis. Children who had been diagnosed with asthma anytime in their lives were 7.2% of the study subjects.

Among the study subjects, 12,908 children (40.9%) had Non-S parents, and 18,676 children (59.1%) had at least 1 smoking parent. The smoking group was further divided into THS (40.6%) and SHS (18.5%) according to the parental smoking pattern.

Smoking Status of Parents and Asthma-Related Symptoms in Children

When respiratory symptoms as surveyed with the ISAAC questionnaires were compared according to the parents' smoking pattern, the frequency of ''wheezing ever'' and ''asthma diagnosis ever'' were not different between the Non-S group and the passive smoking group (Table 2). However, the frequency levels of ''wheezing within recent 1 year'' and ''sleep disturbance by wheezing within 1 year'' were increased in the SHS compared to the Non-S groups ($P = 0.001$, $P < 0.0001$, respectively). The SHS group also showed a higher rate of ''wheezing after exercise within 1 year'' and ''nocturnal cough'' compared to the Non-S group ($P < 0.0001$ and $P < 0.0001$, respectively). Except for ''asthma diagnosis ever'' and ''school absence due to dyspnea or cough,'' most items on the ISAAC questionnaire showed a slight but statistically significant difference between the SHS and THS groups ($P < 0.05$). Comparing Non-S and THS, the difference was not remarkable, except for ''nocturnal cough,'' which was significantly higher in the THS group compared to the Non-S group ($P < 0.0001$).

Smoking Status of Parents and Other Lower Respiratory Symptoms in Children

The SHS group showed the highest prevalence of cough-related symptoms among the three groups in all questions (Table 3). The THS group also had higher frequencies compared to the Non-S group on the items of ''dry cough'' and ''5-continuous cough'' ($P = 0.024$, $P = 0.011$, respectively). When the two passive smoking groups were compared, most cough-related symptoms were more prevalent in the SHS group than the THS group.

All three sputum-related symptoms were significantly more prevalent in the SHS group then they were in either Non-S or THS. However, sputum-related items showed no significant differences between the Non-S group and the THS group.

Smoking Status of Parents and Upper Respiratory and Ocular Symptoms in Children

All four nasal symptoms except nasal itching were significantly more prevalent in the SHS group compared to the Non-S group (Table 4). There was no difference between the Non-S group and the THS group in terms of nasal symptoms. Compared in terms of ETS patterns, significantly more children in the SHS group complained of symptoms of ''watery rhinorrhea,'' ''sneezing,'' and ''nasal obstruction'' than the THS group ($P < 0.0001$, $P < 0.0001$, and $P = 0.046$, respectively). In the categories of ''eye irritation'' and ''lacrimation,'' the SHS group showed more frequent morbidity of those symptoms compared to Non-S and THS ($P < 0.01$).

TABLE 1—Study Populations Included in the Study

Year	Non-S	THS	SHS	Total
N	12,908 (40.9%)	12,818 (40.6%)	5,858 (18.5%)	31,584
Age	9.35 ± 1.80	9.11 ± 1.79	9.47 ± 2.29	9.27 ± 1.91
Male (%)	52.3%	51.9%	52.3%	52.1%
Height (cm)	136.99 ± 11.64	135.69 ± 11.87	137.29 ± 11.69	136.52 ± 11.76
Weight (kg)	32.96 ± 9.14	31.92 ± 9.01	33.06 ± 9.53	32.48 ± 9.17
Body mass index (kg/m^2)	17.64 ± 2.84	17.56 ± 2.85	17.84 ± 3.06	17.64 ± 2.89
Past medical history				
Pulmonary tuberculosis	0.2%	0.2%	0.2%	0.2%
Atopic dermatitis	14.6%	15.3%	14.5%	14.9%
Nasal disease	33.9%	34.1%	32.3%	33.7%
Asthma	7.3%	7.4%	7.9%	7.2%

Non-S, non-smoking group; THS, third-hand smoking group; SHS, second-hand smoking group.

TABLE 2—ISAAC Questionnaires Used in the Study

	Frequency (%)			Chi-square for linear trend	*P*-value	OR (95% CI)[a]		
	Non-S	THS	SHS			THS versus Non-S	SHS versus Non-S	SHS versus THS
Wheezing ever	10.5	10.4	11.3	2.1598	0.1417	0.968 (0.892–1.051)	1.050 (0.998–1.104)	1.138 (1.028–1.259)
Wheezing within 1 year	4.1	3.8	5.2	7.1445	0.0075	0.907 (0.797–1.031)	1.136 (1.055–1.223)	1.426 (1.226–1.658)
Sleep disturbance d/t wheezing within 1 year	1.7	1.6	2.5	9.2273	0.0024	0.865 (0.710–1.053)	1.214 (1.090–1.352)	1.707 (1.368–2.130)
Limit speech d/t wheezing within 1 year	1.2	1	1.4	0.6614	0.4161	0.826 (0.646–1.057)	1.101 (0.958–1.265)	1.491 (1.117–1.989)
Wheezing after exercise within 1 year	2.7	2.5	4	16.9571	<0.0001	0.970 (0.827–1.137)	1.238 (1.135–1.351)	1.593 (1.335–1.900)
Nocturnal cough within 1 year	9.6	11.6	13.4	63.1121	<0.0001	1.196 (1.101–1.299)	1.218 (1.160–1.280)	1.241 (1.127–1.366)
Asthma diagnosis ever	7.3	7.4	7.9	1.6702	0.1962	1.015 (0.921–1.119)	1.049 (0.988–1.114)	1.094 (0.970–1.234)
Treatment for asthma within 1 year	2.3	2.3	2.6	1.1318	0.2874	0.976 (0.824–1.156)	1.087 (0.982–1.203)	1.231 (1.004–1.511)
Absence due to dyspnea or cough	0.8	0.9	1.1	3.1587	0.0755	1.126 (0.857–1.480)	1.164 (0.991–1.369)	1.220 (0.889–1.674)

Non-S, non-smoking group; THS, third-hand smoking group; SHS, second-hand smoking group.
[a]Multivariate logistic regression analysis involving age and sex.

TABLE 3—Questionnaires for Lower Respiratory Symptoms

	Frequency (%)			Chi-square for linear trend	*P*-value	OR (95% CI)[a]		
	Non-S	THS	SHS			THS versus Non-S	SHS versus Non-S	SHS versus THS
Cough persists >3 weeks within 3 years	6	6.6	7.6	14.1734	0.0002	1.056 (0.945–1.179)	1.130 (1.059–1.207)	1.191 (1.047–1.356)
Dry cough	7.9	8.9	11	43.7429	<0.0001	1.111 (1.014–1.217)	1.210 (1.147–1.277)	1.314 (1.182–1.460)
5-Continuous cough	3	3.6	3.8	9.6823	0.0019	1.203 (1.043–1.388)	1.148 (1.053–1.252)	1.093 (0.923–1.293)
Cough persists >3 months/year	2.2	2.6	3.1	14.6731	0.0001	1.156 (0.981–1.362)	1.209 (1.098–1.332)	1.249 (1.035–1.508)
Frequent cough on daytime	7.2	7.2	9.5	12.954	0.0003	1.136 (0.989–1.305)	1.174 (1.082–1.274)	1.203 (1.024–1.412)
Cough on waking-up	7	7	7.4	1.0908	0.2963	0.973 (0.880–1.075)	1.039 (0.976–1.105)	1.107 (0.978–1.254)
Sputum for >3 months/year	2.2	2.1	2.9	7.5535	0.006	0.969 (0.814–1.153)	1.150 (1.042–1.270)	1.370 (1.121–1.673)
Frequent sputum expectoration	3.3	3.6	4.4	18.7739	<0.0001	1.000 (0.990–1.112)	1.153 (1.086–1.224)	1.335 (1.184–1.506)
Sputum on waking-up	5.8	6.1	6.9	6.8603	0.0088	1.036 (0.921–1.166)	1.099 (1.026–1.177)	1.169 (1.019–1.341)

Non-S, non-smoking group; THS, third-hand smoking group; SHS, second-hand smoking group.
[a]Multivariate logistic regression analysis involving age and sex.

TABLE 4— Questionnaires for Upper Respiratory and Eye Symptoms

	Frequency (%)			Chi-square for linear trend	*P*-value	OR (95% CI)[a]		
	Non-S	THS	SHS			THS versus Non-S	SHS versus Non-S	SHS versus THS
Watery rhinorrhea	12.5	12.5	14.9	14.6462	0.0001	0.999 (0.926–1.079)	1.108 (1.058–1.160)	1.226 (1.118–1.344)
Nasal itching	11.3	10.5	11.4	0.223	0.6367	0.938 (0.865–1.018)	1.012 (0.962–1.064)	1.087 (0.982–1.203)
Sneezing	15.2	14.6	17.1	7.0536	0.0079	0.983 (0.916–1.056)	1.081 (1.036–1.129)	1.186 (1.087–1.293)
Nasal obstruction	31.2	32	33.3	7.9684	0.0048	1.035 (0.980–1.093)	1.053 (1.018–1.090)	1.072 (1.001–1.148)
Postnasal drip	18.3	18.7	19.9	5.750	0.0165	1.022 (0.954–1.094)	1.057 (1.012–1.103)	1.080 (0.990–1.177)
Eye itching	6.9	6.8	7.6	1.4584	0.2272	0.983 (0.890–1.085)	1.041 (0.980–1.107)	1.100 (0.973–1.243)
Eye irritation	1.7	1.4	2.4	4.6918	0.0303	0.810 (0.661–0.993)	1.161 (1.041–1.294)	1.657 (1.316–2.088)
Lacrimation	2.5	2.2	3.8	15.7058	<0.0001	0.895 (0.757–1.057)	1.243 (1.137–1.358)	1.750 (1.455–2.103)

Non-S, non-smoking group; THS, third-hand smoking group; SHS, second-hand smoking group.
[a]Multivariate logistic regression analysis involving age and sex.

Linear Trend of Children's Symptom Prevalence According to the Degree of ETS Exposure

A linear trend was observed in the prevalence of asthma symptoms, respiratory symptoms, and eye symptoms according to the degree of exposure to ETS from their parents (Tables 2–4). When taking the degree of exposure to smoking into consideration, we noted a linear trend in the prevalence of asthma-related symptoms (Table 2). There was gradual increment in the frequencies of "nocturnal cough within 1 year" according to the degree of exposure to cigarette smoke. This linear pattern is more prominent in lower respiratory symptoms, especially the cough categories, except for "cough on waking-up" (Table 3). The sputum-related questions also showed the same tendency. These linear patterns were also observed in upper respiratory and ocular symptoms but were less prominent compared to asthma-related symptoms or lower respiratory symptoms (Table 4).

Time-Series Analysis Between Parental Smoking and Cough Morbidity Among Children

The proportions of the Non-S group and the THS group have increased in recent years, while the SHS group has shown meaningful decreases annually (Fig. 1A). Relative risks (RR) were 0.9418 (95% CI: 0.9327–0.9510) in total smoking and 0.8310 (95% CI: 0.8151–0.8471) in SHS annually.

With a decline in the parents' smoking rate, the frequency of respiratory symptoms in children also decreased gradually. This trend was more pronounced in the cough-related questions. For example, the frequency of "nocturnal cough" showed a gradual reduction, and RR was 0.961 (95% CI: 0.942–0.98; Fig. 1B). "Dry cough" and "cough persisting longer than 3 weeks" also showed a serial decrement similar to the decreasing pattern of the total smoking rate and the SHS rate in the parents (RR = 0.953, 95% CI: 0.932–0.974 and RR = 0.921, 95% CI: 0.895–0.946, respectively; Fig. 1C and D).

DISCUSSION

ETS is composed of side stream smoke from the end of a cigarette as the cigarette burns (80–90%) and mainstream smoke exhaled by the smoker (10–20%). The gas itself and the particulate matter consist of a complex and dynamic mixture of more than 4,000 substances collectively classified as an indoor air pollutant with numerous human carcinogens.[2] According to data from the early 1990s in the USA, 38% of children were exposed to ETS in indoor spaces.[4] The smoking rate of parents of children in South Korea was still high, at 60% in the early 2000s, and substantial numbers of

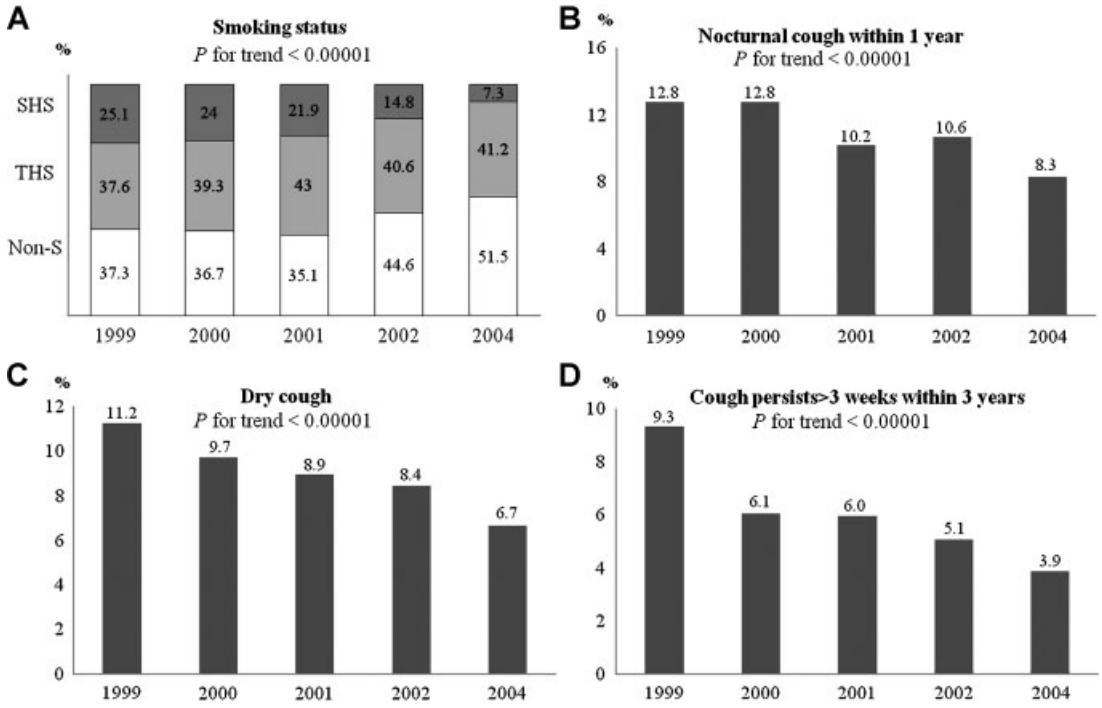

Fig. 1. Parental smoking behavior and prevalence of cough: serial trend. (A) Serial trend of smoking status, (B) nocturnal cough with 1 year, (C) dry cough, (D) cough persistent more than 3 weeks within 3 years. Non-S, non-smoking group; THS, third-hand smoking group; SHS, second-hand smoking group.

Korean children are assumed to be at risk of ETS at home at present.[6]

Childhood is a period of rapid growth of both the respiratory and immune systems. Therefore, children are more vulnerable to the detrimental effects of ETS than adults.[7] When children are exposed to SHS, lung functions such as the forced expiratory volume in 1 sec (FEV_1) decline. This decrement can persist into adulthood.[8] Cunningham et al.[9] reported that maternal smoking during pregnancy and ETS exposure after birth causes an 8.1% deficit of $FEF_{25-75\%}$ and a 2.0% deficit of FEV_1.

A close relationship has been observed between smoking and asthma. In a meta-regression analysis by Vork et al., SHS showed a significant association with ever or current asthma.[10] In particular, the longer children with atopy were exposed to their parent's smoking, the more enhanced airway hyper-responsiveness they had. SHS not only increases the incidence of asthma but also worsens pre-existing asthma; it increases the number of out-patient visits, lowers the response to medicine, and aggravates asthma by increasing the chance of infection in either the upper or lower respiratory system.[11] Asthmatics showed a greater decline of their lung function by SHS compared to subjects without asthma.[12]

Several hypotheses have been conceived about the relationship between exposure to cigarette smoke and children's allergic diseases. Exposure to tobacco during childhood is known to promote allergic sensitization in a child,[13] and this phenomenon can be explained by the enhanced permeability of respiratory membranes to allergens and antigen-presenting cell signaling modified by the noxious gas from cigarettes. In fact, parental smoking is reported to increase serum IgE, allergen-specific IgE, and the likelihood of the development of atopy in children.[14]

In the present study, children in the SHS group showed the highest symptom prevalence in all items on the ISAAC questionnaires. Most of them were statistically significant. In particular, the SHS group showed a higher prevalence by 40% or more of ''sleep disturbance due to wheezing within 1 year,'' ''wheezing after exercise within 1 year,'' and ''nocturnal cough within 1 year'' compared to the Non-S group. ISAAC questionnaires are widely used tools for investigating the prevalence of asthma in large populations and are known to show good diagnostic performance.[15] Therefore, the results here show an increased prevalence of asthma in children exposed to SHS. In addition to the ISAAC questionnaires, other non-specific lower respiratory symptoms were also more prevalent in the SHS group.

In the SHS group, the proportion of children who experienced ''dry cough'' and ''cough persisting longer than 3 months/year'' were about 40% larger compared to the Non-S group.

ETS can affect the upper as well the lower respiratory tracts. In one cohort study, home SHS exposure raised the risk of symptoms of allergic rhinitis and increased the manifestation of rhinoconjunctivitis in children.[16] ETS also causes the occurrence of nasal obstructions and hinders mucociliary clearance, even in non-allergic rhinitis.[17] In this study, we surveyed the categories related to rhinoconjunctivitis and found a significantly higher rate of naso-ocular symptoms in the SHS group than the Non-S group, as expected. In the case of ''lacrimation,'' the SHS group showed a 52% higher frequency than the Non-S group.

In addition to the health hazards of SHS, investigations of the effect of THS have increased.[18–20] In contrast to SHS, which is directly caused by exposure to tobacco smoke,[2] THS occurs by exposure to hazardous substances that have accumulated in the air or on surfaces when people enter a space previously exposed to cigarette smoke. Many chemicals and fine dust found in cigarette smoke can adhere to walls, furniture, clothes, household fixtures, and on the body surface of smokers. The gas and dust deposited on the skin or on clothing can be re-emitted into the air, and the residual contaminants in the lung and airways of a smoker can come back during exhalation, even when active smoking occurred in a remote location in the recent past.[6,21,22] Therefore, it is important to recognize that the risk of THS still exists everywhere, even after the cigarette has been extinguished.

THS is composed of mainly nicotine, phenol, cresols, formaldehyde, 3-ethenylpyridine, naphthalene, and tobacco-specific nitrosamines.[23–25] Some components of THS are not found in SHS.[26] THS exposure can be even more harmful, as its effects last longer than those of SHS and because THS particles can be ingested by mouth and inhaled as an aerosol.[21]

In fact, a difference was noted in the nicotine concentration in the household dust between families with smokers and families without smokers and a close relationship was observed between the intensity of smoking and the level of nicotine in the household dust.[19] The nicotine level in the hair and serum was high not only in children whose parents smoked inside but also in children whose parents smoke outside.[18,20] Recently, another study revealed that THS can have a deleterious effect on human beings after combining with other indoor materials over time and transforming into a more toxic carcinogen such as tobacco-specific nitrosamines from the reaction of adsorbed nicotine with nitrous acid.[23]

In the present study, the THS group did not show a significant increase of the frequency of wheezing, sputum, upper respiratory, or ocular symptoms, but the cough symptoms of ''nocturnal cough,'' ''dry cough,'' and ''5 continuous cough'' were more prevalent compared to the Non-S group. These findings imply that THS can affect children's lower respiratory systems to some degree, even when it does not increase the prevalence of asthma.

We also noted a linear trend in the symptom frequency among the three groups according to the degree of ETS exposure. Various symptoms, including the cough, sputum, and nasal symptom categories, showed the highest incidences in the SHS group, with the next highest being the THS and Non-S groups in that order. These findings suggest THS has less harmful effects than SHS but still has a substantial effect on the respiratory system of children.

During the study period, as the rate of parent's active smoking gradually decreased, children's symptoms such as coughing showed a meaningful decrease. Although we cannot ascertain a direct cause–effect relationship, it is possible that decreased exposure to ETS contributes to this decline in children's respiratory symptoms.

The major limitation of this study was its failure to collect data about sources of ETS other than parents; for instance, exposure during outdoor activities or to other family members was not taken into account. A second limitation is the lack of information such as the number of cigarettes smoked or the duration of exposure. We also noted limitations when evaluating other confounding factors, such as social economic status, prenatal exposure to smoking or other allergens, and the presence of a pet. This study is based on a questionnaire pertaining to symptoms and does not contain objective evidence regarding the presence of asthma, such as variations of the lung function or the existence of airway responsiveness. Nonetheless, this study presents preliminary large-scale epidemiologic evidence of the deleterious effects of not only SHS but also THS exposure by parents on their children's respiratory morbidity. Therefore, our results along with all other supporting evidence clearly demonstrates the urgent need for larger public campaigns to inform parents of newborns and children that complete smoking cessation is required in order to protect their children fully from the noxious effects of cigarette smoke.

CONCLUSION

This study suggests that parents' smoking behavior pattern may affect the respiratory morbidity of their children and even THS exposure can increase cough morbidity. Therefore, in order to protect children fully

from respiratory illness caused by exposure to ETS, complete cessation of smoking by parents is necessary.

REFERENCES

1. Moritsugu KP. The 2006 Report of the Surgeon General: the health consequences of involuntary exposure to tobacco smoke. Am J Prev Med 2007;32:542–543.
2. Matt GE, Quintana PJ, Zakarian JM, Fortmann AL, Chatfield DA, Hoh E, Uribe AM, Hovell AF. When smokers move out and non-smokers move in: residential thirdhand smoke pollution and exposure. Tob Control 2011;20:e1.
3. Roberts JW, Wallace LA, Camann DE, Dickey P, Gilbert SG, Lewis RG, Takaro TK. Monitoring and reducing exposure of infants to pollutants in house dust. Rev Environ Contam Toxicol 2009;201:1–39.
4. Gergen PJ, Fowler JA, Maurer KR, Davis WW, Overpeck MD. The burden of environmental tobacco smoke exposure on the respiratory health of children 2 months through 5 years of age in the United States: Third National Health and Nutrition Examination Survey, 1988 to 1994. Pediatrics 1998;101:E8.
5. Asher MI, Keil U, Anderson HR, Beasley R, Crane J, Martinez F, Mitchell EA, Pearce N, Sibbald B, Stewart AW, Strachan D, Weiland SK, Williams HC. International Study of Asthma and Allergies in Childhood (ISAAC): rationale and methods. Eur Respir J 1995;8:483–491.
6. Jang AS, Choi IS, Lee S, Nam HS, Kweon SS, Son MH, Lee JH, Park SW, Kim DJ, Uh ST, Kim YH, Park CS. The effect of passive smoking on asthma symptoms, atopy, and airway hyperresponsiveness in schoolchildren. J Korean Med Sci 2004;19: 214–217.
7. Janson C, Chinn S, Jarvis D, Zock JP, Toren K, Burney P. Effect of passive smoking on respiratory symptoms, bronchial responsiveness, lung function, and total serum IgE in the European Community Respiratory Health Survey: a cross-sectional study. Lancet 2001;358:2103–2109.
8. Rasmussen F, Taylor DR, Flannery EM, Cowan JO, Greene JM, Herbison GP, Sears MR. Risk factors for airway remodeling in asthma manifested by a low postbronchodilator FEV_1/vital capacity ratio: a longitudinal population study from childhood to adulthood. Am J Respir Crit Care Med 2002;165:1480–1488.
9. Cunningham J, Dockery DW, Gold DR, Speizer FE. Racial differences in the association between maternal smoking during pregnancy and lung function in children. Am J Respir Crit Care Med 1995;152:565–569.
10. Vork KL, Broadwin RL, Blaisdell RJ. Developing asthma in childhood from exposure to secondhand tobacco smoke: insights from a meta-regression. Environ Health Perspect 2007;115: 1394–1400.
11. Cook DG, Strachan DP. Health effects of passive smoking. 3. Parental smoking and prevalence of respiratory symptoms and asthma in school age children. Thorax 1997;52:1081–1094.
12. Landau LI. Tobacco smoke exposure and tracking of lung function into adult life. Paediatr Respir Rev 2008;9:39–43; quiz-4.
13. Annesi-Maesano I, Oryszczyn MP, Raherison C, Kopferschmitt C, Pauli G, Taytard A, Tunon de Lara M, Vervloet D, Charpin D. Increased prevalence of asthma and allied diseases among active adolescent tobacco smokers after controlling for passive smoking exposure. A cause for concern? Clin Exp Allergy 2004;34:1017–1023.
14. Ownby DR, McCullough J. Passive exposure to cigarette smoke does not increase allergic sensitization in children. J Allergy Clin Immunol 1988;82:634–638.
15. Lukrafka JL, Fuchs SC, Moreira LB, Picon RV, Fischer GB, Fuchs FD. Performance of the ISAAC questionnaire to establish the prevalence of asthma in adolescents: a population-based study. J Asthma 2010;47:166–169.
16. Zuraimi MS, Tham KW, Chew FT, Ooi PL, David K. Home exposures to environmental tobacco smoke and allergic symptoms among young children in Singapore. Int Arch Allergy Immunol 2008;146:57–65.
17. Shusterman D. Environmental nonallergic rhinitis. Clin Allergy Immunol 2007;19:249–266.
18. Nelson R. Smoking outside still causes second-hand smoke exposure to children. Lancet 2002;359:1675.
19. Hein HO, Suadicani P, Skov P, Gyntelberg F. Indoor dust exposure: an unnoticed aspect of involuntary smoking. Arch Environ Health 1991;46:98–101.
20. Matt GE, Quintana PJ, Hovell MF, Bernert JT, Song S, Novianti N, Juarez T, Floro J, Gehrman C, Garcia M, Larson S. Households contaminated by environmental tobacco smoke: sources of infant exposures. Tob Control 2004;13:29–37.
21. Invernizzi G, Ruprecht A, De Marco C, Paredi P, Boffi R. Residual tobacco smoke: measurement of its washout time in the lung and of its contribution to environmental tobacco smoke. Tob Control 2007;16:29–33.
22. Gordon SM, Wallace LA, Brinkman MC, Callahan PJ, Kenny DV. Volatile organic compounds as breath biomarkers for active and passive smoking. Environ Health Perspect 2002;110:689–698.
23. Sleiman M, Gundel LA, Pankow JF, Jacob P III, Singer BC, Destaillats H. Formation of carcinogens indoors by surface-mediated reactions of nicotine with nitrous acid, leading to potential thirdhand smoke hazards. Proc Natl Acad Sci USA 2010;107:6576–6581.
24. Singer BC, Wenzel TP. Estimated emission reductions from California's enhanced Smog Check program. Environ Sci Technol 2003;37:2588–2595.
25. Destaillats H, Singer BC, Lee SK, Gundel LA. Effect of ozone on nicotine desorption from model surfaces: evidence for heterogeneous chemistry. Environ Sci Technol 2006;40:1799–1805.
26. Matt GE, Quintana PJ, Destaillats H, Gundel LA, Sleiman M, Singer BC, Jacob Iii P, Benowitz N, Winickoff JP, Rehan V, Talbot P, Schick S, Samet J, Wang Y, Hang B, Martins-Green M, Pankow JF, Hovell MF. Thirdhand tobacco smoke: emerging evidence and arguments for a Multidisciplinary Research Agenda. Environ Health Perspect 2011;119:1218–1226.