

The Effect of Passive Smoking on Asthma Symptoms, Atopy, and Airway Hyperresponsiveness in Schoolchildren

Passive smoking is a major cause of respiratory morbidity, and is associated with increased bronchial responsiveness in children. To evaluate the effect of smoking by a parent on asthma symptoms, atopy, and airway hyperresponsiveness (AHR), we conducted a cross-sectional survey of 503 schoolchildren that involved questionnaires, spirometry, allergy testing, and a bronchial challenge test. If the PC₂₀ methacholine was less than 16 mg/mL, the subject was considered to have AHR. The prevalence of a parent who smoked was 68.7%. The prevalence of AHR was 45.0%. The sensitization rate to common inhalant allergens was 32.6%. Nasal symptoms such as rhinorrhea, sneezing, nasal itching, and nasal obstruction were present in 42.7%. Asthma symptoms such as cough and wheezing were present in 55.4%. The asthma symptoms were significantly more prevalent in children who had a parent who smoked than in those whose parents did not. The nasal symptoms, atopy, and AHR did not differ according to whether a parent smoked. In a multiple logistic regression model, the asthma symptoms and atopy were independently associated with AHR, when adjusted for confounding variables. Passive smoking contributed to asthma symptoms in schoolchildren and was not an independent risk factor of airway hyperresponsiveness in an epidemiological survey.

Key Words : Tobacco Smoke Pollution; Bronchial Asthma; Bronchial Hyperreactivity; Child

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INTRODUCTION

The prevalence of asthma continues to rise. A number of environmental factors including air pollution, cigarette smoking, and allergen exposure have been proposed to explain the changes in the prevalence of asthma (1). The prevalence of asthma has increased in Korea from 5.7% in 1980 to 10.0% in 1995 (2).

Passive smoking is a major cause of respiratory morbidity in children. High levels of parental smoking at home are associated with a reduction in health care contacts for asthma. Children with asthma who have parents who smoke heavily may not be receiving adequate management (3). The smoking rate among parents of asthmatic children is significantly higher than among parents of normal children (4).

An increase in the total IgE in relation to active smoking has been demonstrated in the general population. An increase in IgE was also observed in first-degree relatives exposed to passive smoking (5).

Passive smoking increases the likelihood of experiencing respiratory symptoms and is associated with increased bronch-

ial responsiveness. Decreasing involuntary exposure to tobacco smoke in the community, especially in the workplace, should improve respiratory health (6).

To investigate the effect of exposure to parental cigarette smoking on asthma symptoms, we gave schoolchildren questionnaires that asked about asthma symptoms, atopy, and airway hyperresponsiveness.

MATERIAL AND METHODS

Study population and Questionnaire

All children were 10-12 yr of the age. The question on respiratory and allergic disorders included those developed for the International Study of Asthma and Allergies in Childhood (ISAAC). The focus of this survey was on responses to the ISAAC questions on "wheeze in the last 12 months", "number of wheezing attacks in the last 12 months", and on "sleep-disturbing" and "speech-limiting" wheezing in the last 12 months. Symptoms of allergic rhinitis ("sneezing or runny,

Table 1. Characteristics of school children

	Parent smoking (+)	Parent smoking (-)
Age (yr)	10.96±0.04	10.90±0.06
Sex (M/F)	162/188	83/70
Asthma symptoms (%)	58.5	48.3
Nose symptoms (%)	63.7	58.8
Atopy (%)	31.7	34.6
PC ₂₀ (mg/mL)	16.7±0.54	16.6±0.79
FEV ₁ (%pred)	102.8±0.46	104.5±0.06
FVC (%pred)	105.8±0.38	103.8±0.46
FEV ₁ /FVC	97.7±0.4	98.6±0.6

% pred, group mean value of individual percentage predicted lung function parameter; FEV₁, forced expiratory volume in one second; FVC, forced vital capacity; M, male; F, female.

or blocked nose without a cold") were assessed (7). In addition, a question on morning cough as a symptom of nonspecific airway irritation was asked ("Did you frequently cough in the morning right after waking up in the last 12 months?"). Also a question on smoking status of parents was asked. A total of 503 schoolchildren was enrolled in this study (Table 1). No subjects took any drugs within 72 hr of tests such as anti-histamine, cromolyn, theophylline, sympathomimetics, which could interfere with the performance of skin tests. The all guardians signed informed consent before the study.

Pulmonary function study

Spirometry was performed with SensorMedics 2200 spirometer (Cardiopulmonary care company™, Yorba Linda, California, U.S.A.). Baseline measurements of VC and FEV₁ were selected according to American Thoracic Society criteria (8) and reference values were taken from Choi et al. (9).

Allergy skin prick test

Allergy skin prick tests were performed with eleven common allergen extracts [*Dermatophagoides farinae*, *Dermatophagoides pteromyssinus*, *Aspergillus* spp, alder, birch, hazel, rye, timothy, mugwort, ragweed, *Blatella germanica*; Allergopharma Co, Reinbek, Germany], and histamine (1 mg/mL) and saline as a positive control and a negative control. The reactions were read 15 min later. When the wheal size was equal or greater than that of histamine (positive control), it was read as 3+. Atopy was defined as a reactor who showed ≥ 3+ response to one or more allergens on skin prick tests (10).

Airway hyperresponsiveness

Methacholine challenge tests were carried out by a modified method described by Chai et al. (11). Concentrations of 0.075, 1.25, 2.5, 5, 10 and 25 mg/mL methacholine were prepared by dilution with buffered saline. A Micro-dosimeter (S&M Instrument Co, Doylestown, PA) was used to deliver

Table 2. Multivariate logistic regression on airway hyperresponsiveness

Variables	Odd ratios	95% confidence interval
Age	1.147	0.912-1.451
Sex	1.050	0.727-1.494
Status of parent's smoking	1.136	0.783-1.642
Asthma symptoms*	1.895	1.213-3.801
Family history ¹	0.865	0.529-1.521
FEV ₁ % predicted	0.980	0.958-1.114
Atopy*	1.657	1.332-2.956

Stepwise logistic regression was used to select factors significantly associated AHR risk (* $p < 0.05$). ¹defined by having or not parent's allergic diseases.

the aerosol generated by a DeVilbiss 646 nebulizer. Subjects inhaled 5 breaths of increasing concentrations of methacholine until FEV₁ fell by more than 20% of its basal value or the highest concentration was reached. The largest value of triplicate FEV₁ at 30 or 90 or 180 sec after each inhalation was adopted for analysis. If PC₂₀ was less than 16 mg/mL, a subject was considered to have AHR.

Statistical analyses

All data were analyzed using the SPSS version 7.5 for Windows. Each biochemical assays were repeated at least two times. Data are expressed as mean ± standard error of mean (SEM). Statistical analysis was performed by Student's t test, Mann-Whitney U test. In multivariate analysis, logistic regression was performed with a stepwise selection method with $p < 0.05$ for entry into the model. Atopy and asthma symptoms were treated as binary outcome variables in logistic regression analysis. A p -value of < 0.05 considered to be significant.

RESULTS

The characteristics of children of school age were shown in Table 1. The prevalence of parent's smoking was 68.7% (346/503). The prevalence of AHR was 45.0% (227/503). The sensitization rate (skin prick test A/H ≥ 3+) to common inhalant allergens were 32.6% (164/503). Nasal symptoms such as rhinorrhea, sneezing, nasal itching, and nasal obstruction were present in 42.7%. Asthma symptoms such as coughing, wheezing, and chest tightness were present in 55.4%. The asthma symptoms were significantly more prevalent in children who had a parent who smoked than in those whose parents did not [(205/350 (58.5%) vs. 74/157 (47.1%), $p = 0.016$]. The nasal symptoms, atopy, and AHR did not differ according to whether a parent smoked. In a multiple logistic regression model, the asthma symptoms and atopy were independently associated with AHR, when adjusted for confounding variables (OR = 1.895; 95% CI = 1.213-3.801, $p = 0.046$; OR = 1.657; 95% CI = 1.332-2.956, $p = 0.029$, Table 2).

DISCUSSION

This epidemiological survey demonstrated that passive smoking contributes to asthma symptoms and is not an independent risk factor of AHR in schoolchildren.

The prevalence of asthma has increased dramatically in recent years, especially among children. In our study, the prevalence of a PC₂₀ less than 16 mg/mL was 45.0%, which represents the AHR prevalence among schoolchildren according to the American Thoracic Society criteria (12). Long-term follow up to clarify the association between AHR and asthma in schoolchildren is needed. The factors leading to the increase in asthma and allergies have not been identified, although exposure related to general changes in the domestic environment is likely involved. Environmental factors play an important role in the development and manifestation of allergic conditions in genetically predisposed subjects (13). Passive smoking increases the risk of allergic sensitization. It also increases the frequency and severity of symptoms in children with asthma (14). In this study, the observed prevalence of atopy in schoolchildren was 32.6%, and atopy was an independent risk factor in AHR, indicating that atopy plays an important role in the development of asthma in schoolchildren. We did not observe an effect of parental smoking on atopy in schoolchildren. Exposure to tobacco smoke has been associated with a decline in peak flow and with increases in respiratory symptoms and bronchodilator use in asthmatic children (15). In most of the families that we studied in which a parent smoked, the father smoked (97%). Although parental smoking did not affect AHR, it was associated with asthma symptoms. Whether a parent smoked had no effect on AHR in children. Therefore, our data suggest that parental smoking does not cause asthma, but aggravates it.

The risk of asthma in Nepalese children was lower in subjects exposed to cattle kept inside the house and higher in subjects exposed to passive smoking and indoor use of smoky fuels (16). Another study found that the risk factors for childhood asthma increased with increasing numbers of smokers and the total minutes spent smoking by relatives in front of both the child and the mother while she was pregnant with the child (17). We did not measure the total minutes or amount of smoking by parents in front of children. Further study is needed to clarify the effects of exposure time and the amount of passive smoking on AHR. A greater baseline exposure to environmental tobacco smoke has also been related with poorer physical health and asthma-specific quality-of-life at longitudinal follow up (18). In our study, having a parent who smoked was not a risk factor for AHR, but contributed to asthma symptoms. This suggests that it is necessary to prohibit parents from smoking to prevent asthma-related symptoms in children. These findings emphasize the need for continuing effort to decrease exposure to passive smoke among schoolchildren and to increase understanding of the harmful health effects of smoking exposure. Parents of children with asthma

should be advised not to smoke, especially in rooms that their children use.

Kurukulaaratchy et al. (19) reported factors that influenced symptom expression in children with bronchial hyperresponsiveness at 10 yr of age. When only factors present in the first 4 yr of life were considered, parental smoking at 4 yr of age, maternal asthma, and atopic sensitization at 4 yr of age were independently associated with symptomatic AHR at 10 yr of age. In our study, although asthma symptoms and atopy were independent risk factors for AHR, parents' smoking status was not. We evaluated exposure to passive smoking with a questionnaire that did not distinguish between current and former smokers. Further studies are needed to clarify the role of passive smoking in the development of AHR.

In conclusion, we found that passive smoking was associated with asthma symptoms and that parents' smoking was not a risk factor in AHR, suggesting that further effort should be made to convince the parents of asthmatic children not to smoke in the house.

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