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SHORT COMMUNICATION

EFFECT OF PATERNAL SMOKING ON THE PULMONARY FUNCTIONS OF ADOLESCENT MALES

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Abstract : Objective : The present study was conducted to evaluate the effect of paternal smoking on the respiratory function of adolescents.

Study method : The study group comprised thirty healthy males between the ages of 12–18 years whose fathers were smokers (Group 1). They were compared with age and sex matched thirty active smoker males (Group 2). The control group comprised age and sex matched thirty males who were neither active nor passive smokers (Group 3). Pulmonary function was assessed using Sibelmed Datospir120B spirometer in a closed room.

Results : FEV1/FVC ratio and FEF25%–75% (forced expiratory flow at 25%–75% of vital capacity) were significantly lower in the subjects exposed to paternal smoking as compared to non-smokers. Vital capacity & expiratory reserve volume were significantly lower in active smokers as compared to non-smokers.

Conclusions : Our findings suggest a significant reduction in indices of lung functions of adolescents exposed to paternal smoke.

Key words : paternal smoking adolescent pulmonary functions environmental tobacco smoke India

INTRODUCTION

In the past, little attention was paid to the consequences of secondhand smoke on health. It was considered just another public nuisance. However, today, passive smoking, or Environmental Tobacco Smoke (ETS) exposure, is an important health concern worldwide. Numerous studies have highlighted the health consequences of ETS exposure, and people exposed to ETS have been considered to have the same risk profile as smokers (1, 2).

ETS is a combination of side-stream smoke, emitted from the burning end of a cigarette, which constitutes about 85% of the smoke in the room where active smokers smoke, and the remainder of main-stream smoke, exhaled by a smoker. The side-

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stream smoke contains many potentially toxic components, some of which may exist in even higher concentrations than in main-stream smoke (3).

It is possible that environmental tobacco smoke exposure may affect pulmonary functions in a manner similar to active smoking. A number of studies have highlighted several subtle as well as overt effects of ETS on pulmonary function (4–7).

Very few studies have been conducted to evaluate the effect of ETS on pulmonary function in developing countries like India, where smoking has been on the rise (8, 9). Taking into consideration environmental conditions like overcrowding, poor ventilation in homes and cramped livings conditions, the health effects of ETS exposure may be even more pronounced. Moreover, the effects of ETS exposure on children and young adolescents maybe an important aspect, as their respiratory and immune systems are still in developmental stages, possibly putting them at greater risk of being affected by second hand smoke. In addition, parental smoking assumes greater importance, as children and adolescents spend a lot of their time at home. Such considerations become even more important in a country like India, because of its large child and adolescent population.

METHODS

The study was carried out at the Environmental Physiology Laboratory, Department of Physiology, UCMS and GTB Hospital, Delhi. It was a case control study.

The subjects of the Present study were healthy males between 12 and 18 years of age, thirty in each group selected on the basis of a questionnaire.

Group 1 – Passive smokers: Non-smoking subjects whose fathers had smoked for greater than or equal to one year.

Group 2 – Active Smokers: Subjects who had smoked for greater than or equal to one year.

The Control group (Group 3) comprised thirty healthy males from the same age group who had never smoked, nor were exposed to ETS at home.

None of the above suffered from any acute or chronic respiratory disorder, or any systemic illness that may directly or indirectly affect the respiratory system. None of the subjects' mothers were smokers, or had smoked during pregnancy. Additionally, wood or coal was not used for heating or cooking in any home. No female subjects were included.

The pulmonary function tests were carried out using Sibelmed Datospir120 B precision spirometer with a built in computer program, using the standard laboratory methods. The apparatus provided a detailed analysis of predicted and derived values. Anthropometric measurements i.e. height and weight were recorded for each subject. Relevant data (name, age, sex, height, weight) was entered into the computer program. The test module was activated and the subject was given proper instructions about the procedure to be performed. All the

pulmonary function tests were performed with the subject comfortably seated in an upright position. Tests were done in one sitting under standard laboratory conditions. The subject was instructed on how to use the mouthpiece and was asked to breathe in order to familiarize himself with the equipment. A nose clip was applied during the entire procedure. During the test, the subject was adequately encouraged to perform to his optimum level. Tests were repeated three times and the best matching results were considered for analysis.

Statistical analyses were carried out using SPSS 10.0 statistical package. The three groups were compared on one way ANOVA with a 5% level of significance, using the Tukey test.

The study was approved by the Institute's Ethical Committee and a written informed consent was received from each subject. All procedures followed were in accordance with the ethical standards of the Institutional Ethical Committee – Human Research of the University College of Medical Sciences, Guruteg Bahadur Hospital, Delhi.

RESULTS

Table I shows the lung volumes and capacities of non-smokers, active smokers and passive smokers. Results show a lower vital capacity (VC), tidal volume (TV), expiratory reserve volume (ERV) and maximum ventilator volume (MVV) in both active and passive smokers as compared to nonsmokers. However, these were significant only for VC ($p = 0.031$) and ERV ($p = 0.007$) in active smokers (Group 2). The inspiratory reserve volume (IRV), inspiratory capacity (1C), forced vital capacity (FVC) and forced expiratory volume in the 1st second (FEV_1), were not significantly different in the three groups. However, passive smokers (Group 1) had significantly lower FEV_1/FVC values ($p = 0.021$) as compared to non-smokers.

TABLE I: Lung volumes and capacities in passive and active smokers as compared to non-smokers.

Parameter	*Group 1: Passive smokers*	*Group 2: Active smokers*	*Group 3: Non-smokers*	*Significance*
VC (l)	3.65±0.83	3.44±0.68*	3.93±0.59	Non smokers is significantly different from active smokers
TV (l)	0.89±0.31	0.95±0.31	0.98±0.34	not significant
ERV (l)	1.23±0.60	1.15±0.49**	1.58±0.54	Non smokers is significantly different from active smokers
IRV (l)	1.54±0.54	1.34±0.55	1.38±0.56	not significant
IC (l)	2.43±0.42	2.29±0.43	2.35±0.49	not significant
MVV (l/min)	108.28±26.46	108.84±27.36	119.65±26.55	not significant
Best FVC (l)	3.62±0.79	3.29±0.57	3.41±0.45	not significant
Best FEV1 (l)	3.28±0.55	3.02±0.44	3.27±0.41	not significant
FEV1/FVC (%)	89.08±16.77*	92.86±7.90	97.12±3.78	Non smokers is significantly different from passive smokers

*P<0.05; **P<0.01

Table II shows the flow rates of the three groups. Peak expiratory (PEF) and inspiratory (PIF) flow rates were not significantly different in the three groups. However the $FEF_{25-75\%}$ (forced expiratory flow at 25 to 75 percent of vital capacity) was significantly ($p = 0.004$) lower in passive smokers (Group 1) as compared to non-smokers.

The flow volume curves in a subject of each group (viz passive smokers, active smokers and non-smokers) are shown in Fig. 1.

DISCUSSION

Several studies have shown that exposure to ETS is associated with poor respiratory

TABLE II: Respiratory flow rates in passive and active smokers as compared to non-smokers.

Parameter	*Group 1: Passive smokers*	*Group 2: Active smokers*	*Group 3: Non-smokers*	*Significance*
PEF (l/s)	6.26±1.88	6.33±2.02	6.21±1.37	Not significant
$FEF_{25\%-75\%}$ (l/s)	3.58±1.01**	3.89±1.13	4.47±0.90	Non smokers is significantly different from passive smokers
PIF (l/s)	1.98±1.31	1.49±1.08	1.06±1.34	Not significant

*P<0.05; **P<0.01

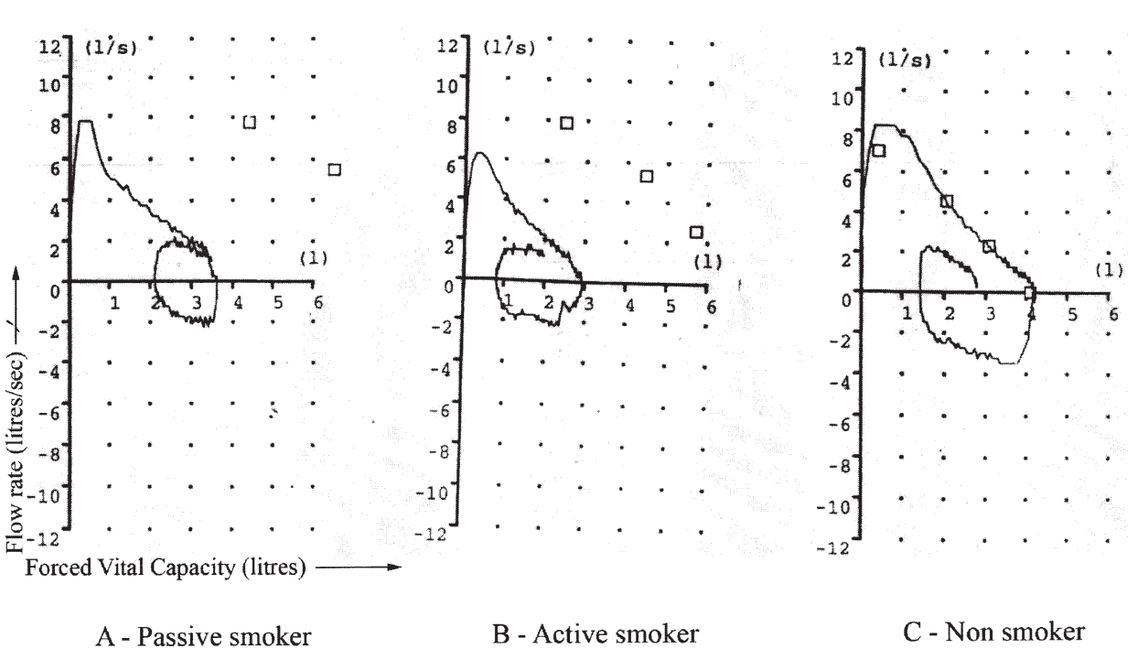

Fig. 1: Representative flow volume curves.

health in children (10–12). More recent studies have also demonstrated this effect in adults (13).

In the present study, two parameters of lung function were found to be significantly lower in passive smokers as compared to non-smokers. A significantly lower FEV_1/FVC ratio, as seen in the current study, suggests an obstructive effect of ETS. This finding is consistent with previous studies showing a lower ratio in children exposed to ETS (14–16).

$FEF_{25-75\%}$ indicates flow rates in small airways, i.e. airways with internal diameters less than 2 mm, which is reduced in both restrictive and obstructive diseases (17). A significantly lower $FEF_{25-75\%}$ in subjects exposed to paternal smoking as compared to non-smokers, as reported in this study, is also consistent with findings of previous studies showing lower $FEF_{25-75\%}$ as an effect of paternal smoking (18, 19). These values were also lower in active smokers, but were not statistically significant.

In our study, VC and ERV were significantly lower in active smokers. Other pulmonary function test parameters were also low in these subjects, but did not reach the level of statistical significance. A reason for this may be that the active smokers selected in the present study had been smoking for just 2–3 years, while in other studies subjects were chronic smokers for many years. This study may also have become biased by the ‘healthy smoker’ effect i.e. those subjects already smoking could be ‘healthy smokers’ or ‘survivors’ of the detrimental effects of cigarette smoke (6). This means that those less susceptible individually to any harm that may result from smoking may have continued to smoke, while others smoked less or stopped.

Our results regarding FEV_1 and FVC were not statistically significant and somewhat ambiguous. Results of previous studies showing the effect of parental smoking and exposure to household ETS on FVC are also inconsistent. Some studies have shown a significant association between maternal smoking and increased FVC (4–16, 18–20), while others reported a 13.4% decline in FVC in children who lived with at least one adult who smoked one or more packs a day (21). Cook et al. reported only a borderline effect of ETS on FVC, and Venners et al. reported small but detectable, albeit statistically insignificant, deficit in FVC in children of smoking fathers as compared to children of non-smoking fathers (22, 4). There has also been a report of finding no consistent and significant trend of reduced FVC (23). Results of previous studies regarding FEV_1 are more consistent, with a majority showing a lower FEV_1 in children exposed to parental smoking (22). However, there are also reports of statistically insignificant deficits as well as no significant trend of reduced FEV_1 (4, 23).

Previous studies on effects of paternal smoking have also yielded inconsistent results. Among some studies comparing the effects of maternal smoking and paternal smoking, nearly all report the effect of maternal smoking to be greater than that of paternal smoking (often reported to be zero), and none found a significant effect of smoking by the father alone (20, 24–28). On the other hand, there have been a number of studies showing a clear effect of paternal smoking (18, 19, 23, 29). Confounding by maternal

smoking due to selection of families including smoking mothers might be an important reason the above mentioned studies have not detected an effect of paternal smoking (20, 24–28). Effects due to paternal smoking may also be reduced due to shorter duration of contact, as most fathers are working.

There are some limitations that may influence the interpretation of our cross sectional results. Exposure to tobacco smoke was assessed retrospectively using questionnaire responses, and this study did not permit investigation of cumulative exposure to paternal smoking in relation to a subject's pulmonary function. Our data did not permit any investigation of the effects of prenatal versus postnatal exposure to paternal smoking. Although use of wood or coal as a household fuel was excluded, we were unable to determine the extent of indoor air pollution in the homes of the subjects. There was no quantification of ETS exposure, such as cotinine levels or cotinine/creatinine ratio (1). Thus, the effect of changes in ETS exposure on the subjects could not be taken into account. Any variation in the degree to which the subjects' fathers smoked inside or outside the home was also unaccounted for. The level of community pollution could not be taken into account, which has been shown to have effects similar in magnitude to the effects of home exposure to passive smoke on respiratory conditions (23). In addition, we lacked information on a number of potential confounders, such as maternal nutrition status and intake of alcohol, or other potentially toxic substances, during pregnancy.

Longitudinal studies are required to determine the lifelong impact of childhood exposure to ETS in a population with a high prevalence of tobacco use among men, and to determine whether the effects seen in cross-sectional studies are reversible. In addition, longitudinal studies of lung function in relation to cotinine levels are required, as they could take into account changes in ETS exposure as children spend less time with parents while they grow older, and thus their ETS exposure falls even while parental smoking habits remain constant. However, we can conclude that exposure to paternal smoking has a significant effect on lung functions of adolescents. In addition, significantly lower FEV_1/FVC and $FEF_{25–75\%}$ in Group 1 subjects suggest a detrimental obstructive effect of ETS, and a detrimental effect on the small airways, respectively. Thus, it is important to emphasize that exposure to ETS, especially in the developmental stages in life, may have effects as profound as those of active smoking.

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