Original articles

The effect of passive smoking on pulmonary function during childhood

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Summary

Passive smoking, especially of maternal origin, is known to influence adversely the development of children's pulmonary function. In this study, the effect of parental smoking on the pulmonary function of 360 primary school children aged 9–13 (mean 10.8±0.7) years was investigated. Information on parental smoking history was collected using a questionnaire, and spirometric measurements were performed on the children.

All spirometric indices were lower in children who had been passively exposed to parental tobacco smoke than those not exposed. The percentage of households in which at least one parent smoked was 81.5%. This figure was significantly lower for mothers (27.5%) than for fathers (79%). Paternal smoking was associated with reduced levels of forced expiratory flow between 25–75% of vital capacity, peak expiratory flow, and flow rates after 50% and 75% of vital capacity expired ($p<0.05$). Maternal smoking did not have statistically significant adverse effects on children's pulmonary function. This result might be due to the low occurrence of either pre- or post-natal smoking among mothers and confirms that, in our population, the main target group for anti-tobacco campaigns should be fathers.

Keywords: passive smoking; pulmonary function; tobacco smoke

Passive smoking is defined as the exposure of a nonsmoker to tobacco smoke in the environment.¹ This condition is a major environmental health problem, especially for children, and is becoming increasingly important in Turkey, due to increasing cigarette consumption. In some local studies it has been reported that the percentage of families in which at least one parent smokes is 75%.^{2,3}

It is widely known that smoking is the most common preventable cause of mortality and morbidity. Exposure to environmental tobacco smoke (ETS) has also been found to be a cause of preventable morbidity and mortality in non-smokers, both children and adults. The adverse effects of smoking may be due to one or more of the hundreds of different chemicals in tobacco smoke, although our knowledge of these substances, apart from carbon monoxide and nicotine, is limited.⁴ There is almost no

organ system that is not affected by smoking. The most commonly accepted effect of smoking is lung cancer,^{5,6} and there are many studies showing an association between passive smoking and wheezy bronchitis, asthma, bronchial hyper-reactivity, atopy predisposition and otitis media.^{7,8} Recently, it has been shown that passive smoking increases the morbidity of asthma and usage of asthma medications in school-aged children, in whom pulmonary function is especially affected by maternal smoking.^{9,10}

We performed a cross-sectional study to evaluate the association of paternal and maternal smoking with their children's pulmonary function.

Materials and methods

This study was designed to examine the influence of passive smoking on pulmonary function tests of children in the 4th and 5th classes of two primary schools in the Altındag district of Ankara in April 1996.

Information on the exposure of the children to cigarette smoke in the home was obtained by means of a questionnaire containing questions on the child, the parents, their medical history, smoking habits, duration of parental smoking, maternal smoking during pregnancy, home facilities, and the socioeconomic status of the family. We planned to include 420 students in the study but only 390 (92%) returned completed questionnaires. Spirometric measurements using a portable spirometer (Pony, Cosmed, Italy), were carried out on 360 students (30 children were excluded from the study due to insufficient cooperation). All measurements were performed by the same two pediatricians, and every manoeuvre was repeated at least three times to get the optimum results. The following indices were measured: forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), peak inspiratory and expiratory flows (PIF, PEF), FEV_1/FVC ratio, forced expiratory flow between 25–75% of vital capacity (FEF_{25-75}), and maximum flow rates during 25, 50, and 75% of vital capacity exhaled ($V_{max\ 25, 50, 75}$). Independent variables were parental smoking, amount and duration of smoking, maternal smoking during pregnancy and the medical history of child and parents. As dependent variables we used FEV_1 , FVC, FEF_{25-75} , PEF, $V_{max\ 25, 50, 75}$. We used the Student's t -test to compare independent sample means and variance analysis for the comparison of more than two group means. All

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statistical analysis were made using the Statistical Programs for Social Sciences (SPSS) software.

Results

The study group consisted of 360 students aged between 9–13 years (mean 10.8 ± 0.7), 169 girls (46.9%) and 191 boys (53.1%). As a measure of passive smoking by the children, table 1 shows the numbers of parents who smoked in our study group. (Eight fathers were missing, dead or divorced; data on those eight families are excluded from the table.) The percentage of houses in which at least one parent smoked was quite high (81.5%). This figure was significantly higher for fathers (79%) than for mothers (27.5%). The duration and number of cigarettes smoked by fathers was also significantly higher than by mothers (56% of smoker mothers had been smoking for 10 years or more, compared with 85% for fathers).

The population in our study was from a low socioeconomic group. In 92% of the families, the mothers were housewives. Among fathers, almost 50% were unemployed, and only 43% of the families had their own home.

A comparison of the spirometric indices (FEF_{25-75} , FEV_1 , FVC, PEF, V_{max25} , V_{max50} , V_{max75}) in different groups of children on the basis of parental smoking habits is given in table 2. We found no statistically significant differences between the spirometric indices of children whose mothers were active smokers and those whose mothers were nonsmokers, although the percentages of means of all spirometric parameters in the smoking group were lower than those in the nonsmoking group.

For paternal smoking, the decrease in spirometric indices of children whose fathers smoked were greater than in the case of maternal smoking. These decreases in the paternal

Table 1 The smoking pattern of the families (eight fathers were missing, dead or divorced; the remaining 352 families are described)

	Fathers nonsmoking (n=74)	Fathers smoking (n=278)
Mothers nonsmoking (n=255)	65	190
Mothers smoking (n=97)	9	88

Table 2 Comparison of the children's mean spirometric indices according to maternal and paternal smoking patterns (pulmonary function values are % of predicted normal). Figures in parentheses are standard errors of the mean

	Mother			Father		
	Smoking (n=105)	Nonsmoking (n=255)	p	Smoking (n=278)	Nonsmoking (n=74)	p
FEF_{25-75}	80.23 (1.92)	82.72 (1.18)	0.26	80.50 (1.09)	86.17 (2.46)	0.02
FEV_1	83.91 (1.10)	85.90 (0.69)	0.12	84.78 (0.65)	86.40 (1.38)	0.26
FVC	84.43 (1.28)	85.15 (0.73)	0.62	84.60 (0.73)	85.05 (1.39)	0.77
PEF	76.95 (1.81)	80.28 (1.11)	0.11	77.94 (1.06)	82.87 (2.17)	0.03
V_{max25}	76.39 (1.87)	78.51 (1.17)	0.33	76.61 (1.09)	81.14 (2.37)	0.06
V_{max50}	81.30 (1.92)	83.88 (1.14)	0.23	81.52 (1.08)	87.93 (2.27)	0.008
V_{max75}	93.15 (2.39)	95.62 (1.63)	0.40	92.89 (1.45)	101.67 (3.42)	0.009

FEF_{25-75} : forced expiratory flow between 25–75% of vital capacity; FEV_1 : forced expiratory volume at one second; FVC: forced vital capacity; PEF: peak expiratory flow; V_{max25} , V_{max50} , V_{max75} : flow rates after 25%, 50% and 75% of vital capacity expired, respectively.

Table 3 Comparison of mean FEV_1 values according to maternal smoking during pregnancy

	n	Mean FEV_1 (% of predicted)	SD	SEM
Mother smoked prenatally	37	84.94	11.15	0.62
Mother not smoked prenatally	323	85.37	11.01	1.81

FEV_1 : forced expiratory volume at one second; SD: standard deviation; SEM: standard error of the mean

smoking group for FEF_{25-75} , PEF, V_{max50} and V_{max75} were all statistically significant ($p < 0.05$).

There was no significant reduction in pulmonary function tests of children whose mothers had smoked during pregnancy. The comparison of FEV_1 values according to the maternal smoking during pregnancy is shown in table 3. Maternal smoking prevalence during pregnancy was low (10%) and only 5% admitted smoking more than 10 cigarettes per day.

We also analysed the influence of the number of cigarettes smoked by the parents per day. The spirometric indices of children were compared on the basis of how much their parents smoked per day (<5, 5–10, 10–20 and >20 cigarettes per day). No significant dose-dependent reductions in pulmonary function tests were found ($p > 0.05$).

Discussion

In this study, paternal smoking was shown to be associated with significantly decreased spirometric indices in children. This decrement in pulmonary function levels associated with paternal smoking may be due to a combination of demographic and socioeconomic factors and to the smoking pattern of our population. The high rates of smoking among fathers who spend more time at home because of unemployment may result in greater exposure of the child to ETS than maternal smoking. Our results are consistent with earlier results documenting a reduction of pulmonary function tests in passively smoking children, although we failed to find a dose-dependent reduction in pulmonary function. Some disadvantages of the questionnaire method may result in deficiencies in the data collected on the amount of exposure. These could have been partly resolved by measuring the level of urinary cotinine, still the most valid marker for tobacco smoke exposure.

In contrast to earlier results, we did not find any association between pulmonary function tests and maternal smoking.^{9,10} Other studies have shown that maternal smoking is associated with a greater exposure to ETS than paternal smoking. In our study, the daily cigarette consumption among mothers, including consumption during pregnancy, was low. This may explain our failure to demonstrate an effect of maternal smoking on pulmonary function in children. Further studies would be needed to confirm this.

Passive smokers are exposed to cigarette smoke in two ways: by direct exposure to the

smoke of passively burning cigarettes (side-stream), and exposure to the smoke exhaled by the smoker.¹¹ There are also some qualitative and quantitative differences between the exposures of active and passive smokers. Because the temperature of the burning cone is lower as the cigarette smoulders than during active puffing, combustion is less complete in side-stream than in mainstream smoke. Consequently, side-stream smoke has higher concentrations of some toxic and carcinogenic substances than mainstream smoke.⁷ Smoking in an enclosed area increases the concentration of respirable particles such as nicotine, polyacrylic hydrocarbons, carbon monoxide, acrolein and nitrogen dioxide. The impact of smoking on indoor air quality depends on the number of smokers, the intensity of smoking, the size of the indoor space, the rate of exchange of the air of the indoor space with the outdoor air, and the use of air cleaning devices. It is obvious that the effect of smoking of the parent with more, and closer, contact with the child, usually the mother, will be greater.⁷

In the children of women who smoke during pregnancy, forced expiratory flow rates corrected for volume have been shown to be decreased significantly.¹² The pathophysiologic mechanism responsible for this decrease is as follows: passively exposed smoke causes mucus gland hypertrophy and hyperplasia directly influencing the bronchial mucosa and alveoli. Cough, sputum and mucus secretion increases mucosal permeability against allergens. As a result, chronic inflammation of the small

airways may cause bronchial obstruction.^{1 10}

The result, confirmed by most recent studies, is that the FEV₁ values of children are significantly decreased by parental smoking, especially of maternal origin, and this association is dose dependent.¹³⁻¹⁸ In our study, the spirometric indices of the children were influenced negatively by paternal smoking and this influence was more prominent in pulmonary flows representing small airways than in larger bronchi. We must assume that cigarette smoke effects smaller airways more than larger ones. It causes infiltration of cells and hypertrophy and hyperplasia of mucus glands. Chronic inflammation of smaller airways may lead to bronchial obstruction.

Our results show that exposure to ETS due to paternal smoking also has a strong effect on children's pulmonary function. It has been demonstrated that modifications of the smoking behaviour of parents can result in lower metabolites in their children.¹⁹ Therefore, more practical, legal and management precautions should be taken in order to decrease ETS and especially to reduce the exposure of children. However, the sanctity of the family unit restricts the ability of governmental action to diminish exposure in the home. Healthcare providers would appear to have a central and critical role to play in educating parents on this point, as they interact with parents at key times, such as during pregnancy, at birth, and at well-child visits, as well as on visits for illness. We believe that continued efforts to provide a smoke-free home for all children remain essential.

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