The effect of passive smoking on pulmonary function during childhood

Kenan Bek, Nazan Tomaç, Ali Delibas, Fevzi Tuna, H Tahsin Teziç, Metin Sungur

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 antitobacco campaigns should be fathers.

Keywords: passive smoking; pulmonary function; tobacco smoke

Passive smoking is defined as the exposure of a non-smoker to tobacco smoke in the environment. 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%.

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 nonsmokers, 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. There is almost no

organ system that is not affected by smoking. The most commonly accepted effect of smoking is lung cancer, and there are many studies showing an association between passive smoking and wheezy bronchitis, asthma, bronchial hyper-reactivity, atopy predisposition and otitis media. 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.

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 Altindag 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, during 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 (FEV1), peak inspiratory and expiratory flows (PIF, PEF), FEV1/FVC ratio, forced expiratory flow between 25–75% of vital capacity (FEF25–75), and maximum flow rates during 25, 50, and 75% of vital capacity exhaled (Vmax 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 FEV1, FVC, FEF25–75, PEF, Vmax 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
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
(FEF25-75, FEV1, FVC, PEF, \(V_{max}\) 25, 50, 75) 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
smoking group for FEF25-75, PEF, \(V_{max}^{50}\) and
\(V_{max}^{75}\) were all statistically significant (p<0.05).

Other studies have shown that maternal smoking is
associated with a greater exposure to ETS than
paternal smoking. In our study, the daily cigare-
tette 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

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

<table>
<thead>
<tr>
<th>Fathers</th>
<th>Mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonsmoking (n=74)</td>
<td>Smoking (n=278)</td>
</tr>
<tr>
<td>Nonsmoking (n=255)</td>
<td>65</td>
</tr>
<tr>
<td>Smoking (n=97)</td>
<td>9</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Smoking</th>
<th>Non-smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother (n=105)</td>
<td>(n=225)</td>
</tr>
<tr>
<td>FEF25-75</td>
<td>80.23 (1.92)</td>
</tr>
<tr>
<td>FEV1</td>
<td>83.91 (1.10)</td>
</tr>
<tr>
<td>FVC</td>
<td>84.43 (1.28)</td>
</tr>
<tr>
<td>PEF</td>
<td>76.95 (1.81)</td>
</tr>
<tr>
<td>(V_{max}^{25})</td>
<td>76.39 (1.87)</td>
</tr>
<tr>
<td>(V_{max}^{50})</td>
<td>81.30 (1.92)</td>
</tr>
<tr>
<td>(V_{max}^{75})</td>
<td>93.15 (2.39)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Smoking</th>
<th>Non-smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father (n=278)</td>
<td>(n=74)</td>
</tr>
<tr>
<td>FEF25-75</td>
<td>80.50 (1.09)</td>
</tr>
<tr>
<td>FEV1</td>
<td>84.78 (0.65)</td>
</tr>
<tr>
<td>FVC</td>
<td>84.63 (0.73)</td>
</tr>
<tr>
<td>PEF</td>
<td>77.94 (1.06)</td>
</tr>
<tr>
<td>(V_{max}^{25})</td>
<td>76.61 (1.09)</td>
</tr>
<tr>
<td>(V_{max}^{50})</td>
<td>81.52 (1.08)</td>
</tr>
<tr>
<td>(V_{max}^{75})</td>
<td>92.89 (1.45)</td>
</tr>
</tbody>
</table>

FEF25-75: forced expiratory flow between 25–75% of vital capacity; FEV1: forced
expiratory volume at one second; FVC: forced vital capacity; PEF: peak expiratory
flow; \(V_{max}^{25}\), \(V_{max}^{50}\), \(V_{max}^{75}\): flow rates after 25%, 50% and 75% of vital capacity expired, respectively.

Table 3 Comparison of mean FEV1 values according
to maternal smoking during pregnancy

<table>
<thead>
<tr>
<th>Smoking</th>
<th>Non-smoking</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother smoked</td>
<td>(n=278)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>83.91 (1.10)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>84.78 (0.65)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>84.63 (0.73)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>77.94 (1.06)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>76.61 (1.09)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>81.52 (1.08)</td>
</tr>
<tr>
<td>Maternal smoking</td>
<td>92.89 (1.45)</td>
</tr>
</tbody>
</table>

Discussion
In this study, paternal smoking was shown to
be associated with significantly decreased
spirometric indices in children. This decre-
ment in pulmonary function levels associated
with paternal smoking may be due to a combi-
nation of demographic and socioeconomic fac-
tors and to the smoking pattern of our popula-
tion. 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 func-
tion tests in passively smoking children, although
we failed to find a dose-dependent reduction in
pulmonary function. Some disadvan-
tages 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. Other studies
have shown that maternal smoking is associ-
ated with a greater exposure to ETS than
paternal smoking. In our study, the daily cigare-
tette 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.
smoke of passively burning cigarettes (side-stream), and exposure to the smoke exhaled by the smoker. There are also some qualitative and quantitative differences between the exposures of active and passive smokers. Because the temperature of the burning zone 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. 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 effects of passive smoke cause 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. 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. 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. 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.