The Impact of Passive Smoking on the Development of Lower Respiratory Tract Infections in Children

by Pembe Keskinoglu,[a] Dilek Cimrin,[b] *and* Gazanfer Aksakoglu[a]
[a]*Dokuz Eylul University, School of Medicine, Department of Public Health, Inciralti/Izmir, Turkey*
[b]*Dokuz Eylul University Hospital of Medicine School, Director of Central Laboratory*

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Summary

Environmental tobacco smoke is an important public health problem. The objective of this study was to evaluate the effect of passive smoking on lower respiratory tract infections (LRTIs) in children aged 2–12 years. A case-control study was conducted on matched-pair design. One-hundred and fifty children with LRTIs and 150 healthy children were included in the study. Data were collected through questionnaire and urine samples for the determination of cotinine levels, and were analysed by McNemar chi-square, paired *t*-test and Pearson correlation tests. The prevalence of parental self-reported, indoor smoking was 71.3% in children with LRTI and 72.0% in healthy children. Employing 30 ng mg^{-1}, the cut-off level of urinary cotinine/creatinine as commonly accepted, 87.3% of the children with LRTIs and 84.7% of healthy children were found to be passive smokers ($p = 0.61$, odds ratio (OR) = 0.93; confidence interval (CI) = 0.34–2.53). If 60 ng mg^{-1} of urinary cotinine/creatinine was accepted as a cut-off level, it was observed that the rates of passive smoking were 76.7% and 50.7%, respectively ($p = 0.000$, OR = 4.72; 95% CI = 2.62–8.52). Dose-dependent exposure to environmental tobacco smoke was found to be associated with the incidence of LRTI.

Key words: children, cotinine, lower respiratory tract infection, passive smoking.

Introduction

Environmental tobacco smoke (ETS) is an important public health problem causing adverse effects on the health outcomes of children [1–7]. Parental smoking has an increasing impact on infectious and non-infectious lower respiratory tract diseases in children [8–13]. In several studies it has been demonstrated that infants and preschool children who were exposed to parental smoking developed bronchiolitis and bronchopneumonia. Bronchitis, pneumonia and chronic cough were seen more frequently among school children [14–16]. In ETS-exposed children, asthma occurred at an earlier period during childhood and its frequency and severity tend to increase [17–19].

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Correspondence: Pembe Keskinoglu, Dokuz Eylül University, School of Medicine, Department of Public Health, 35340, Inciralti/Izmir, Turkey. Tel: +90 232 412 40 01; Fax: +90 232 278 68 64;
E-mail: <pembe.keskinoglu@gmail.com>.

Exposure intensity of ETS shows a strong correlation with indoor parental and especially maternal smoking, the number of cigarettes smoked, the number and volume capacity of the rooms where cigarettes are smoked and individual metabolic diversities [2, 7, 20–23]. Globally the percentage of children exposed to ETS is considerably high and varies from 29% to 69% [24]. In a study carried out in elementary school children in Turkey, the prevalence of passive smoking was 81.3% [25]. Determination of the prevalence of passive smoking in children can be performed by parental self-reports and by the measurements of cotinine which is the major metabolite of nicotine. Epidemiological studies on ETS exposure report that urine cotinine measurement is more reliable compared to parental self-reporting, has a higher sensitivity and indicates a longer period of exposure [26–29].

The objective of this study was to determine the impact of passive smoking on lower respiratory tract infection (LRTI) in children aged 2–12 years.

Material and Methods

The study was performed in the Narlidere district of Izmir, which is located at west region of Turkey. There are three health centres in this area.

A case-control study was conducted on matched-pair design. To detect an odds ratio (OR) of 2 with

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80% power, and assuming 50% of the control group will be exposed to ETS, approximately 139 cases and 139 controls were required as the sample size of the study. One hundred and fifty cases and 150 control children were included in the study. The study was approved by the Ethics Committee of the Faculty of Medicine and written informed consent was obtained from each parent.

Case group

This group included children 2–12 years of age with LRTIs (bronchiolitis, pneumonia, or acute exacerbation of asthma induced by infection) who were diagnosed in health centres. Case definitions were performed according to the diagnostic criteria of the World Health Organization (WHO) [30]. The cases were selected daily from the patient records of health centres.

Control group

This group included healthy children during the study, and they were paired with the cases obtained from the same health centre district according to age, gender and street of the residing area (just one child per family). The controls were selected randomly from information in health centres.

The dependent variable was LRTI. The independent variables were socio-economic variables, variables of health status, variables related to housing and variables related with passive smoking.

In the literature, a urinary cotinine/creatinine ratio (CCR) of $30\,ng\,mg^{-1}$ creatinine is considered to be the cut-off level that indicate exposure to passive smoking [31]. However, as the prevalence of passive smoking in developing countries such as Turkey is very high, this cut-off level would be unable to indicate the causative effect of ETS in LRTI correctly. For this reason, ETS exposure was also evaluated using a cut-off level of $60\,ng\,mg^{-1}$ to investigate whether there is any correlation between ETS exposure and LRTI in an intensive exposure status and to assess the dose-dependent relationship.

This case-control study was carried out between October 2003 and March 2004. The residences of children diagnosed with LRTI at health centres were visited within first 3 days of diagnosis. A specially designed questionnaire was used, and urine samples were collected for the determination of cotinine. Control children were visited at home at the same days, together cases and the same methods were applied.

The urinary cotinine was measured by RIA method (Diagnostic Products Corporation, Los Angeles, US). The detection level of urinary cotinine was $10\,ng\,ml^{-1}$. To adjust the effect of variable dilution on the spot concentration of cotinine, urinary CCR was calculated and expressed as nanogram per milligram of creatinine.

Data were analysed on SPSS 11.0. McNemar chi-square test, *t*-test and Pearson correlation coefficient were used for statistical analysis. A $p < 0.05$ was accepted as statistically significant.

Results

The mean age of the cases and control children was 5.1 ± 2.7 years (range 2–12, SD $\pm$ 2.7). One hundred-ninety two (64.0%) of the children were male. Mean number of household was 4.25 (SD $\pm$ 1.36) in case groups; 4.26 (SD $\pm$ 1.26) in controls group, with no significant difference ($p = 0.499$). Table 1 shows some characteristics of the groups. Total 71.3% of the children were aged 2–6 years; 71.3% of cases and 74.3% of controls had at least one sibling. The percentage of the uneducated mothers was higher in the case group (29.3% vs. 23.3%). The percentage of the mothers who were unemployed and housewives in the case group was higher than the control group, but difference was not significant. The percentage of

TABLE 1
Distribution of cases and controls according to demographic and economic characteristics

Characteristics	Case ($n = 150$) (%)	Control ($n = 150$) (%)	p^*	OR	95% CI
Age (2–6 years old)	71.3	71.3	1.000	1.000	
Siblings (yes)	71.3	74.3	0.525	0.824	0.072–3.836
Mother's education					
Never attended school	29.3	23.3	0.222	1.529	0.786–2.818
Mother's occupation					
Unemployed, housewife	87.3	82.7	0.296	1.585	–
Father's education					
Middle school or under	70.0	83.3	0.004	0.333	0.157–0.707
Father's occupation					
Unemployed, irregularly employed	42.7	45.3	0.671	0.852	0.460–1.790
Perceived economical status bad	43.3	43.3	1.000	1.000	–

*McNemar chi-square test.

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children with LTRI whose fathers graduated from middle school or under was significantly lower than the controls ($p = 0.004$).

The rate of cases living in apartments and detached houses with a lesser number of windows was higher than the controls; with no significant difference ($p = 0.499$). The percentage of indoor pollution fuels in the homes like wood, coal and gas were quite high in two groups (81.3% in case group; 90.7% in control group). Central heating and electric stoves were more common in the houses of the cases, while fuels used for cooking or heating, such as firewood, charcoal and gas were more common in the houses of controls ($p = 0.007$). No significant difference was found between the case and control groups according to the mean air volume per individual ($p = 0.201$) (52.7 vs. 50.2 m^3). The ratio of LRTI in the last two years was found significantly higher in the case group (42.7 vs. 16.7%; $p < 0.001$). Existence of chronic diseases in parents were found at similar ratios while the ratio of chronic diseases in the cases was higher; the difference was not significant ($p = 0.077$).

Paternal smoking frequency was 54.7 and 61.3% in the cases and the controls, respectively. Maternal smoking frequency was 34.7 and 39.0%, respectively. The percentange of other smokers was determined as 9.3% in both groups. The rates of paternal, maternal or other's smoking at home were similar in two groups.

According to cut-off value of 30 ng mg^{-1} of urinary CCR, the prevalence of passive smoking was 87.3% in case group and 84.7% in control group ($p = 0.618$). However, a significant difference was found between passive smoking prevalence in groups at level of 60 ng mg^{-1} of urinary CCR ($p < 0.001$). The children with LRTI had more dense ETS exposure compared to the healthy children (OR = 4.72; 95% CI = 2.62) (Table 2). None in groups was active smoker.

Indoor smoking status of two groups is shown in Table 3. In the case group, the mean number of cigarettes smoked at home and the mean urinary CCR were significantly higher than the control group ($p = 0.040$, $p = 0.016$, respectively). The cases with LRTI were heavy smokers and were exposed to a higher intensity of ETS.

In the case group the mean number of cigarettes smoked per day by the mother, by other individual living in the home or in the child's room was higher than the control group although it did not cause a significant difference. The correlations between some variables in groups are shown in Table 4.

As the number of cigarettes smoked at home increased, urinary CCR in the cases and the controls increased significantly ($p = 0.016$, $p < 0.001$, respectively). As the number of smokers at home increased, the number of cigarettes smoked at home and urinary CCR also increased ($p = 0.007$, $p < 0.001$, $p < 0.001$, respectively). Additionally, as it was more

TABLE 2
Distribution of cases and controls according to passive smoking status

Characteristics	Case ($n = 150$) (%)	Control ($n = 150$) (%)	p^*	OR	95% CI
Smoking in residence	71.3	72.0	1.000	0.970	–
Paternal smoking	54.7	61.3	0.289	0.756	0.451–1.263
Maternal smoking	34.7	39.3	0.489	0.829	0.488–1.409
Other residents smoking	9.3	9.3	0.843	1.000	–
Urinary CCR ≥ 30 ng mg^{-1} creatinine	87.3	84.7	0.618	0.937	0.346–2.538
Urinary CCR ≥ 60 ng mg^{-1} creatinine	76.7	50.7	0.000	4.727	2.624–8.516

*McNemar chi-square test.

TABLE 3
Comparison of case and control groups according to mean passive smoking exposure

Characteristics	Case		Control		p^*
	Mean	Standard error	Mean	Standard error	
Cotinine ng mg^{-1} creatinine	141.78	11.9	108.95	10.6	0.016
Number of cigarettes (at home/day)	7.97	0.72	5.96	0.52	0.040
Number of cigarettes (mother/day)	4.44	0.58	3.56	0.48	0.244
Number of cigarettes (father/day)	10.24	0.99	10.13	0.87	0.936
Number of cigarettes (other residents/day)	1.96	0.60	1.17	0.36	0.228
Number of cigarettes (children's room/day)	2.62	0.36	2.13	0.27	0.268

*Paired *t*-test.

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TABLE 4
Correlation between some variables in case and control groups

	u-CCR		Number of cigarettes		Number of smokers		m^3 per individual	
	r	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Case group ($n=150$)								
Number of cigarettes smoked at home	0.197	0.016						
Number of smokers at home	0.218	0.007	0.686	0.000				
m^3 per individual at home	−0.036	0.665	−0.128	0.119	−0.091	0.268		
Age of child	−0.295	0.000	−0.039	0.633	−0.144	0.078	0.104	0.206
Control group ($n=150$)								
Number of cigarettes smoked at home	0.331	0.000						
Number of smokers at home	0.358	0.000	0.692	0.000				
m^3 per individual at home	−0.142	0.082	−0.138	0.093	0.207	0.011		
Age of child	−0.262	0.001	0.068	0.406	0.032	0.701	−0.001	0.992

TABLE 5
Comparison of smoking based on parental reports and urine cotinine levels

	u-CCR $\geq 30\,ng\,mg^{-1}$ creatinine		u-CCR $< 30\,ng\,mg^{-1}$ creatinine		Total		p^*
	Number	(%)	Number	(%)	Number	(%)	
Case group ($n=150$)							
Cigarettes smoked at home[a]	96	89.7	11	10.3	107	100.0	0.001
Cigarettes not smoked at home	35	81.4	8	18.6	43	100.0	
Total	131	87.3	19	12.7	150	100.0	
Control group ($n=150$)							
Cigarettes smoked at home[a]	99	91.7	9	8.3	108	100.0	0.003
Cigarettes not smoked at home	28	66.7	14	33.3	42	100.0	
Total	127	84.7	23	15.7	150	100.0	

*McNemar chi-square test.
[a]Parental report.

apparent in the case group, there was significant negative correlation between the child's age and the urinary CCR. As the child's age decreased, urinary CCR increased. In this study 55.3% of the LRTI was determined in younger children (2–4 years old).

There was discordance between the parental reports and cotinine measurements and this incompatibility has been observed more in the case group. Urinary CCR were $>30\,ng\,mg^{-1}$ in 35 (81.4%) of the 43 cases and in 28 (66.7%) of the 42 controls whose parents reported that they did not smoke at home ($p=0.001$, $p=0.003$, respectively) (Table 5).

Maternal smoking prevalence during pregnancy were similar in the two groups.

Discussion

In this study, passive smoking prevalence determined by urine cotinine measurement was 87.3% in children with LRTI and 84.7% in healthy children. The prevalence based on quantitative measurement is higher than parental self-reports and this finding was consistent with the literature [7, 16, 23, 26, 28, 29]. A higher discordance was present between the parental self-reports and urine cotinine levels in the case group compared with the control group. This finding may suggest that families usually hide or not consider the possibility of ETS exposure by their smoking or the child may be exposed to ETS out of home.

The prevalence of active smoking among females aged 15 and over in Turkey was 20% to 29%, and >60% among males aged 15 and over [24]. In our study, paternal smoking prevalence was similar in the case and control groups. However, maternal smoking prevalence was higher in both groups (34.7% vs. 39.3%). This finding is not surprising because smoking prevalence is significantly higher in urban and slum areas compared to the general population in Turkey.

In the present study, the confounding factors such as age, gender, crowded living conditions that

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facilitated the development of LRTI were controlled by pairing age, gender and residing street. The number of household (4.25 vs. 4.26) was not a risk factor for LRTI. The distribution of age and gender was same among the two groups and the variables like the occupation of parents, mother's education and perceived economical status were also similar. In a study carried out by Koch *et al*. [14], it was found that crowded living conditions were a risk factor in the 0–2 years old children. Prietsch *et al*. [15] observed that low educated mothers, bad environmental conditions, low income and crowded living conditions were risk factors for LRTI in children between 0–59 months old. In this study, cases and controls were matched according to age, gender and other environmental factors during the study planning stage leaving the ETS exposure intensity the only variable effecting LRTI. The presence of smoke in the home from fuel such as firewood, coal, gas had no impact on LRTI.

Lux [32] showed that, maternal smoking during pregnancy caused the development of LRTI in children between 18–30 months of age. Several researchers also, have reported that maternal smoking during pregnancy is an important risk factor for LRTI, especially in early childhood [3, 4, 7, 17, 33]. In the current study any relationship between maternal smoking during pregnancy and LRTI was not found. It is assumed that the high percentage of the older children according to young children in the study impeded the determination of the mentioned effect.

Several studies demonstrated that maternal smoking facilitated the development of LRTI and increased the severity of acute asthmatic attacks [11, 18, 19] Especially pre-school children spend the majority of their time with their mothers and therefore the impact of passive smoking on these children is expected to be more intense. The prevalence of maternal smoking in this study was found similar in the two groups. The mean number of cigarettes smoked per day by mothers of children with LRTI was higher than the control group (4.44 vs. 3.56); this difference was not statistically significant. However, the total number of cigarettes smoked at home was significantly higher in the cases. This can be an indication of poor reporting of cigarette smoking at home by mothers in the case group.

According to correlation analyses, there was a significant positive correlation between urinary CCR and number of cigarettes smoked at home in two groups, significancy being higher in control group. This finding suggests that the parents of children in case group may under-report the child's exposure to tobacco smoking. This is consistent with the findings of Kohler *et al*. [34].

Although the ETS exposure rates were similar between the case and control groups at cut-off level of $30\,ng\,mg^{-1}$ creatinine of CCR, the mean number of cigarettes smoked per day and the mean urinary CCR were significantly higher in the case group. When the cut-off level of urinary CCR was accepted as $60\,ng\,mg^{-1}$ creatinine it was found that passive smoking increased LRTI 4.7 times in children. It shows dose-dependant exposure. However this is an expected result and is consistent with the studies in the literature [5, 18, 23, 35]. The level of $30\,ng\,mg^{-1}$ creatinine of CCR as passive smoking indicator may be appropriate for countries having the low smoking prevalence. Nevertheless, a higher cut-off level of CCR (e.g. $60\,ng\,mg^{-1}$ creatinine) must be accepted for countries having higher exposure rates, like Turkey.

LRTI within the last 2 years is significantly higher in the case group. Chronic diseases were more frequent in the case group compared with the control group. The 58.9% of the previous chronic diseases in children are recurrent otitis media, adenoid hypertrophy and chronic tonsillitis, where passive smoking could be have a causal effect. In the literature, a positive correlation between passive smoking and upper respiratory tract infections it also reported [1, 3, 22]. These covariates are considered as important markers demonstrating the impact of ETS exposure on LRTI and the child's health. In present study, exposure to ETS was higher in preschool age.

In conclusion, passive smoking prevalence is very high in children with LRTI and in healthy children. A higher discordance was present between the parental self-reports and urine cotinine levels. Severe exposure to passive smoking in children was correlated positively with LRTI. It was concluded that passive smoking was an important risk factor for LRTI in children.

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