

Does raising awareness in families reduce environmental tobacco smoke exposure in wheezy children?

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Abstract

Introduction: Environmental tobacco smoke (ETS) is thought to increase the severity and number of attacks in wheezy children. Objective assessments are needed to change the behavior of families to reduce the exposure of wheezy children to ETS.

Aim: To determine whether informing families about their children's urinary cotinine levels curtailed the exposure of children to ETS.

Material and methods: A survey was used to determine the ETS exposure level, and the urinary cotinine level of each patient was tested. Children with positive urinary cotinine levels were included in the second part of the study. The families were randomly divided into two groups: an intervention group that was advised about urinary cotinine levels by telephone and a non-intervention group that was not so advised. The groups were followed-up 2 months later, and urinary cotinine levels were measured once again.

Results: The intervention group contained 65 children of average age of 24.4 ±8.9 months, of whom 46 (70.8%) were male. The non-intervention group contained 69 children of average age of 25.3 ±9.8 months ($p > 0.05$), of whom 52 (75.4%) were male. The urinary cotinine levels at the time of the second interview were lower in both groups. The number of cigarettes that fathers smoked at home decreased in the intervention group ($p = 0.037$).

Conclusions: Presenting objective evidence on ETS exposure to families draws attention to their smoking habits. Measurement of cotinine levels is cheap, practical, and noninvasive. Combined with education, creating awareness by measuring cotinine levels may be beneficial.

Key words: cotinine, children, episodic wheezing, environmental tobacco smoke.

Introduction

Environmental tobacco smoke (ETS) exposure is defined as exposure to tobacco smoke and the products thereof caused by tobacco consumption by others [1]. The World Health Organization (WHO) estimates that approximately one-half of all children in the world are exposed to ETS [2]. Because children breathe faster, they inhale toxic chemicals in tobacco smoke more intensely. Environmental tobacco smoke has both facilitative and aggravating effects on lower respiratory tract diseases, which is more obvious in early childhood [2]. Environmental tobacco smoke exposure at an early age is thought to cause inflammation of the airway mucosa and to facilitate the development of wheezing illness in the future [3–7].

Environmental tobacco smoke is one of the most important risk factors for episodic wheezing [4]. Even when warned of the dangers of ETS, families frequently do not change their smoking habits and ETS exposure continues. Objective assessments are needed to change the smoking habits of families to ensure that they cease smoking in the company of children who have episodic wheezing.

Aim

In our present study, we sought to determine whether educating such families and informing them about the urinary cotinine levels of their children would decrease ETS exposure.

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Material and methods

Patients

The study was performed in the Allergy and Immunology Department of Dr. Behcet Uz Children Hospital's (Izmir, Turkey) between December 2012 and December 2013. Children younger than 3 years with episodic wheezing were included in the study if their parents declared that they did not smoke in any environment shared by their children, although at least one person in the household was a smoker. The European Respiratory Society (ERS) classification was used to identify children with episodic wheezing [8]. Thus, preschool children who had episodic wheezing attacks triggered by viral upper respiratory tract infections and who were symptom-free between attacks were included. All participants were advised about the nature of the study and all families signed informed consent forms. The study was approved by our local Ethics Committee.

Study design

A survey instrument was first completed by the families of 237 patients eligible for the study. Training brochures on ETS exposure were prepared and they contained detailed information on ETS and the harm caused by ETS, as well as instructions about how to protect against ETS. Via the questionnaire, the number of parents smoking at home, the number of cigarettes smoked daily both at home and outside, and the presence or absence of smoking bans at home were explored. Urinary cotinine levels were measured in children. Of these, 193 participants who had a positive urinary cotinine level were randomly divided into two groups: an intervention group that was informed about their urinary cotinine levels and a non-intervention group that was not informed. The intervention group was given education about the harm by ETS exposure and ways to avoid it, and the other group was not.

The urinary cotinine levels of the two groups were measured again 2 months later, and the survey was repeated (Figure 1). Finally, the groups were compared in terms of alterations in cotinine levels and changes in family attitudes to smoking indoors.

Urinary cotinine level

Urinary cotinine levels were measured (in ng/ml) using an IMMULITE® 2000 (Siemens, USA) chemiluminescence assay employing an IMMULITE 2000 nicotine metabolite kit. The manufacturer indicated that 10 ng/ml was the limit for ETS exposure. A value below this threshold indicated "no ETS exposure" and a value of 10 ng/ml or greater indicated "ETS exposure."

Statistical analysis

The Statistical Package for the Social Sciences version 19.0 was used in statistical analyses. The χ^2 test

was used to compare grouped data. The paired *t*-test was used to compare group-specific measurements and the independent Student's *t*-test to compare measurements among independent groups. Pearson's correlation analysis was used to explore correlations between the measurements. *P*-values < 0.05 were considered statistically significant.

Results

Cotinine was present in the urine of 193 (81.4%) of the 237 children who participated in the study. The average cotinine level was 46.1 ± 57.9 ng/ml in the intervention group and 48.1 ± 54.1 ng/ml in the non-intervention group (Figure 2). We found no significant difference in early cotinine levels between the two groups ($p = 0.836$). Fifty-nine (30.5%) subjects did not attend their follow-up visits, including 32 (16.5%) from the intervention group and 27 (14%) from the non-intervention group. These subjects were excluded from the study. Application of the "intention to treat" test showed that the data of subjects who were excluded did not change the results.

The intervention group included 65 children: 46 (70.8%) males and 19 (29.2%) females. The non-intervention group consisted of 69 children: 52 (75.4%) males and 17 (24.6%) females. The average age of children in the intervention group was 24.4 ± 8.9 months, and the aver-

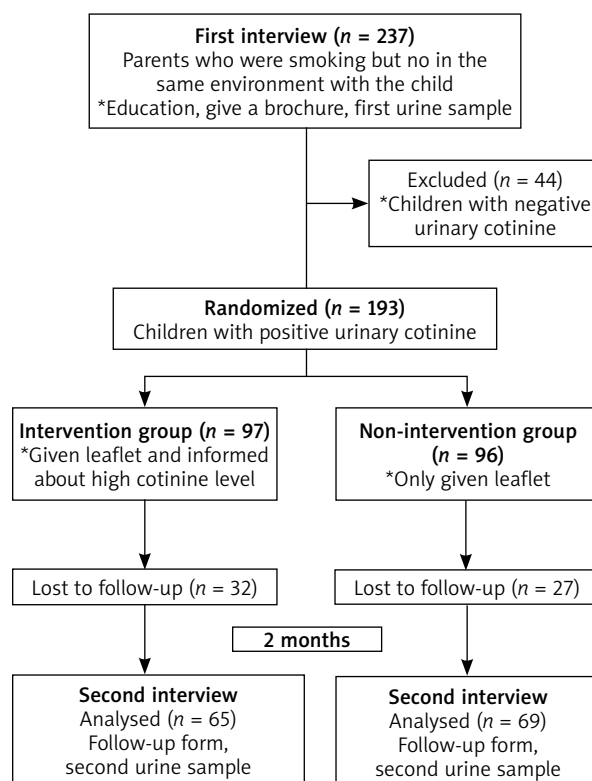


Figure 1. Study flow diagram

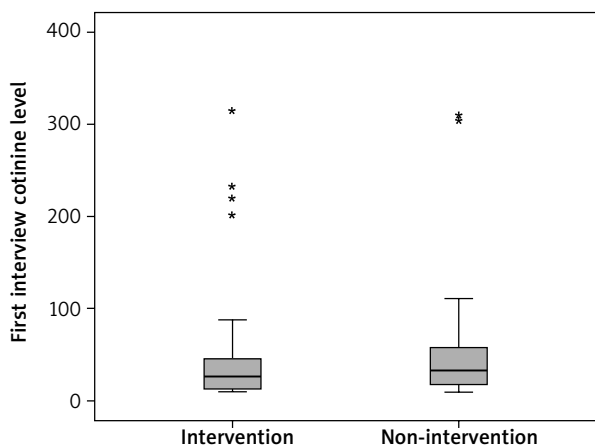


Figure 2. First interview cotinine level

age age in the non-intervention group 25.3 ±9.8 months. There was no significant difference between the intervention and non-intervention groups with respect to the sex of the children or the age or educational levels of the parents ($p = 0.549$, $p = 0.779$, $p = 0.292$ and $p = 0.733$, respectively) (Table 1). According to data obtained in the first survey, 87.6% of fathers and 52.3% of mothers in the intervention group, and 76.8% of fathers and 52.1% of mothers in the non-intervention group, were smokers. Mothers who smoked only in areas from which children were excluded constituted 32.3% of the intervention group, 37.9% of the non-intervention group, and 35.1%

in total. Fathers who smoked only in areas where children were not present constituted 81.5% of the intervention group, 73.9% of the non-intervention group, and 77.6% in total; exposure to fathers' smoking was thus twice that of exposure to mothers' smoking.

When the correlations between the number of cigarettes to which children were exposed and urinary cotinine levels were examined, it was clear that the more cigarettes smoked near children, the higher the level of cotinine in their urine ($p < 0.05$). Although the correlation between the urinary cotinine level and the number of cigarettes smoked daily by the father at home was low ($r = 0.193$), it was nonetheless statistically significant ($p = 0.04$). The correlation between the urinary cotinine level and the number of cigarettes smoked daily by the mother at home was high ($r = 0.559$), and the number of cigarettes the mother smoked daily at home was strongly associated with the cotinine level in children ($p < 0.001$). We found a moderate correlation between the urinary cotinine level and the number of cigarettes smoked daily at home ($r = 0.364$). As the number of cigarettes smoked daily at home increased, the cotinine level also rose ($p < 0.001$) (Table 2).

At the time of the second interview, the cotinine levels were 41.2 ±57.8 ng/ml in the intervention group and 40.8 ±43.3 ng/ml in the non-intervention group (Figure 3). Although the cotinine level of children in the intervention group was thus reduced at the time of the second interview, no significant difference was evident between the two surveys ($p = 0.355$). In the intervention group, significant decreases in the number of cigarettes

Table 1. The distribution of groups according to the results of the first survey

Parameter		Intervention group	Non-intervention group	P-value
Gender, n (%)	Boy	46 (70.8)	52 (75.4)	0.549
	Girl	19 (29.2)	17 (24.6)	
Age at admission [months]		24.4 ±8.9	25.3 ±9.8	0.779
Educational status of mothers', n (%)	Primary school	37 (56.9)	33 (47.8)	0.292
	High school	28 (43.1)	36 (52.2)	
Educational status of fathers', n (%)	Primary school	33 (50.8%)	33 (47.8)	0.733
	High school	32 (4.2%)	36 (52.2)	
Number of smoking fathers, n (%)		57 (87.6)	53 (76.8)	0.362
Number of smoking mothers, n (%)		34 (52.3)	36 (52.1)	0.510
Number of fathers smoking at home, n (%)		53 (81.5)	51 (73.9)	0.290
Number of mothers smoking at home, n (%)		21 (32.3)	26 (37.6)	0.515
Total number of cigarettes smoked at home daily		12.6 ±11.9*	11.5 ±11.9*	0.619
Number of cigarettes smoked by mother daily		11.4 ±7.7*	9.02 ±6.1*	0.336
Number of cigarettes smoked by father daily		16.8 ±10.3*	16.9 ±9.2*	0.408
Number of cigarettes smoked by mother at home daily		7.1 ±3.9*	5.9 ±4.1*	0.268
Number of cigarettes smoked by father at home daily		7.7 ±7.1*	6.3 ±4.7*	0.206

*Data are shown as mean ± standard deviation for normally distributed variables.

Table 2. Correlation between the number of cigarettes and the urinary cotinine level of children involved in the study

Variable	r	P-value
Number of cigarettes smoked by father at home daily	0.193	0.040
Number of cigarettes smoked by mother at home daily	0.559	< 0.001
Total number of cigarettes smoked by parents at home daily	0.364	< 0.001

Pearson correlation test.

that fathers smoked both daily and at home were evident ($p = 0.001$ and $p = 0.001$, respectively). Although the number of cigarettes smoked daily by mothers both at home and outside decreased, the decreases were insignificant ($p = 0.272$ and $p = 0.376$, respectively). Thus, although the cotinine level of children in the control group was lower at the time of the second interview than at the first, this change was not significant ($p = 0.101$). We also found no significant changes in the numbers of cigarettes that fathers and mothers smoked either outside or at home (both p -values > 0.05) (Table 3).

Twenty (14.9%) cases in whom cotinine levels became negative for ETS exposure and 114 (85.1%) in whom cotinine levels remained positive were compared in terms of parental behaviors that would lower ETS exposure. The only behavioral factor significantly related to a shift from positive to negative cotinine levels was a suggestion by one spouse to the other that he/she should quit smoking ($p = 0.021$). Apart from that, there was no significant difference between groups in terms of behavioral changes ($p > 0.05$) (Table 4). We found no correlation between

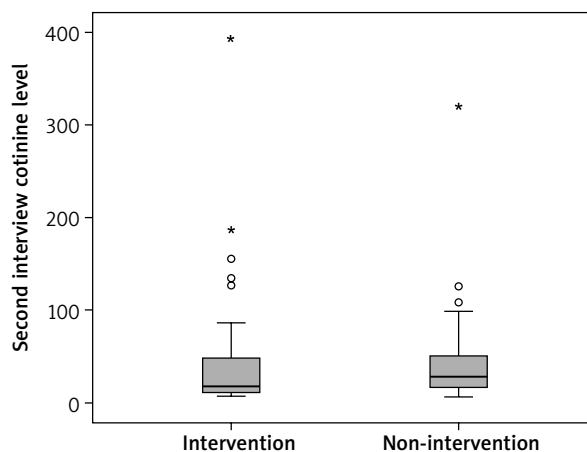


Figure 3. Second interview cotinine level

the educational levels of mothers ($r = 0.023$, $p = 0.791$) or fathers ($r = -0.036$, $p = 0.683$) of children related to continuous ETS exposure among the children.

Discussion

Although exposure of children to ETS is highest in developing countries, such exposure is relatively high, 29–69%, worldwide [9]. Urinary cotinine levels are used to evaluate ETS exposure levels in epidemiological research because the test is easy to use, accurate, and highly sensitive, and it can be performed more often than evaluation of serum cotinine levels, which require blood sampling [10–13]. To provide an objective measure of ETS exposure, we evaluated urinary cotinine levels. In line with previous research, we found no correlation

Table 3. Influence levels of environmental tobacco exposure in the first and second interviews of intervention and non-intervention groups

Variable	Group	First interview	Second interview	P-value
Cotinine level	Intervention (median, min–max)	46.14 ±57.99 (26.3, 10.1–314)	41.2 ±57.8 (18.4, 7–394)	0.355
	Non-intervention (median, min–max)	48.16 ±54.17 (33, 10.1–310)	40.8 ±43.3 (28.8, 7–321)	0.101
Number of cigarettes smoked by mother daily	Intervention	11.4 ±7.73	11.1 ±8.59	0.272
	Non-intervention	9.02 ±6.10	9.65 ±8.30	0.672
Number of cigarettes smoked by father daily	Intervention	16.8 ±10.3	14.5 ±9.01	0.001
	Non-intervention	16.9 ±9.23	15.3 ±8.62	0.190
Number of cigarettes smoked by mother at home	Intervention	7.12 ±3.94	6.19 ±4.66	0.376
	Non-intervention	5.89 ±4.05	5.69 ±4.38	0.927
Number of cigarettes smoked by father at home	Intervention	7.69 ±7.08	3.96 ±3.62	0.001
	Non-intervention	6.27 ±4.66	5.09 ±5.56	0.615

Data are shown as mean ± standard deviation for normally distributed variables.

Table 4. Distribution of the behaviour change executed by families of cases whose ETS exposure disappeared or continued

Variable	Cases whose ETS exposure continued n (%)	Cases whose ETS exposure disappeared n (%)	P-value
I did nothing	54 (47.4)	9 (45)	0.845
I took the child away from smoking area	14 (12.2)	4 (20)	0.350
I opened the window	3 (2.6)	0 (0)	0.463
I put up a “no smoking” notice	3 (2.6)	1 (5)	0.566
I told my spouse to smoke less	14 (12.3)	3 (15)	0.736
I told my spouse not to smoke at home	23 (20.2)	6 (30)	0.325
I told my spouse not to smoke near the child	15 (13.2)	1 (5)	0.299
I told my spouse to put off a cigarette before coming near the child	3 (2.6)	1 (5)	0.566
I recommended my spouse to quit smoking	6 (5.3)	4 (20)	0.021

between child urinary cotinine levels and parentally reported ETS exposure levels, suggesting that parents, consciously or unconsciously, do not acknowledge ETS exposure, or report incomplete information [14–17]. We also found high urinary cotinine levels in the children of parents who claimed that they never smoked when their children were nearby. In the present study, the ETS exposure level was 81.4%. Such a high level may be attributable to a lack of parental awareness rather than conscious untruth, with the parents not realizing that smoking at home, irrespective of whether the children are present, exposes the children to ETS.

Although parents may be aware of the damage caused by ETS to child health, it is very difficult to change smoking habits because smoking is addictive [18]. Interventions aimed at changing attitudes and behaviors in terms of smoking at home are important to reduce the incidence of respiratory tract diseases caused by ETS and to prevent ETS exposure in children. Many recent studies have investigated the effectiveness of various interventions. Some studies have suggested that informing parents about the urinary cotinine levels of their children is more influential than simple training. Wilson *et al.* [19] consulted mothers with asthmatic children on the levels of ETS exposure and then informed the mothers about their children’s urinary cotinine levels. In that study, information on cotinine levels was more successful in reducing admissions to hospital than a training-based intervention. Emmons *et al.* [20] determined that a limited intervention targeting parents who smoked and using only educational materials was not effective in decreasing the ETS exposure of children aged 3 years or younger. Wakefield *et al.* [21] studied households in South Australia where at least one parent smoked. The intervention involved informing parents by mail and telephone (twice) of the urinary cotinine levels of the children. They found that this approach was more successful than an intervention featuring only education, and it increased the fre-

quency of banning smoking at home. Ekerbicer *et al.* [22] also reported that imposition of smoking bans at home increased after parents were informed by mail about the urinary cotinine levels of their children. In a study of mothers of 108 children under 4 years of age, Hovell *et al.* [2] found that the number of weekly home exposures to cigarette smoking in the intervention group was significantly lower than that in the control group. Yücel *et al.* [23] showed in a study of 80 mothers that ETS exposure of their children decreased after either comprehensive or limited training interventions. Compliance with smoking bans at home was higher among mothers in the intervention groups. Wilson *et al.* [24] showed in a study of 519 asthmatic children that training the parents and informing them about cotinine levels were effective in reducing the frequency of child asthma attacks.

In studies comparing different approaches, it is very important that the groups exhibit similar distributions of features that may affect the results. The present study included patients who were followed up for episodic wheezing and whose parents stated that they did not smoke near their children. The children with positive urinary cotinine levels were randomly divided into two groups. We found no difference between the groups in terms of basic variables such as smoking status prior to intervention, sociodemographic features such as the gender of the children, or the average age or educational backgrounds of the parents. Thus, the two groups were statistically comparable. There was a significant decrease in the number of total cigarettes that the fathers smoked after learning about urinary cotinine levels, and also in the number of cigarettes smoked at home. This outcome is particularly crucial in terms of changing the smoking habits of fathers at home in Turkey, where the frequency of smoking is higher among men than among women.

The ETS exposure of 14.9% of the children involved in the study was eliminated during the study, as indicated by negative ETS test results. When we analyzed the behav-

iors of families in which ETS exposure was reduced, the frequency of suggesting that a spouse should quit smoking was significantly higher among the families of children whose ETS exposure was eliminated. This shows that education alone can be effective in decreasing ETS exposure. Although the urinary cotinine level became negative in a somewhat greater number of children in the intervention group, no significant difference was evident between the two groups in this regard. This highlights the need to reinforce our approach with recurrent follow-up interviews to enhance parental knowledge and awareness.

In the present study, we found a moderate correlation between the urinary cotinine level and the number of cigarettes smoked at home. As the number of cigarettes smoked at home increased, the urinary cotinine level also rose. Irvine *et al.* [10] found positive correlations of the number of cigarettes smoked at home and the number of smokers at home with the urinary cotinine levels of children. Keskinoglu *et al.* [9] reported that when the number of smokers at home and the number of cigarettes smoked at home increased, urinary cotinine levels in children rose significantly as children aged. Mannino *et al.* [25] found positive correlations of the number of cigarettes smoked and the number of smokers at home with increased cotinine levels in children. When the number of cigarettes smoked by mothers was compared with that smoked by fathers, the correlation between the number of cigarettes smoked by mothers and the urinary cotinine level of the children was higher. This finding shows that children in Turkey are at a greater risk of ETS exposure from mothers than from fathers who smoke, as the mothers are mostly homemakers and spend more time with their children than fathers.

The limitations of this study are that we did not investigate the effect of ETS exposure on airway inflammation or the frequency and severity of attacks in children subjected to ETS exposure and exhibiting episodic wheezing. Clinical follow-up of wheezing cases associated with ETS exposure will enable the effectiveness of various approaches to be evaluated in further studies.

Conclusions

To help control wheezing attacks, which are important causes of morbidity in childhood, ETS exposure must be prevented at home where children spend most of their time. As shown in the present study, ETS exposure cannot be reliably estimated based on the reports of parents. This study indicates that training families on the effects of ETS exposure is crucial; however, informing parents of the urinary cotinine levels of their children has not yet been very effective. More influential strategies are required to reveal the effects of ETS exposure to families more objectively. We consider that the cost of such strategies is of little importance when it is recognized that protection of even one wheezy child at risk from ETS exposure would save a great

deal of money. In high-risk cases, cotinine measurements should indeed continue to be considered.

Conflict of interest

The authors declare no conflict of interest.

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