

Original Article

Environmental tobacco smoke exposure in school children: parent report and urine cotinine measures

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Background: Environmental tobacco smoke (ETS) in the home continues to be a major health risk for children around the world. Measuring ETS is a central feature of clinical and epidemiological studies, with children's exposure often assessed through parental estimates. The authors examined the relationship between parent-reported estimates of children's exposure to ETS and children's urinary cotinine levels and evaluated the ETS exposure and its effect on respiratory health in children.

Methods: A total of 188 school children were included in the study. Parents were asked to complete a questionnaire about their smoking habits, their children's respiratory morbidity status and housing conditions. Urinary cotinine levels were measured in children.

Results: According to the responses, 72.3% of the children came from households with smokers, and 34.6% had daily exposure to ETS. When urine cotinine levels of >10 ng/mL were used as the yardstick of exposure, 76% of the children were identified as ETS exposed. No relation was detected between the symptoms of respiratory tract diseases and ETS exposure. To determine the amount of ETS exposure, the contribution of parental reports was low.

Conclusion: To evaluate the level of ETS exposure of children, the parents' reports were not reliable. The addition of a biological measure results in a more informative estimate of ETS exposure in children.

Key words

children, cotinine, environmental tobacco smoke.

Environmental tobacco smoke (ETS) is a term now widely used to refer to the mixture of side-stream smoke and exhaled mainstream smoke that pollutes air in locations where tobacco is being smoked.¹ ETS exposure adversely affects the health of both children and adults. Children who are disturbed by ETS may not always complain, they may be ignored or reprimanded when they express their complaint. Therefore, children are more likely to suffer from the impact of ETS exposure on health, compared to adults, and the house is the most important site of such exposure. It is now well established that children's exposure to ETS results in substantial public health and economic consequences.

It is reported that ETS is among the most important reasons for respiratory morbidity and mortality in children especially at the pre-school phase.^{2–6} Children exposed to ETS display a higher number of symptoms of cough, wheeze, respiratory ill-

nesses, decrease in lung function and increase in airway responsiveness.^{7–10} Furthermore, smoke exposure in childhood is associated with the early development of asthma, increased severity of asthma and the development of allergy, nasal and sinus diseases, dental caries, behavior problems and childhood cancer.^{11–14}

Various methods are utilized such as the monitorization of the environment air and self-reporting questionnaires for the purpose of determining ETS exposure at home. Although questionnaire forms are reliable in indicating the smoking data at home and the number of cigarettes smoked per day, the frequency of underreporting is high.^{15,16} The integrity of self-reported data, therefore, varies according to population and the social context in which the data are collected. Four factors influence the individual patient's responses to questions about smoking status; 'characteristics of the individual respondents, the method and setting of the encounter, cognitive demands imposed by the question and the motivation of the respondent as mediated by the social desirability of the subject of inquiry'.¹⁷ It was shown that especially in studies conducted on pregnant women, the rate of false reports is high.^{18,19} Similarly,

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the assessment of ETS exposure in children by means of a questionnaire results to have a low validity and reliability.²⁰ Therefore, the use of another indicator is suggested to be used for the purpose of enhancing the reliability when assessing ETS exposure.^{21–24} Cotinine is the most frequently used indicator among these. Cotinine is a major metabolite of nicotine and is considered the best measure of nicotine consumption. It has a half-life of approximately 20 h, superior sensitivity and specificity, and is not altered by environmental variables.²⁵ Cotinine may be studied in many biological samples such as blood, urine, saliva, cervical exudate, semen, meconium and hair.^{26–30} Being a non-invasive method, urinary cotinine study is among the most frequently used methods.

The aims of this study were to assess the concordance between parental self-reported tobacco uses, their children's urinary cotinine levels and to evaluate the ETS exposure and its effect on respiratory health in children.

Methods

The present study was designed as a cross-sectional study and the data were collected in May 2004. The study was carried out in a primary school in the Korfez (Gulf) District in Kocaeli, Turkey. In Korfez, there are many large industrial factories such as the petro-chemistry refinery (TUPRAS) and two fertilizer factories. Also, Korfez lies between two major motorways and all of these factors lead to a heavy air pollution which can affect the respiratory tract disease morbidity.

The most populated primary school was included in this study for the purpose of including a sufficient number of students. Only one school was selected in order to provide homogeneity in the study group and eliminate the variability of air pollution in different areas. All first three classes were included in the study without sampling. Older students were not included in the study, for the purpose of ruling out the probability of their smoking habits.

The dependent variable of the study is the cotinine level measured from urine which is an indicator of ETS. Urinary cotinine level was measured by Diagnostic Product Corporation (DPC) brand Immulite model device (DPC, Los Angeles, CA, USA), upon using the chemiluminescent method. Cotinine levels were calculated in terms of ng/mL. Since the cotinine level distribution was not fitting standard normal distribution ($P < 0.05$),

this was not used in the analysis as a continuous variable. The urinary cotinine variable was categorized into four groups as quartiles, which resulted in the values in Table 1.

The independent variables are living conditions (type of housing, number of persons per room, number of siblings), mother's education, socioeconomic status (father's education, car ownership as a proxy indicator of socioeconomic status), respiratory morbidity and nutritional status. Respiratory morbidity was investigated by cough, chronic cough, wheezing and upper acute respiratory infection during the last 2 weeks to rule out the negative impact of memory.^{31,32}

The data was collected using a questionnaire which had been supplied to the parents. Further examination of the children was performed by both anthropometric measurements and urine collection. The anthropometric measurements were entered on Epi Info Version 5 (Centers for Disease Control and Prevention, Atlanta, GA, USA) to calculate z score. A comparison was made between parental reports and urinary cotinine level.

Out of 258 students, 204 questionnaires were returned and the measurements were carried out on 188 students. The analysis was conducted on these 188 students and the participation rate was 72.9%.

The data was analyzed in the Statistical Package for the Social Sciences 11.5 (SPSS Inc., Chicago, IL, USA). Since the urine cotinine was not normally distributed, parametric tests (Student's *t*-test or one-way ANOVA) could not be performed. To figure out this obstacle, the cotinine level was divided into four categories (as mentioned above) by creating an ordinal variable that was progressively increasing. To analyze the association between this new variable and the other independent variables, χ^2 test for trend was performed, since it provides a more powerful test than the unordered independence tests in such situations. The test for trend is often used for dose-response studies and can also be used to test for trends with any ordered variable.^{33,34}

Results

Social and demographic status

The age range of the children participating in the study varied between 5.9–11.0 years, with the mean age being calculated as 8.1 ± 0.92 years. When evaluating the socioeconomic status of the families, it may be observed that the family heads were mostly blue-collar and white-collar workers, and 41.5% of them owned a car. When analyzing the other factors influencing the prevalence of respiratory tract diseases, it was determined that in 48.4% of the families, the number of persons per room in the house was more than 1, therefore, implying crowdedness, and that in 53.7% of the families, there were also other children in addition to the one who participated in this study (data not shown).

Table 1 Urinary cotinine variables

Cotinine groups	Urinary cotinine (ng/mL)
First quartile	<11.0
Second quartile	11.0–20.9
Third quartile	21.0–33.7
Fourth quartile	>33.7

Smoking behavior

When evaluating the questionnaires filled in by the families, it was observed that for 136 out of 188 children (72.3%) at least one family member was a smoker. For 69 children, only the father was a smoker (50.7%) while for 18 children, only the mother was a smoker (13.2%) and for 45 children, both the mother and the father were smokers (33.1%). When considering the smoking behavior within the house, despite the presence of smokers in the house, no smoking took place in the homes of 74 children (39.4%). Out of the families who declared that smoking took place in the home, 49 stated that (26.1%) no one smokes in the room where children are present, whereas the remaining 65 families (34.6%) expressed that smoking could take place in the room where children are present as well as in the other rooms, that is, in every corner of the house. Among the children who were reported to be present when smoking took place, 54 (83.1%) were subject to the smoke of 1–10 cigarettes per day while 16.9% were subject to the smoke of more than 10 cigarettes per day (Table 2).

The correlation of cotinine levels with other parameters

As a consequence of the measurement of the urinary cotinine levels in children, it was determined that the mean was 25.6 ng/mL \pm 21.6 (min–max, 0–163) and the median was 21.0 (Fig. 1).

There was no statistically significant relation determined between the education status of the parents of the children and the cotinine levels (Table 3).

When analyzing the relation between smoking habits and the cotinine levels in the urine, both ‘no smoker’ and ‘both’ groups were found to be making a difference; the number of

Table 2 Some characteristics of parents’ smoking behaviors

Variables	No.	%
Parental smoking status		
No smoker	52	27.7
Only father	69	36.7
Only mother	18	9.6
Both mother and father	45	23.9
Others	4	2.1
Smoking behavior in the home		
No smoker	52	27.7
No smoking in the home	22	11.7
Smoking in the home but not in the same room	49	26.1
Smoking both in same room and other rooms of the home	65	34.6
Number of cigarettes		
1–10	54	83.1
11 and higher	11	16.9

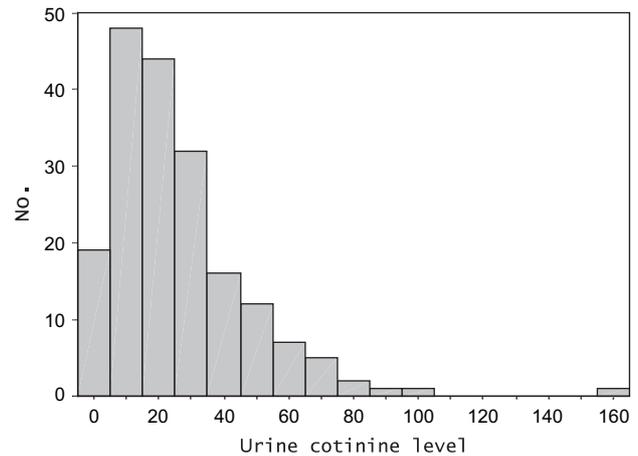


Fig. 1 Distribution of the urine cotinine level (ng/mL; mean, 25.6; median, 21.0; SD, 21.6).

children at the first quartile were higher in the ‘no smoker’ group, while the fourth quartile was higher in the ‘both smoking’ group ($P < 0.005$). There is a difference in the urinary cotinine levels of children in whose house there were no smokers and of children in whose house there were smokers but no one smoked in any part of the house ($P < 0.001$). Furthermore, for the children of families declaring to be smoking at home, no difference was detected between smoking in the room where the child was present and smoking in a room where the child was not present ($P > 0.05$; Table 4, Fig. 2).

No relation was detected between the symptoms of respiratory tract diseases and ETS exposure (Table 5).

Validity of parental reports

Validity and agreement of the parental reports are shown in Table 6. It was observed that the reliability of the declarations of the parents in the estimation of ETS exposure of the children was low. Only in 39% of the children exposed to ETS was it reported that they smoked in the presence of their children. The sensitivity of determining ETS exposure of smoking at home was higher (70%).

Discussion

Limitations of the study

This is a cross-sectional study conducted for the purpose of detecting the relation between the features related to ETS exposure in school children and the relation between this exposure and respiratory tract diseases. As participation rate is one of the most crucial characteristics in cross-sectional trials, the fact that the participation rate was 73% in our study means it is the most evident limitation. Despite the fact that the rate of responding families was 79%, all data were complete in 73% of

Table 3 The association between urine cotinine with the parental education

Variables	Cotinine groups				Total	Statistics
	First quartile	Second quartile	Third quartile	Fourth quartile		
Father's education						
≤8 years	24 (25.0%)	20 (20.8%)	26 (27.1%)	26 (27.1%)	96 (100.0%)	$\chi^2 = 0.350, P > 0.05$
>8 years	20 (21.7%)	29 (31.5%)	23 (25.0%)	20 (21.7%)	92 (100.0%)	
Mother's education						
≤8 years	30 (25.9%)	25 (21.6%)	29 (25.0%)	32 (27.6%)	116 (100.0%)	$\chi^2 = 0.184, P > 0.05$
>8 years	14 (19.4%)	24 (33.3%)	20 (27.8%)	14 (19.4%)	72 (100.0%)	

the urine collection phase. Therefore, the analyses were conducted in this group.

Another feature to be taken into account when evaluating the results of the study was the heavy air pollution observed in the area where the study was conducted. There is a renowned relation between atmospheric air pollution and respiratory diseases.³⁵⁻³⁸ A higher prevalence in respiratory diseases due to air pollution in the area and, therefore, the failure to detect the relation between ETS and respiratory diseases had been foreseen before the study. For the purpose of minimizing this negative aspect, this study was conducted in only one school instead of various schools at various distances and efforts were made in order to prevent the results from being influenced by this factor.

The rate of environmental tobacco smoke exposure

It is reported that in many countries throughout the world and especially in children, the rate of ETS exposure is very high. Therefore, ETS exposure is a major public health problem for every age group starting from childhood. It is reported that

38% of pre-school children in the USA are exposed to tobacco smoke at home, while this frequency is 50% in the UK and 57% in Northern European countries.³⁹⁻⁴¹ Despite the lack of sufficient epidemiological studies, it is believed that a high rate such as 75% of the children in Turkey are exposed to ETS.⁴²⁻⁴⁴ In this study, according to the reports of the parents, it was determined that 33.5% of mothers and 60.6% of fathers are smokers and that there is a third smoker in the house, in 2.1% of the group. Again, in accordance with the declaration of the parents, 11.7% do not smoke at all at home despite being cigarette addicts, 26.1% smoke at home but only in areas where the child is not present (such as a room or the kitchen), 34.6% smoke anywhere, including in the presence of their child. Therefore, in accordance with the declarations of the parents, the frequency of ETS exposure in children was reported as 34.6%. Although the cotinine level measured in the urine is an evident indicator of ETS exposure, no cut-off point was defined for ETS exposure. Therefore, it would not be possible to specify clearly the ETS exposure frequency of children over cotinine. But, when we take 10 ng/mL as the cut-off point, it may be observed that the rate of children with a urinary cotinine level of 10 ng/mL and below (first quartile) is

Table 4 The association between urine cotinine with the characteristics of environmental tobacco smoke

Variables	Cotinine groups				Total	Statistics
	First quartile	Second quartile	Third quartile	Fourth quartile		
Parental smoking status ^{††}						
No smoker	25 (48.1%)	22 (22%)	5 (9.6%)	— (0.0%)	52 (100.0%)	$\chi^2 = 73.833, P = 0.000$
Only father	13 (18.8%)	17 (24.6%)	22 (31.9%)	17 (24.6%)	69 (100.0%)	
Only mother	1 (5.6%)	5 (27.8%)	9 (50.0%)	3 (16.7%)	18 (100.0%)	
Both mother and father	4 (8.9%)	3 (6.7%)	13 (28.9%)	25 (55.6%)	45 (100.0%)	
Smoking behavior in the home						
No smoker [§]	25 (48.1%)	22 (42.3%)	5 (9.6%)	— (0.0%)	52 (100.0%)	$\chi^2 = 43.454, P = 0.000$
Smoker exist but not smoking in the home	6 (27.3%)	7 (31.8%)	4 (18.2%)	5 (22.7%)	22 (100.0%)	
Smoking in the home but not in the same room	4 (8.2%)	8 (16.3%)	18 (37.7%)	19 (38.8%)	49 (100.0%)	
Smoking both in same room and anywhere of the home	9 (13.8%)	12 (18.5%)	22 (35.1%)	22 (35.1%)	65 (100.0%)	

[†]Except the other groups ($n=4$).

^{††}The groups creating difference are 'no smoker' and 'both'.

[§]The group which makes a difference.

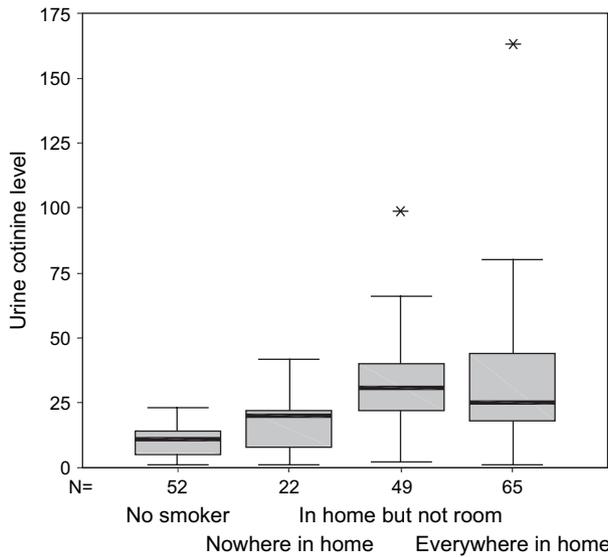


Fig. 2 The relationship between urine cotinine level and smoking behavior in the home.

24% while it is 11 ng/mL and above in the remaining 76% children, which is in line with the ETS exposure results obtained among children in Turkey. It is also observed that such a rate is significantly higher than the frequency reported in other countries.

Smoking behavior

Different results were reported in different countries in the studies investigating the relation between the level of educa-

tion and smoking habits. For instance, in a study conducted in the USA, it was reported that the use of cigarettes decreased as the level of education increased, whereas in a study carried out in France it was indicated that the rate of smoking grew with the increase in the level of education.^{45,46} In this study, no relation has been detected between the ETS measured with the cotinine level and the education level of the parents.

Smoking by both parents and mothers significantly correlated with ETS exposure in children, while both smoking resulted in higher cotinine levels. It is believed that this indicates that most of the mothers are housewives and spend most of their time at home. Actually, it has been displayed in many studies that there is a close relation between smoking mothers and ETS exposure in pre-school children.¹¹

When comparing ETS exposure measured with urinary cotinine level with data on smoking behavior at home, it was detected that the cotinine level in children exposed to ETS at home is significantly higher than the children in the house of whom there are no smokers. The cotinine levels of children whose parents have reported that they do not smoke in the presence of the child in the room is significantly higher than the children in whose house there are no smokers; this is an indicator that the smoke of the cigarettes smoked at home may also impact persons in the other rooms. The fact that the urinary cotinine levels of children in whose family there is at least one smoker but none of these are reported to be smoking at home is higher than the group where there are no smokers at home, brings under discussion the reliability of self-reports. These data indicate that the declaration of the parents is not reliable, or that tobacco smoke reaches the child although smoked outside the house or both. In the cohort study conducted by Johansson *et al.*, in comparison with pre-school children whose parents have never smoked, the ETS exposure

Table 5 The relationship between urine cotinine level and respiratory morbidity in the school children

Variables	Cotinine groups				Total	Statistics
	First quartile	Second quartile	Third quartile	Fourth quartile		
Upper ARI						
No	40 (25.0%)	40 (25.0%)	40 (25.0%)	40 (25.0%)	160 (100.0%)	$\chi^2=0.717, P>0.05$
Yes	4 (14.3%)	8 (28.6%)	9 (32.1%)	7 (25.0%)	28 (100.0%)	
Cough						
No	24 (20.9%)	31 (27.0%)	32 (27.8%)	28 (24.3%)	115 (100.0%)	$\chi^2=0.244, P>0.05$
Yes	19 (27.1%)	17 (24.3%)	17 (24.3%)	17 (24.3%)	70 (100.0%)	
Wheezing [†]						
No	29 (21.2%)	36 (26.3%)	35 (25.5%)	37 (27.0%)	137 (100.0%)	$\chi^2=3.202, P>0.05$
Yes	11 (32.4%)	10 (29.4%)	8 (23.5%)	5 (14.7%)	34 (100.0%)	
Chronic coughing [‡]						
No	35 (23.3%)	38 (25.3%)	43 (28.7%)	34 (22.7%)	150 (100.0%)	$\chi^2=0.752, P>0.05$
Yes	4 (14.8%)	9 (33.3%)	5 (18.5%)	9 (33.3%)	27 (100.0%)	
Unknown	5 (45.5%)	1 (9.1%)	1 (9.1%)	4 (36.4%)	11 (100.0%)	

[†]Except 17 students which are reported as ‘unknown’.

[‡]Except 11 students which are reported as ‘unknown’.
ARI, acute respiratory infection.

Table 6 Validity and agreement of the parental reports

	High (>10 ng/mL)	Low (≤10 ng/mL)	Total
Smoking in the same room [†]			
Yes	56	9	65
No	88	35	123
Total	144	44	188
Smoking in the home [‡]			
Yes	101	13	114
No	43	31	74
Total	144	44	188

[†]Sensitivity, 38.9%; Specificity, 79.5%; kappa, 0.114; $P < 0.000$.

[‡]Sensitivity, 70.1%; Specificity, 70.5%; kappa, 0.328; $P < 0.000$.

has been measured as 2 times higher in those smoking outside the house, 3.2 times higher than those smoking in the kitchen with the ventilator on, and 15 times higher than those smoking in the house.⁴⁷ In the studies carried out by Matt *et al.*, it was indicated that in comparison with the children of families who never smoked, the children of families who smoked outside the house or away from their children for the purpose of protecting them, were exposed to an ETS of 5–7 higher, that in the infants of the families smoking at home, the ETS exposure was 3–8 times higher than the children of smokers who tried to protect their children; as a consequence, it was reported that not smoking close to their infants or smoking outside slightly decreased ETS exposure but has not fully prevented it.⁴⁸

The fact that the result is not modified by smoking outside for the purpose of protecting the child or paying attention not to smoke in the room where the child is present and the lack of statistical difference in the cotinine levels in both cases is evaluated as the key result of the study.

Various results have been obtained in the studies aiming to protect children from ETS exposure upon explaining the damages of smoking on children to smoking families. Some researchers reported that despite the training provided to them, no significant changes took place in the smoking habits of parents with children who have been diagnosed with various respiratory tract diseases.^{49–51} While some indicated a decrease in the ETS exposure of infants whose mothers have received regular trainings.⁵²

Association of environmental tobacco smoke with respiratory tract diseases

Tobacco smoke disrupts the integrity of the epithel of the respiratory tract and mucosal activity, therefore, preparing the ground for respiratory tract infections.⁵³ Gurkan *et al.* have shown that acute tobacco smoke exposure prepares the ground for acute respiratory tract infections.⁵⁴ Furthermore, it has been indicated in many studies that ETS exposure plays a role in the

development of asthma, triggers asthma attacks and increases the severity of the attacks.^{11,55,56} Chen *et al.* concluded that children living in a smoking family were more likely to cough than those living in a non-smoking family.² Similarly, many studies have shown that there was an increased risk associated with parental smoking and respiratory symptoms among asthmatic and non-asthmatic children.^{8,9,57}

Although the association between ETS exposure and respiratory diseases was shown in many studies,^{9–11,56,57} the respiratory symptoms were not regarded as associated with ETS exposure in this study. The reason for not being able to detect a relation between ETS exposure and respiratory diseases may be that our study group lives in a heavily polluted industrial city. Furthermore, due to the fact that the majority of the students in our study have siblings and the transportation of viral upper respiratory tract infections among siblings may have rendered it more difficult to associate respiratory symptoms with ETS exposure. The fact that the number of cigarettes daily exposed by the majority of the children in our study group was below 10 may have led them to be less affected. As the studies were conducted on pre-school children and, therefore, the children spent more time at home, it is possible that they may have been affected more by tobacco smoke at home. Furthermore, we believe that the high smoking rate of mothers included into these studies may have increased the severity of the exposure in this age group which spends more time at home with their mother.

Validity and agreement of parental reports

In studies evaluating ETS exposure, it was emphasized that the reliability of parental reports was controversial and that various markers would be required in addition to parental reports for obtaining a more objective evaluation.^{10,22,58} While the tobacco exposure at home declared by the parents was 34.6%, this rate was detected as 76% by the urinary cotinine levels (when we took the value 10 ng/mL as the cut-off value in the urinary cotinine measurements), which brought the reliability of the parents' reports under discussion. In particular, the response given to the question regarding the habit of smoking next to the child was considered low in reliability. The results of this study support the proposal that parental reports should not be trusted when evaluating ETS exposure and that it would be necessary to measure cotinine levels in the urine, saliva and hair and, where possible, to detect chronic exposure upon measuring the house dust concentrations of nicotine at home, as suggested by Willers *et al.*²⁴

Conclusion

The ETS exposure rates of children in our country is very high. Parental statement of the exposure consistently results in

underreporting of the amount of cigarettes smoked in the presence of children, regardless of social or educational status. Parental education is of paramount importance to avoid ETS-related pediatric health problems. Our results indicate that the use of cotinine levels may provide tangible and objective evidence for ETS and may help evaluate the effectiveness of parental training programs aimed at decreasing smoking.

References

- Samet JM. Workshop summary: assessing exposure to environmental tobacco smoke in the workplace. *Environ. Health Perspect.* 1999; **107** (Suppl 2): 309–12.
- Chen Y, Rennie DC, Lockinger LA, Dosman JA. Effect of environmental tobacco smoke on cough in children with a history of tonsillectomy or adenoidectomy. *Eur. Respir. J.* 1998; **11**: 1319–23.
- Victora CG, Fuchs SC, Flores JA, Fonseca W, Kirkwood B. Risk factors for pneumonia among children in a Brazilian metropolitan area. *Pediatrics* 1994; **93**: 977–85.
- Berman S. Epidemiology of acute respiratory infections in children of developing countries. *Rev. Infect. Dis.* 1991; **13** (Suppl 6): S454–62.
- Fonseca W, Kirkwood BR, Victora CG, Fuchs SR, Flores JA, Misago C. Risk factors for childhood pneumonia among the urban poor in Fortaleza, Brazil: a case-control study. *Bull. World Health Organ.* 1996; **74**: 199–208.
- de Francisco A, Morris J, Hall AJ, Armstrong Schellenberg JR, Greenwood BM. Risk factors for mortality from acute lower respiratory tract infections in young Gambian children. *Int. J. Epidemiol.* 1993; **22**: 1174–82.
- Nafstad P, Kongerud J, Botten G, Hagen JA, Jaakkola JJ. The role of passive smoking in the development of bronchial obstruction during the first 2 years of life. *Epidemiology* 1997; **8**: 293–7.
- Jinot J, Bayard S. Respiratory health effects of exposure to environmental tobacco smoke. *Rev. Environ. Health* 1996; **11**: 89–100.
- Corbo GM, Forastiere F, Agabiti N *et al.* Passive smoking and lung function in alpha(1)-antitrypsin heterozygote schoolchildren. *Thorax* 2003; **58**: 237–41.
- Tutka P, Wielosz M, Zatonski W. Exposure to environmental tobacco smoke and children health. *Int. J. Occup. Med. Environ. Health* 2002; **15**: 325–35.
- Cook DG, Strachan DP. Health effects of passive smoking-10: summary of effects of parental smoking on the respiratory health of children and implications for research. *Thorax* 1999; **54**: 357–66.
- Aligne CA, Moss ME, Auinger P, Weitzman M. Association of pediatric dental caries with passive smoking. *JAMA* 2003; **289**: 1258–64.
- Weitzman M, Gortmaker S, Sobol A. Maternal smoking and behavior problems of children. *Pediatrics* 1992; **90**: 342–9.
- Sasco AJ, Vainio H. From in utero and childhood exposure to parental smoking to childhood cancer: a possible link and the need for action. *Hum. Exp. Toxicol.* 1999; **18**: 192–201.
- Kemmeren JM, van Poppel G, Verhoef P, Jarvis MJ. Plasma cotinine: stability in smokers and validation of self-reported smoke exposure in nonsmokers. *Environ. Res.* 1994; **66**: 235–43.
- Lewis SJ, Cherry NM, McL Niven R, Barber PV, Wilde K, Povey AC. Cotinine levels and self-reported smoking status in patients attending a bronchoscopy clinic. *Biomarkers* 2003; **8**: 218–28.
- Shaffer HJ, Eber GB, Hall MN, Vander Bilt J. Smoking behavior among casino employees: self-report validation using plasma cotinine. *Addict. Behav.* 2000; **25**: 693–704.
- DeLorenze GN, Kharrazi M, Kaufman FL, Eskenazi B, Bernert JT. Exposure to environmental tobacco smoke in pregnant women: the association between self-report and serum cotinine. *Environ. Res.* 2002; **90**: 21–32.
- Walsh RA, Redman S, Adamson L. The accuracy of self-report of smoking status in pregnant women. *Addict. Behav.* 1996; **21**: 675–9.
- Nafstad P, Botten G, Hagen JA *et al.* Comparison of three methods for estimating environmental tobacco smoke exposure among children aged between 12 and 36 months. *Int. J. Epidemiol.* 1995; **24**: 88–94.
- Patrick DL, Cheadle A, Thompson DC, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. *Am. J. Public Health* 1994; **84**: 1086–93.
- Cornelius MD, Goldschmidt L, Dempsey DA. Environmental tobacco smoke exposure in low-income 6-year-olds: parent report and urine cotinine measures. *Nicotine Tob. Res.* 2003; **5**: 333–9.
- Binnie V, McHugh S, Macpherson L, Borland B, Moir K, Malik K. The validation of self-reported smoking status by analysing cotinine levels in stimulated and unstimulated saliva, serum and urine. *Oral Dis.* 2004; **10**: 287–93.
- Willers S, Hein HO, Jansson L. Assessment of environmental tobacco smoke exposure: urinary cotinine concentrations in children are strongly associated with the house dust concentrations of nicotine at home. *Indoor Air* 2004; **14**: 83–6.
- Perez-Stable EJ, Benowitz NL, Marin G. Is serum cotinine a better measure of cigarette smoking than self-report? *Prev. Med.* 1995; **24**: 171–9.
- Seccareccia F, Zuccaro P, Pacifici R *et al.* Serum cotinine as a marker of environmental tobacco smoke exposure in epidemiological studies: the experience of the MATISS project. *Eur. J. Epidemiol.* 2003; **18**: 487–92.
- Poppe WA, Peeters R, Daenens P, Ide PS, Van Assche FA. Tobacco smoking and the uterine cervix: cotinine in blood, urine and cervical fluid. *Gynecol. Obstet. Invest.* 1995; **39**: 110–14.
- Vine MF, Hulka BS, Margolin BH *et al.* Cotinine concentrations in semen, urine, and blood of smokers and nonsmokers. *Am. J. Public Health* 1993; **83**: 1335–8.
- Derauf C, Katz AR, Easa D. Agreement between maternal self-reported ethanol intake and tobacco use during pregnancy and meconium assays for fatty acid ethyl esters and cotinine. *Am. J. Epidemiol.* 2003; **158**: 705–9.
- Woodruff SI, Conway TL, Edwards CC, Hovell MF. Acceptability and validity of hair collection from Latino children to assess exposure to environmental tobacco smoke. *Nicotine Tob. Res.* 2003; **5**: 375–85.
- Harrison LH, Moursi S, Guinena AH *et al.* Maternal reporting of acute respiratory infection in Egypt. *Int. J. Epidemiol.* 1995; **24**: 1058–63.
- Lanata CF, Quintanilla N, Verastegui HA. Validity of a respiratory questionnaire to identify pneumonia in children in Lima, Peru. *Int. J. Epidemiol.* 1994; **23**: 827–34.
- Schlesselman JJ. *Case-Control Studies*. Oxford University Press, New York, 1982.

- 34 Dean A. *Manual for Epi Info: A Word-Processing, Database, and Statistics Program for Public Health*. Center for Disease Control, Atlanta, 1996.
- 35 Ranzi A, Gambini M, Spattini A *et al*. Air pollution and respiratory status in asthmatic children: hints for a locally based preventive strategy. AIRE study. *Eur. J. Epidemiol.* 2004; **19**: 567–76.
- 36 Kim JJ, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, Ostro B. Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. *Am. J. Respir. Crit. Care Med.* 2004; **170**: 520–26.
- 37 Karita K, Yano E, Tamura K, Jinsart W. Effects of working and residential location areas on air pollution related respiratory symptoms in policemen and their wives in Bangkok, Thailand. *Eur. J. Public Health* 2004; **14**: 24–6.
- 38 Chauhan AJ, Johnston SL. Air pollution and infection in respiratory illness. *Br. Med. Bull.* 2003; **68**: 95–112.
- 39 Gergen PJ, Fowler JA, Maurer KR, Davis WW, Overpeck MD. The burden of environmental tobacco smoke exposure on the respiratory health of children 2 months through 5 years of age in the United States: Third National Health and Nutrition Examination Survey, 1988 to 1994. *Pediatrics* 1998; **101**: E8.
- 40 Jarvis MJ, Goddard E, Higgins V, Feyerabend C, Bryant A, Cook DG. Children's exposure to passive smoking in England since the 1980s: cotinine evidence from population surveys. *BMJ* 2000; **321**: 343–5.
- 41 Lund KE, Skrondal A, Vertio H, Helgason AR. To what extent do parents strive to protect their children from environmental tobacco smoke in the Nordic countries? A population-based study. *Tob. Control* 1998; **7**: 56–60.
- 42 Kalyoncu AF, Selcuk ZT, Enunlu T *et al*. Prevalence of asthma and allergic diseases in primary school children in Ankara, Turkey: two cross-sectional studies, five years apart. *Pediatr. Allergy Immunol.* 1999; **10**: 261–5.
- 43 Karadag B, Karakoc F, Ceran O, Ersu R, Inan S, Dagli E. Does passive smoke exposure trigger acute asthma attack in children? *Allergol. Immunopathol. (Madr)* 2003; **31**: 318–23.
- 44 Dagli E, Basaran M, Hayran O *et al*. Prevalence of asthma in two district around in İstanbul with different levels of air pollution (Abstract). *Eur. Respir. J.* 1993; **6**: 616.
- 45 Wagenknecht LE, Perkins LL, Cutter GR *et al*. Cigarette smoking behavior is strongly related to educational status: the CARDIA study. *Prev. Med.* 1990; **19**: 158–69.
- 46 Kauffmann F, Tager IB, Munoz A, Speizer FE. Familial factors related to lung function in children aged 6–10 years. Results from the PAARC epidemiologic study. *Am. J. Epidemiol.* 1989; **129**: 1289–99.
- 47 Johansson A, Hermansson G, Ludvigsson J. How should parents protect their children from environmental tobacco-smoke exposure in the home? *Pediatrics* 2004; **113**: e291–5.
- 48 Matt GE, Quintana PJ, Hovell MF *et al*. Households contaminated by environmental tobacco smoke: sources of infant exposures. *Tob. Control* 2004; **13**: 29–37.
- 49 Irvine L, Crombie IK, Clark RA *et al*. Advising parents of asthmatic children on passive smoking: randomised controlled trial. *BMJ* 1999; **318**: 1456–9.
- 50 McIntosh NA, Clark NM, Howatt WF. Reducing tobacco smoke in the environment of the child with asthma: a cotinine-assisted, minimal-contact intervention. *J. Asthma* 1994; **31**: 453–62.
- 51 Eriksen W, Sorum K, Bruusgaard D. Effects of information on smoking behaviour in families with preschool children. *Acta Paediatr.* 1996; **85**: 209–12.
- 52 Hovell MF, Zakarian JM, Matt GE, Hofstetter CR, Bernert JT, Pirkle J. Effect of counselling mothers on their children's exposure to environmental tobacco smoke: randomised controlled trial. *BMJ* 2000; **321**: 337–42.
- 53 Selwyn BJ. The epidemiology of acute respiratory tract infection in young children: comparison of findings from several developing countries. Coordinated Data Group of BOSTID Researchers. *Rev. Infect. Dis.* 1990; **12**(Suppl 8): S870–88.
- 54 Gurkan F, Kiral A, Dagli E, Karakoc F. The effect of passive smoking on the development of respiratory syncytial virus bronchiolitis. *Eur. J. Epidemiol.* 2000; **16**: 465–8.
- 55 Bjorksten B. The environmental influence on childhood asthma. *Allergy* 1999; **54** (Suppl 49): 17–23.
- 56 Saraclar Y, Sekerel BE, Kalayci O *et al*. Prevalence of asthma symptoms in school children in Ankara, Turkey. *Respir. Med.* 1998; **92**: 203–7.
- 57 Al-Dawood K. Parental smoking and the risk of respiratory symptoms among schoolboys in Al-Khobar City, Saudi Arabia. *J. Asthma* 2001; **38**: 149–54.
- 58 Callais F, Momas I, Roche D, Gauvin S, Reungoat P, Zmirou D. Questionnaire or objective assessment for studying exposure to tobacco smoke among asthmatic and healthy children: The French VESTA Study. *Prev. Med.* 2003; **36**: 108–13.