The Lancet, 29th July 1995, Page 280

Objective passive-smoking indicators and respiratory morbidity in young children

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Exposure to environmental tobacco smoke is associated with increased respiratory morbidity in young children, but few studies have assessed such exposure objectively by urinary cotinine measurements. 501 children aged 1–5 years, a random 5% sample of children attending an outpatient clinic, were classified as exposed or non-exposed to environmental tobacco smoke with a cut-off of 10 ng cotinine per mg creatinine in urine. Exposed children were 3·5 times (95% CI 1·56–7·90, p<0·0024) more likely to have increased respiratory morbidity (three or more episodes during the previous 12 months) than non-exposed children after adjustment for potential confounding factors.

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Several studies have shown an increased frequency of respiratory illnesses in young children exposed to environmental tobacco smoke. Most, however, have assessed this exposure through parental interviews, which may be susceptible to information bias.[1] Cotinine, the major metabolite of nicotine, has a long half-life in body fluids and is agreed to be the most appropriate indicator of exposure to environmental tobacco smoke among non-smokers.[2] Few studies have examined the association between body-fluid cotinine and respiratory morbidity in children; the results have been inconclusive.[3] Our study is one of the largest on this topic and was undertaken in Greece, which has the highest tobacco consumption per head in the European Union.[4]

During the 6 coldest months of 1992, 501 children aged 1–5 years, a random 5% sample of children attending an emergency outpatient clinic, were enrolled in the study. These children were brought to the clinic for various disorders but had not been referred by a physician for specific illness. Three investigators interviewed parents, examined the children, completed a precoded questionnaire, and collected a urine sample from each child. Information was obtained about patterns of smoking at home, socioeconomic status, housing, and demographic indicators as well as the child's respiratory morbidity. We defined respiratory morbidity by the number of episodes affecting the upper (rhinitis, tonsillitis, sinusitis, otitis media, laryngitis) or lower (bronchitis, bronchiolitis, asthma, bronchopneumonia, pneumonia) respiratory system, as reported by parents, which had been treated by a physician during the previous 12 months. We defined more than two such episodes as increased morbidity.

5 mL urine samples were immediately frozen, coded, and mailed to the American Health Foundation, Valhalla, New York, USA. Cotinine concentrations were measured by a radioimmunoassay[5] with a sensitivity of 1 ng per mg creatinine. Cotinine concentrations were standardised by adjustment for creatinine excretion.[6]

Urinary cotinine excretion ranged from not detectable to 388 ng per mg creatinine (mean 68·6 ng per mg); in 21 (4%) children no cotinine was detectable (<2 ng per mg). Urinary cotinine in children was significantly related to self-reported parental smoking (mean 23·7 [SD 41·7] ng per mg creatinine for no-smoking parents; 57·5 [62·6] ng per mg for father-only smoking; 70·1 [62·7] ng per mg for mother-only smoking; 114 [83·4] for both parents smoking; r_s=0·54, p<0·0001). In multiple regression analyses, the number of smokers at home and the number of cigarettes smoked in the child's presence were positively associated with urinary cotinine, whereas the child's age showed an inverse association.

Number of episodes	Number of children		
	Upper	Lower	Total
0	147	354	101
1	190	169	114
2	89	39	95
3	65	21	68
4	35	8	45
5	17	9	26
6	6	4	23
>7	12	3	29

Table 1: Distribution of respiratory episodes

Respiratory morbidity	Number of children with increased morbidity		Crude odds ratio	Adjusted odds ratio (95% CI)*	p
	Exposed	Not exposed			
Total	180	11	3·07	3·50 (1·56–7·90)	0·0024
Upper	127	8	2·03	2·00 (1·13–8·03)	0·0280
Lower	45	2	3·30	7·73 (0·95–62·8)	0·0554

* Adjusted for age (in single years), sex, day care attendance, history of chronic respiratory disease in at least one parent, number of residents per m² in the home, and socioeconomic status (manual vs non-manual).

Table 2: Results of logistic regression analysis of respiratory morbidity versus exposure to tobacco smoke

Children exposed to environmental tobacco smoke were distinguished from non-exposed children with a cut-off of 10 ng cotinine per mg creatinine as recommended previously.[6] The distribution of the number of respiratory illnesses was skewed (table 1); the mean numbers of episodes were 2·36 for total, 1·70 for upper, and 0·65 for lower respiratory morbidity.

Children exposed to environmental tobacco smoke were 3·5 times more likely to have had increased respiratory morbidity than non-exposed children (table 2). The risk was increased for upper and lower respiratory morbidity separately. There was no confounding influence of day-care attendance, history of chronic respiratory illness in at least one parent, overcrowding, or socioeconomic status on the association between exposure to environmental tobacco smoke and increased respiratory morbidity (tables giving full details of logistic regression analyses are available from *The Lancet*).

We have confirmed that passive smoking objectively assessed through urinary cotinine measurements significantly and substantially increases respiratory morbidity in young children. The results appear applicable to both upper and lower respiratory morbidity, although the number of children with increased lower respiratory morbidity was too small for the association to achieve significance. The upper respiratory tract may be more sensitive to environmental tobacco smoke, since direct exposure induces cellular alterations in the production of mucus, which predispose to frequent illness.[7]

This study of a large number of randomly selected children without specific respiratory problems confirms that environmental tobacco smoke is an important preventable cause of early childhood respiratory morbidity.

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The Lancet, 29th July 1995, Page 280

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