

Letter to the Editor

Parental smoking increases the risk for eczema with sensitization in 4-year-old children

To the Editor:

Parental smoking is one of the proposed risk factors for eczema, but results from previous studies appear inconsistent,¹⁻³ and to our knowledge, none of these have divided eczema into atopic and nonatopic forms. We used data from a Swedish prospective birth cohort (BAMSE) to investigate the association between fetal/early infancy exposure to tobacco smoke and eczema at 4 years of age divided into atopic eczema (AE) and non-AE.

The BAMSE study includes 4,089 children born between 1994 and 1996 in Stockholm, Sweden. The children were consecutively recruited to the project at one of their first child health center visits in their first months of life. Access to a community population register ensured that all infants born in the area were included. The cohort has been described in detail elsewhere.^{4,5} At baseline, when the children were about 2 months old, the parents answered a questionnaire focusing on parental allergic disease and environmental factors, including parental smoking habits. At 1, 2, and 4 years of age, new questionnaires contained questions on symptoms and signs of eczema, as well as the child's environmental exposures.

Exposure to tobacco smoke was divided into *maternal smoking during pregnancy*, which was defined as the mother smoking at least 1 cigarette per day during any trimester, and *parental smoking*, which was defined as any parent smoking at least 1 cigarette per day when the child was about 2 months old. *Eczema* was defined as itchy rash for at least 2 weeks with typical distribution and dry skin, a doctor's diagnosis of eczema, or both. This definition has proved to have high sensitivity (92%) and specificity (100%) compared with clinical diagnosis by a dermatologist.⁴ Serum IgE antibodies to inhalant and food allergens were analyzed with Phadiatop and fx5 mixes of relevant inhalant and food allergens, respectively (Pharmacia UniCAP System; Phadia AB, Uppsala, Sweden). Sensitization required a positive Phadiatop, fx5, or both result. Eczema was divided into AE (eczema according to the definition plus sensitization at 4 years of age) and non-AE (eczema according to the definition but no sensitization). Only children with answers to all 4 questionnaires and complete information on exposure to tobacco smoke and covariates, as well as an available measure of serum IgE ($n = 2505$) were included in these analyses.

Statistical analyses were made with the Stata Statistical Software, Release 7.0 (StataCorp, College Station, Tex). Logistic regression models adjusting for established risk factors (parental allergic disease, breast-feeding, furred pets at home, sex, and parental education) were used. Because the analyses had 4 levels of outcome (ie, no eczema and no sensitization, sensitization without eczema, non-AE, and AE), a multinomial logistic regression model was used. The resulting odds ratios (ORs) were interpreted as the odds of being sensitized, having non-AE, and having AE, respectively, relative to the odds of having neither eczema nor sensitization. Proportions and ORs are shown with 95% CIs. A logistic regression model was used, with the categorical exposure variable as continuous, to test for a linear trend over categories.

Maternal smoking during pregnancy was reported in 12% of families, and parental smoking during the child's first months was reported in 20% of families. In 4% of families the mother had smoked during pregnancy without reported smoking during the child's first months, whereas in 12% of families, only smoking during the child's first months had occurred. Smoking both during pregnancy and during the child's first months was reported in 8% of families. There were no differences in the prevalence of potential confounders between the study population and the 4,089 children who answered the baseline questionnaire (data not shown).

Of the 2505 children included in the study, 529 (21%) reportedly had symptoms meeting the definition of eczema at 4 years of age. Of these, 8% had AE, and 13% had non-AE.

Both maternal smoking during pregnancy and parental smoking during the child's first months increased the risk for AE (OR, 1.82 [95% CI, 1.22-2.73] and OR, 1.62 [95% CI, 1.15-2.30], respectively) but not for non-AE (OR, 0.97 [95% CI, 0.65-1.44] and OR, 1.02 [95% CI, 0.75-1.40], respectively) or sensitization (OR, 0.89 [95% CI, 0.62-1.28] and OR, 1.10 [95% CI, 0.83-1.45], respectively) at 4 years of age. When separating prenatal and postnatal exposure, we observed a statically significant increased risk for AE only among children exposed to both maternal smoking during pregnancy and parental smoking during the child's first months (Table I). As shown in Table II, a dose-response effect was indicated for parental smoking during the child's first months, as well as for maternal smoking during pregnancy, although it was more consistent for postnatal exposure.

Both maternal and paternal smoking increased the risk for AE (OR, 2.01 [95% CI, 1.24-3.28] and OR, 1.46 [95% CI, 1.00-2.13], respectively). When separating these exposures from each other, only the effect of maternal smoking reached significance (Table III).

The present study has several advantages. First, the prospective design, following children from birth with annual questionnaires, minimizes recall bias concerning eczema in the child. Second, the first questionnaire containing questions on parental smoking was in most cases completed very early in the child's life and in 86% of cases by both parents. Thus the risk of the child's disease status influencing the answers on the questions concerning parental smoking or recall bias from informant parents regarding smoking in the other parent is low. Third, a validated definition of eczema with high sensitivity and specificity was used.⁴

One limitation of our study is that all data on smoking were self-reported. However, a strong correlation between reported smoking and residential air nicotine concentrations has been reported,⁶ and maternal recall of smoking during pregnancy has been shown to be reproducible and accurate.⁷ Another limitation is that blood samples and complete information on exposure to tobacco smoke and covariates were available only from 61% of the children in the original cohort, leaving 2505 for the analyses of AE and non-AE. However, selection bias occurs only if the relation between environmental tobacco smoke and eczema is different among those who were included and those eligible for the study.

The mechanism behind the increased risk for AE in children of smoking parents is not clear. There is evidence that maternal smoking could have *in utero* effects on immune development,⁸⁻¹⁰

TABLE I. Risk for sensitization, non-AE, and AE at 4 years of age in relation to maternal smoking during pregnancy and parental smoking during the child's first months

Smoking exposure		Sensitization without eczema					Non-AE				AE			
Pregnancy*	Child's first months*	Total no.	No.	Percent	OR†	95% CI	No.	Percent	OR†	95% CI	No.	Percent	OR†	95% CI
No	No	1902	303	15.9	1.00‡		256	13.5	1.00‡		137	7.2	1.00‡	
Yes	No	98	14	14.3	0.87	0.48-1.57	10	10.2	0.77	0.39-1.52	11	11.2	1.56	0.79-3.05
No	Yes	303	54	17.8	1.19	0.85-1.65	36	11.9	0.97	0.66-1.42	27	8.9	1.39	0.89-2.18
Yes	Yes	202	30	14.9	0.95	0.61-1.46	25	12.4	1.08	0.67-1.72	27	13.4	2.14	1.33-3.46

AE, Eczema plus sensitization at 4 years; Non-AE, eczema without sensitization at 4 years.

*One or more cigarettes per day smoked by the mother during any trimester or by any parent during the child's first months.

†Adjusted for sex, parental allergic disease, breast-feeding, pets at home, and parental education.

‡Reference group: children without sensitization or eczema.

TABLE II. Risk for AE at 4 years of age in relation to quantity of maternal smoking during pregnancy and parental smoking during the child's first months

AE*										
Maternal smoking during pregnancy						Parental smoking during child's first months				
	Total no.	No.	Percent	OR	95% CI	Total no.	No.	Percent	OR	95% CI
No smoking	2205	164	7.4	1.00†		2000	148	7.4	1.00†	
Total of 1-10 cigarettes/d	212	26	12.3	1.85‡	1.16-2.94	303	30	9.9	1.44‡	0.94-2.21
Total of ≥11 cigarettes/d	88	12	13.6	1.77‡	0.91-3.46	202	24	11.9	1.95‡	1.19-3.19
P for trend				.007					.003	

*Eczema plus sensitization at 4 years.

†Reference group: children without sensitization or eczema.

‡Adjusted for sex, parental allergic disease, breast-feeding, pets at home, and parental education.

TABLE III. Risk for AE at 4 years of age in relation to maternal and paternal smoking during child's first months

Smoking exposure			AE*			
Maternal†	Paternal†	Total no.	No.	Percent	OR‡	95% CI
No	No	2000	148	7.4	1.00§	
Yes	No	94	13	13.8	2.03	1.06-3.87
No	Yes	317	29	9.2	1.37	0.89-2.11
Yes	Yes	94	12	12.8	2.24	1.14-4.40

*Eczema plus sensitization at 4 years.

†One or more cigarettes per day smoked by the mother or father, respectively, during the child's first months.

‡Adjusted for sex, parental allergic disease, breast-feeding, pets at home, and parental education.

§Reference group: children without sensitization or eczema.

and environmental tobacco smoke exposure might influence the immune system after birth. There are studies suggesting that both prenatal and postnatal tobacco smoke exposure increase the risk of allergic immune responses.¹¹

In conclusion, our prospective study including 2505 Swedish boys and girls indicates that maternal smoking during pregnancy, parental smoking during the child's first months, or both increase the risk for AE, but not non-AE, in 4-year-old children. Decreased exposure to parental smoking might reduce the prevalence of AE in young children.

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