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Exposure to parental smoking in childhood is associated with persistence of respiratory symptoms into young adult life

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Abstract

Exposure to environmental tobacco smoke (ETS) has been established as a significant risk factor for respiratory symptoms and diseases in children. However, it is unclear whether the effects of childhood exposure to ETS track into adult life. By using data from the long-term population-based cohort of the TESAOD study, this study found that parental ETS exposure in childhood significantly increases the risk of persistent cough, chronic cough, and wheeze into young adult life.

Keywords

environmental tobacco smoke; wheeze; cough

To the Editor

According to estimates by the Office of the Surgeon General, nearly 60% of children in the U.S. are exposed at some point to environmental tobacco smoke (ETS).[1] Parental smoking is a child's primary source of ETS exposure.

Exposure to prenatal smoke and ETS during childhood have been established as significant risk factors for respiratory symptoms and diseases like wheeze, cough, asthma, and lung function impairment during childhood[1, 2], possibly through complex gene-by-environment interactions[3]. Among others, our group has previously shown an association between smoking intensity in less educated mothers and incident asthma in the offspring before age

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12 years.[4] However, to date long-term respiratory birth cohorts[5-7] have provided only limited and somewhat inconsistent results on the impact of childhood ETS exposure on respiratory health into adulthood. The goal of the present study was to use the long-term cohort of the Tucson Epidemiological Study of Airway Obstructive Disease (TESAOD) to determine whether childhood exposure to ETS has effects on respiratory symptoms that track into young adult life.

Using census data for Tucson, AZ, 1655 Anglo-white households were enrolled in TESAOD in 1972-3 using a multistage stratified cluster approach. All subjects living in the household were invited to complete clinic visits and questionnaires at baseline upon enrollment and in up to 12 follow-up surveys taken approximately every 2 years subsequently. Children's questionnaires were completed by the parent until the child was 15 years old. After age 15, the subject self completed questionnaires.

Child's ETS exposure was constructed from parents' survey questionnaire responses. If a parent reported current smoking between 0-15 years of a child's life, the child was coded as exposed to ETS. Personal smoking status was assessed through self-report questionnaires at age 18 and older. Positive smoking status (ever versus never) was defined by at least one report of current smoking at age ≥ 18 years during the follow-up. Objective measurements of exposure to ETS or active smoking, such as cotinine level, were not available.

Skin prick tests (SPTs) for house dust mix, Bermuda grass, tree mix, weed mix, and Dematiaceae mold mix (Hollister-Stier Laboratories, Spokane, WA) were completed in up to 5 surveys and positive SPTs were defined as having a wheal ≥ 3mm larger than the control for at least one tested allergen.

Respiratory symptoms and asthma data were collected from questionnaires. Each survey included questions on whether the subject had cough or wheeze. Chronic cough was defined as coughing at least 3 months out of the year.

For statistical analyses, categorical longitudinal variables were generated using prospective data through childhood (defined as < 15 years old) and adulthood (defined as ≥ 18 years old) for each of the following respiratory symptoms: cough, chronic cough, and wheeze. Four categories were defined as follows:

1. The subject did not have the symptom in childhood or adulthood (never)
2. The subject did not have the symptom in childhood but developed it in adulthood (incident)
3. The subject had the symptom in childhood but not in adulthood (remittent)
4. The subject had the symptom in both childhood and adulthood (persistent)

Multinomial logistic models were used to assess the relationship of childhood ETS exposure as an independent variable to the above longitudinal categories as the dependent variable for each of the respiratory outcomes while adjusting for age, sex, total number of surveys completed in adulthood, parental education, and personal smoking.

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There were 412 subjects in TESAOD who 1) were enrolled in the study at survey 1 during childhood, 2) transitioned into adulthood during the study follow-up, and 3) had data on parental ETS exposure during childhood.

As compared with the 205 subjects who were <15 years old at enrollment but did not have follow-up data available in adulthood, the 412 subjects included in the present study were older at enrollment, were more likely to be female, to have non-smoking parents, to have mothers with more years of education, and to have asthma at enrollment, and were less likely to report coughing (Table E1).

Among the 412 subjects included in this study, 55.6% were exposed to any parental smoking, 31.3% to maternal smoking, and 46.7% to paternal smoking. Subjects exposed to parental ETS in childhood were more likely to have mothers and fathers with less years of formal education than subjects with no ETS exposure (Table 1). Subjects exposed to parental ETS in childhood were also more likely to be active smokers in adulthood (Table 1). No significant differences in rates of asthma or respiratory symptoms in childhood were found in relation to parental ETS, although exposed children tended to have higher rates than did children with non-smoking parents.

Results from multinomial models adjusted for age, sex, personal smoking in adult life, total surveys in adulthood, and parental education are presented in Table 2 for each of the longitudinal outcomes. Parental ETS exposure in childhood was associated in adulthood with increased risk for incident cough (RR 1.9, $p = 0.038$) and incident chronic cough (2.7, $p=0.003$), and for persistent cough (RR 5.4, $p<0.001$), persistent chronic cough (3.4, $p=0.022$), and persistent wheeze (1.8, $p=0.028$). Comparable effects of paternal and maternal smoking were found on persistent cough, although paternal smoking tended to have stronger effects on persistent chronic cough and persistent wheeze as compared with maternal smoking. Further adjustment for parental asthma and pack-years of personal smoking did not modify results of parental ETS exposure on persistent outcomes (data not shown). Parental ETS exposure had no effects on SPTs ($OR_{\text{any-parental-smoking}}= 1.05$, $p=0.82$, $OR_{\text{maternal-smoking}}=0.88$, $p=0.603$, $OR_{\text{paternal-smoking}}= 1.04$, $p=0.869$) nor were results from the above models substantially changed when adjusting for SPTs (data not shown).

Consistent with previous studies[8], in multivariate logistic regression analyses predicting personal smoking in adulthood (Table E2) we found parental smoking to increase the likelihood of engaging in active smoking as an adult. Therefore, in order to rule out potential residual confounding by personal smoking, we also stratified models by active smoking as reported in adulthood (Tables E3a and E3b). Although these stratified analyses had a relatively small sample size and associations with cough tended to be stronger among ever smokers, any parental ETS exposure was still significantly associated with both persistent cough and persistent wheeze among never smokers. In our study, childhood asthma was also inversely related to active smoking in adult age (Table E2) and sensitivity analyses were completed after stratification by asthma status. Among the 318 participants with no asthma, ETS exposure remained significantly associated with persistent cough (RR 4.0, $p = 0.01$)

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and a similar trend, although not significant, was found for persistent wheeze (1.9, p=0.07). Associations among participants with asthma were not tested because of insufficient power.

In summary, in this study we found consistent effects of parental ETS exposure in childhood on persistence of respiratory symptoms into adult life. These effects were confirmed both after adjustment for and after stratification by personal smoking in adult life, indicating that they are unlikely to be due to residual confounding by active smoking.

Our study provides prospective evidence that the harmful effects of ETS exposure on respiratory health in childhood track into young adult life and expands results from previous cross-sectional studies that found adults suffering from chronic cough, wheezing, and obstructive lung diseases to be more likely to report retrospectively ETS exposure in childhood.

Some points should be taken into account in interpreting these results. There were some differences between participants who were followed into adulthood and those who were lost to follow-up (Table E1). However, while these differences may limit the generalizability of our findings they are unlikely to have impacted their internal validity. We could not assess the effects of maternal smoking during pregnancy[5] (as that information was unavailable) and we had limited power to dissect the individual contributions of maternal versus paternal smoking. Indeed, in our study paternal ETS exposure appeared to have stronger effects on persistence of respiratory symptoms than maternal ETS exposure did, but these findings should be interpreted with caution because fathers had higher numbers of cigarettes smoked and pack years than did mothers and because the sample sizes of these sub-group analyses were small. Finally, we only assessed exposure to parental smoking based on questionnaire data and did not have cotinine levels or other objective measures of ETS exposure. However, while community smoking contributes to ETS exposure in childhood, parents are the primary source of ETS exposure for their children and the reported number of cigarettes smoked in the home is an important predictor of cotinine levels in children[9]. This may be particularly true for the years (early 1970s) when TESAOD was initiated and smoking indoors was more socially acceptable than it is currently. This also suggests that parental smoking in our cohort may represent a stronger ETS exposure as compared with parental smoking in cohorts that were initiated in more recent years.

Among the strengths of this study are the population-based design, the long-term follow-up, the longitudinal assessment of exposure – which minimizes recall bias – and the prospective categorization of symptoms from childhood into adult age.

In conclusion, exposure to parental smoking in childhood increases significantly the risk of persistent respiratory symptoms into young adult life.

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Capsule Summary:

Long term respiratory *sequelae* of childhood exposure to environmental tobacco smoke remain controversial. This study found exposure to parental smoking in childhood to increase the risk of persistent cough, chronic cough, and wheeze into young adulthood.

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Table 1

Characteristics of subjects by any parent ETS exposure status

	Any Parent ETS Exposure		
	Yes (n=229)	**No (n=183)**	**p value***
Age at enrollment: mean (SD)	8.5 (4.5)	7.9 (4.7)	0.216
Sex: % male	51.1	50.8	0.957
Parent years of formal education:			
% with >12 years			
Mothers (n=403)	33.9	56.0	<0.001
Fathers (n=360)	41.8	59.0	0.001
Asthma in childhood: %	19.2	14.2	0.179
Cough in childhood: %	41.1	34.4	0.169
Chronic cough in childhood: %	18.8	13.1	0.121
Wheeze in childhood: %	49.3	44.8	0.360
Personal smoking in adulthood			
Ever (n=178): %	53.3	30.6	<0.001
Never (n=234): %	46.7	69.4	
Pack years (among ever smokers, n=176): mean (SD)	7.9 (6.7)	4.8 (5.6)	0.003

n is 412 unless otherwise specified

* p value for the comparison between the two groups

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Table 2

Relative risks and 95% CIs associated with the longitudinal patterns of respiratory symptoms by dichotomous (never, ever) ETS exposure type , adjusted for age, sex, total surveys in adulthood, parent education, and personal smoking

	Any	Maternal	Paternal
Cough	n=411	n=402	n=359
Never	Reference	Reference	Reference
Incident	1.9 (1.03-3.31)*	1.8 (0.98-3.35)	1.3 (0.72-2.48)
Remittent	1.0 (0.53-1.81)	1.0 (0.47-1.99)	1.1 (0.57-2.05)
Persistent	5.4 (2.47-11.73)**	2.2 (1.13-4.38)*	3.3 (1.60-6.71)**
Chronic Cough	n=411	n=402	n=359
Never	Reference	Reference	Reference
Incident	2.7 (1.41-5.26)**	1.2 (0.65-2.36)	3.6 (1.80-7.37)**
Remittent	1.3 (0.65-2.58)	1.0 (0.47-2.28)	1.8 (0.87-3.63)
Persistent	3.4 (1.20-9.66)*	0.9 (0.35-2.51)	3.0 (1.13-7.96) *
Wheeze	n=412	n=403	n=360
Never	Reference	Reference	Reference
Incident	1.3 (0.69-2.36)	1.2 (0.62-2.26)	1.5 (0.76-2.86)
Remittent	1.0 (0.52-1.90)	1.2 (0.60-2.51)	1.1 (0.55-2.31)
Persistent	1.8 (1.07-3.03)*	0.9 (0.52-1.65)	2.3 (1.31-3.99)**

* p<0.05,

** p<0.01

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Table E1

Characteristics at enrollment of participating children who transitioned into adulthood during the study follow-up (n=412) and those who did not (n=205)

	Adult Follow-up n=412	No adult Follow-up n=205	p-value
Age at initial survey, mean (SD)	8.3 (4.6)	5.7 (4.1)	<0.001
Sex, male %	51.0	62.4	0.007
Father smoked (n=513), % current	38.3	64.7	<0.001
Mother smoked (n=595), % current	26.1	42.7	<0.001
Father educated (n=513)			
> 12 years, %	49.7	43.8	0.219
Mother educated (n=595)			
> 12 years, %	43.9	22.9	<0.001
Cough, %	9.7	16.1	0.021
Wheeze, %	34.2	32.2	0.615
Asthma (n=616), %	10.9	4.9	0.014

*n=617 unless specified

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Table E2

Logistic regression model predicting personal smoking in adulthood

	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Age at initial survey	1.10 (1.05, 1.15)	<0.001	1.07 (1.02, 1.12)	0.01
Mother ETS exposure	2.60 (1.69, 4.00)	<0.001	1.84 (1.09, 3.11)	0.02
Father ETS exposure	2.42 (1.57, 3.72)	<0.001	2.12 (1.31, 3.42)	0.002
Childhood Asthma	0.47 (0.27, 0.82)	0.008	0.33 (0.17, 0.66)	0.002

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Table E3a

Relative risks and 95% CIs associated with the longitudinal patterns of respiratory symptoms by dichotomous (never, ever) ETS exposure type , adjusted for age, sex, total surveys in adulthood, parent education among *never smokers*

	Any	Maternal	Paternal
Cough	n=234	n=234	n=214
Never	Reference	Reference	Reference
Incident	1.5 (0.65-3.30)	1.4 (0.56-3.65)	1.0 (0.44-2.52)
Remittent	0.7 (0.35-1.47)	0.6 (0.24-1.45)	0.8 (0.39-1.78)
Persistent	4.4 (1.58-12.00)**	1.2 (0.42-3.16)	2.6 (1.01-6.53)*
Chronic Cough	n=234	n=234	n=214
Never	Reference	Reference	Reference
Incident	2.5 (0.83-7.71)	1.3 (0.36-4.82)	3.4 (1.05-10.96)*
Remittent	1.0 (0.44-2.12)	0.5 (0.18-1.54)	1.3 (0.56-2.88)
Persistent	2.5 (0.59-10.44)	0.4 (0.04-3.07)	2.4 (0.60-9.26)
Wheeze	n=234	n=234	n=214
Never	Reference	Reference	Reference
Incident	1.3 (0.49-3.23)	0.8 (0.23-2.68)	1.3 (0.45-3.59)
Remittent	1.3 (0.61-2.80)	1.4 (0.56-3.32)	1.9 (0.83-4.31)
Persistent	2.2 (1.13-4.10)*	1.0 (0.46-2.19)	2.4 (1.22-4.89)*

* p<0.05,

** p<0.01

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Table E3b

Relative risks and 95% CIs associated with the longitudinal patterns of respiratory symptoms by dichotomous (never, ever) ETS exposure type , adjusted for age, sex, total surveys in adulthood, parent education among *ever smokers*

	Any	Maternal	Paternal
Cough	n=177	n=168	n=145
Never	Reference	Reference	Reference
Incident	2.4 (1.02-5.85)*	2.5 (1.04-5.91)*	1.9 (0.74-4.79)
Remittent	2.3 (0.64-7.98)	2.0 (0.55-7.07)	2.2 (0.65-7.83)
Persistent	7.8 (2.28-26.49)**	4.5 (1.64-12.30)**	5.3 (1.67-16.79)**
Chronic Cough	n=177	n=168	n=145
Never	Reference	Reference	Reference
Incident	2.9 (1.25-6.56)*	1.4 (0.64-2.88)	4.0 (1.62-9.88)**
Remittent	5.3 (0.62-45.35)	3.6 (0.80-16.06)	9.4 (1.07-83.29)*
Persistent	5.1 (1.06-24.65)*	1.5 (0.44-4.92)	4.5 (1.05-19.37)*
Wheeze	n=178	n=169	n=146
Never	Reference	Reference	Reference
Incident	1.1 (0.45-2.64)	1.3 (0.58-3.06)	1.5 (0.60-3.82)
Remittent	0.5 (0.13-1.62)	0.9 (0.26-3.21)	0.2 (0.05-1.09)
Persistent	1.2 (0.50-3.13)	0.9 (0.36-2.12)	2.0 (0.75-5.35)

* $p<0.05$,

** $p<0.01$