

## ARTICLE

# Health Effects Related to Environmental Tobacco Smoke Exposure in Children in the United States

## Data From the Third National Health and Nutrition Examination Survey

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**Objective:** To determine the effects of prenatal and postnatal smoke exposure on the respiratory health of children in the United States.

**Design:** Nationally representative cross-sectional survey, including questionnaire information, measurements of serum cotinine (a metabolite of nicotine), and pulmonary function measurement, of 5400 US children.

**Setting and Participants:** Children aged 4 to 16 years in the Third National Health and Nutrition Examination Survey, October 25, 1988, to October 15, 1994.

**Methods:** We stratified the study participants into tertiles, on the basis of serum cotinine levels, and used logistic and linear regression modeling, adjusting for known covariates, to determine the effect of high environmental tobacco smoke (ETS) exposure (on the basis of a high cotinine level) on outcomes such as the prevalence of current asthma, the prevalence of frequent wheezing, school absence, and lung function. For children aged 4 to 11 years, we also determined the effect of prenatal maternal smoking on these outcomes.

**Results:** We observed effects of ETS exposure in all age groups, although the effects varied between age groups. Among all children significant effects associated with high cotinine levels were for wheezing apart from cold in the past year (odds ratio [OR], 1.8; 95% confidence interval [CI], 1.1-2.8); 6 or more days of school absence in the past year (OR, 2.0; 95% CI, 1.4-2.8); and lung function decrements in the forced expiratory volume in 1 second (mean change, -1.8%; 95% CI, -3.2% to -0.4%) and the maximal midexpiratory flow (mean change, -5.9%; 95% CI, -8.1% to -3.4%). Although current and ever asthma were not significantly associated with high cotinine levels in the overall group (OR, 1.5; 95% CI, 0.8-2.7, and OR, 1.3; 95% CI, 0.8-2.2, respectively), they were increased significantly among 4- to 6-year-old children (OR, 5.3; 95% CI, 2.2-12.7, and OR, 2.3; 95% CI, 1.1-5.1, respectively).

**Conclusions:** We investigated recent ETS exposures as important predictors of respiratory health outcomes in children 4 years and older. Environmental tobacco smoke exposure affects children of all ages, although the exact effects may vary between age groups.

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**E**NVIRONMENTAL tobacco smoke (ETS) exposure among children, which can begin before birth and continue through childhood, is an important and preventable cause of morbidity among children. Recent comprehensive reviews by the California Environmental Protection Agency<sup>1</sup> and Cook and Strachan,<sup>2</sup> in the journal *Thorax*, have concluded that ETS exposure increases respiratory symptoms and disease and decreases lung function in children.

Most studies that have examined the health effects of ETS exposure on children have used reported ETS exposure or the presence of smokers in the child's household to define exposure.<sup>3-5</sup> A limitation of these studies is that most chil-

dren in the United States are exposed to ETS,<sup>6</sup> thus children in the "unexposed" category in these studies can have exposures from nonparental sources or in places other than the home. Use of the biomarker, cotinine, can potentially reduce misclassification, allowing one to compare a high-exposure group with a low-exposure group.<sup>7</sup>

Our study analyzed data among children aged 4 through 16 years, from the Third National Health and Nutrition Examination Survey (NHANES III), a nationally representative study of the US population. We used serum cotinine levels as the main basis for classifying children into ETS exposure groups. We also determined whether prenatal maternal smoking affected these outcomes.

## SUBJECTS AND METHODS

### STUDY POPULATION

The NHANES III was conducted from 1988 through 1994 by the National Center for Health Statistics of the Centers for Disease Control and Prevention, Atlanta, Ga.<sup>8</sup> The NHANES III was approved by the National Center for Health Statistics' Institutional Review Board. In this survey, a stratified multistage clustered probability design was used to select a representative sample of the civilian, noninstitutionalized US population. Eighty-one geographic sites were included in the final sample. Survey participants completed extensive questionnaires in the household and a comprehensive physical examination, including pulmonary function testing, at a specially equipped mobile examination center. Questionnaires for participants younger than 17 years were completed by a knowledgeable proxy.

### SUBJECTS AND DEMOGRAPHICS

For our study, the analysis was limited to children aged 4 through 16 years for whom serum cotinine levels were obtained (cotinine levels were not obtained on children younger than 4 years). The NHANES III participants underwent a physical examination, including pulmonary function testing for children 8 years and older. In addition, we excluded children who either reported current smoking or had cotinine levels higher than 113.6 nmol/L, indicating possible current use of cigarettes or spit tobacco.<sup>9</sup>

### VARIABLE DEFINITION

The race/ethnicity of the participants was classified as "non-Hispanic white," "non-Hispanic black," "Mexican American," or "other" and was determined by self-report on the questionnaire. We excluded children of "other" race from the analyses because of the small number of subjects in this category. Socioeconomic status was determined to be "low" if the reference adult in the family (one of the persons who owns the home or pays the rent) had a 12th-grade education or less, or the poverty index for the family was less than 1 (on the basis of the number of people living in home and reported household income).<sup>9</sup> Family size was classified as 5 or more or 4 or less. If either the father or mother of the child reported asthma or hay fever at any age, the child was classified as having a parental history of allergy or asthma. Participants were classified as having a prenatal exposure to smoke if the respondent reported that the mother smoked at any time while pregnant with the child (only asked for children 11 years or younger). Respondents were asked "Has a doctor ever told you that your child has asthma?" and "Does your child still have asthma?" We classified children with a positive response to the first question as "ever asthma" and the second as "current asthma." We classified participants as having recurrent wheeze symptoms if the respondent reported 3 or more episodes of wheezing in the past year. We classified participants as having wheezing apart from a cold if the respondent reported this at any time in the previous year. We also classified participants as having 5 or fewer or 6 or more school absence days in the previous year. For most analyses, we stratified participants into 3 age strata: 4 to 6 years, 7 to 11 years, and 12 to 16 years.

### PULMONARY FUNCTION DATA

Spirometry was conducted on survey participants 8 years and older using a dry rolling seal spirometer in the mobile examination center. Procedures for testing were based on the 1987 American Thoracic Society recommendations.<sup>9</sup> To obtain spirometry acceptable according to the protocol, 5 to 8 forced expirations were performed. Several measures of lung function were used: the forced expiratory volume in 1 second (FEV<sub>1</sub>), the forced vital capacity (FVC), the maximal midexpiratory flow (MMEF) (determined by calculating the mean flow per second from 25% to 75% of the lung volume), and the FEV<sub>1</sub>/FVC ratio. We defined participants with an FEV<sub>1</sub>/FVC ratio of less than 0.80 as having low lung function for use in categorical analyses.

### COTININE LEVELS

Serum cotinine levels were determined using high-performance liquid chromatography atmospheric-pressure chemical ionization tandem mass spectrometry, as is described elsewhere.<sup>9</sup> We stratified the subjects into tertiles, based on cotinine levels of 0.28 nmol/L (the limit of detection; subjects with no detectable cotinine were included in this tertile) to 0.59 nmol/L, 0.60 to 3.23 nmol/L, and 3.24 to 113.6 nmol/L. The same cotinine strata were used in all age strata analyses. An estimated level of 0.19 nmol/L (one half of the level of detection divided by the square root of 2) was used for subjects with no detectable cotinine level when calculating mean exposure levels in the study subjects.

### ANALYSIS

We calculated all estimates using the sampling weight to represent children aged 4 to 16 years in the United States. The purpose of the sampling weight is to provide population estimates that adjust for unequal probabilities of selection and account for nonresponse. The weights were poststratified to the US population as estimated by the Bureau of the Census. For analyses, we used both SAS and SUDAAN, programs that adjust for complex sample design when variance estimates are calculated.<sup>10,11</sup> We did separate analyses both stratified by the 3 age groups specified earlier and grouping all children together, but controlling for age. Using logistic regression, we modeled factors predicting participants with low lung function, 6 or more school absences in the past year, ever asthma, current asthma, 3 or more episodes of wheezing in the past year, or any wheezing apart from colds during the past year. Each model was adjusted for race/ethnicity, socioeconomic status, family size, and parental history of asthma. Each model was evaluated for evidence of effect modification and confounding. We ran separate models in the 2 younger age groups including variables for cotinine tertiles both in the presence and in the absence of maternal prenatal smoking (this was not reported in children 12 years and older) in an attempt to better define the effect that maternal prenatal smoking had on the outcomes in children. For evaluation of continuous lung function data (FEV<sub>1</sub>, FEV<sub>1</sub>/FVC, FVC, and MMEF) and days of school absence, we developed linear regression models adjusting for age, sitting height, sex, race/ethnicity, socioeconomic status, parental history of allergy or asthma, family size, and prenatal maternal smoking and cotinine levels.

Table 1. Covariates and Outcomes Stratified by Age Groups\*

Covariates or Outcomes	Age, y			4-16 (N = 5400)
	4-6 (n = 1533)	7-11 (n = 2225)	12-16 (n = 1642)	
Cotinine level				
Lowest tertile	27.0	36.5	32.9	33.9
Middle tertile	32.2	30.3	35.7	32.7
Highest tertile	40.8	33.3	31.4	33.3
Race/ethnicity				
White	70.5	74.4	72.3	72.9
Black	17.5	16.2	17.8	17.1
Mexican American	12.0	9.4	9.9	10.0
Male sex	52.3	51.2	51.6	51.5
Lower SES	61.2	59.4	60.7	60.2
Family size $\geq 5$	45.9	45.9	43.8	45.1
Maternal prenatal smoking	20.0	19.4	...	19.6
Asthma or allergy in a parent	28.3	24.3	26.9	26.1
Current asthma	6.0	8.2	9.4	8.2
Ever asthma	8.9	10.9	12.4	11.1
FEV <sub>1</sub> /FVC <0.8	...	10.2	14.9	12.7
$\geq 3$ Episodes of wheezing in past year	7.1	7.5	7.8	7.5
Wheezing apart from cold in past year	13.7	9.5	7.8	9.6
$\geq 6$ Days of school absence in past year	18.6	27.3	30.3	26.9

\*SES indicates socioeconomic status; FEV<sub>1</sub>, forced expiratory volume in 1 second; FVC, forced vital capacity; and ellipses, not available. All values are given as percentages.

Table 2. Covariates and Outcomes by Cotinine Tertiles for All Ages\*

Covariates or Outcomes	Tertile Levels			Total (N = 5400)
	Highest (n = 1841)	Middle (n = 1927)	Lowest (n = 1632)	
Race/ethnicity				
White	70.8	69.5	78.5	72.9
Black	24.4	19.0	7.7	17.1
Mexican American	4.8	11.6	13.9	10.0
Male sex	50.3	51.5	52.9	51.5
Lower SES	77.6	57.5	45.2	60.2
Family size $\geq 5$	45.0	42.3	47.9	45.1
Maternal prenatal smoking	41.3	10.7	5.1	19.6
Asthma or allergy in a parent	26.7	21.6	29.7	26.1
Current asthma	9.9	7.2	7.7	8.2
Ever asthma	12.3	10.3	10.7	11.1
FEV <sub>1</sub> /FVC <0.8	18.2	10.2	10.3	12.7
$\geq 3$ Episodes of wheezing in past year	8.3	7.0	7.3	7.5
Wheezing apart from cold in past year	12.4	7.7	8.8	9.6
$\geq 6$ Days of school absence in past year	33.6	23.9	22.9	26.9

\*SES indicates socioeconomic status; FEV<sub>1</sub>, forced expiratory volume in 1 second; and FVC, forced vital capacity. All values are given as percentages.

## RESULTS

Of the 13944 children aged 2 months through 16 years who participated in NHANES III, 4419 were younger than 4 years. Of the remaining 9525 children, 3711 did not have serum cotinine levels obtained, 272 were of "other" race, and an additional 142 either admitted to current smoking or had cotinine levels higher than 113.6 nmol/L, leaving 5400 children available for analysis. The 3711 children who did not have cotinine levels obtained were similar to the 5400 participants with regard to sex, race, socioeconomic status, reported ETS exposure, and parental history of allergy or asthma ( $P > .05$ , for all), but were overrepresented in the youngest age group (52% for 4-6 years, vs 21% and 18% for 7-11 and 12-16 years, respectively) ( $P < .01$ ).

Overall, cotinine was detected in 84.9% (SE, 1.5%) of the children. Children aged 4 through 6 years had a mean cotinine level of 6.25 nmol/L (SE, 0.59 nmol/L) and a median cotinine level of 1.65 nmol/L. Corresponding levels for children aged 7 through 11 years were 4.54 nmol/L (SE, 0.34 nmol/L) and 1.31 nmol/L, and for children aged 12 through 16 years were 5.34 nmol/L (SE, 0.57 nmol/L) and 1.36 nmol/L.

The distribution of the covariates and outcomes are displayed in **Table 1** by age and in **Table 2** by cotinine tertile. The covariates were fairly similar across age strata, whereas the outcomes of current asthma, ever asthma, and 6 or more days of school absence tended to increase in older children and reported wheezing apart from colds tended to decrease in older age strata. Con-

Table 3. Outcomes Among Children, Using Logistic Regression Models\*

Outcome	Cotinine Tertile†	Fully Adjusted ORs (95% CIs) by Age Group, y			
		4-6‡ (n = 1533)	7-11‡ (n = 2225)	12-16‡ (n = 1642)	4-16§ (N = 5400)
Current asthma	High	5.3 (2.2-12.7)	0.8 (0.3-2.0)	1.7 (0.7-7.3)	1.5 (0.8-2.7)
	Medium	1.3 (0.4-3.7)	1.7 (0.8-3.4)	0.7 (0.3-1.7)	1.1 (0.6-1.9)
Ever asthma	High	2.3 (1.1-5.1)	0.8 (0.4-1.8)	1.5 (0.7-3.3)	1.3 (0.8-2.2)
	Medium	0.7 (0.3-1.6)	1.6 (0.9-2.6)	0.8 (0.4-1.8)	1.1 (0.7-1.7)
≥3 Episodes of wheezing in past year	High	3.8 (1.7-8.3)	1.5 (0.6-3.7)	0.9 (0.3-2.2)	1.3 (0.8-2.1)
	Medium	1.8 (0.8-4.5)	1.6 (0.9-3.1)	0.7 (0.3-1.6)	1.1 (0.7-1.6)
Wheezing apart from cold in past year	High	4.8 (2.4-9.9)	1.5 (0.7-3.3)	0.9 (0.3-2.2)	1.8 (1.1-2.8)
	Medium	1.6 (0.5-4.9)	1.1 (0.6-2.2)	0.7 (0.3-2.0)	1.0 (0.6-1.6)
≥6 Days of school absence in past year	High	1.7 (0.8-3.4)	1.5 (0.9-2.4)	3.2 (1.8-5.7)	2.0 (1.4-2.8)
	Medium	1.3 (0.6-2.8)	0.9 (0.5-1.4)	1.5 (0.9-2.4)	1.1 (0.8-1.5)
FEV <sub>1</sub> /FVC < 0.8‖	High	...	1.7 (0.7-4.0)	2.0 (0.9-4.3)	1.8 (1.3-2.4)
	Medium	...	0.8 (0.9-1.7)	1.1 (0.6-2.1)	1.0 (0.7-1.4)

\*ORs indicates odds ratios; CIs, confidence intervals; FEV<sub>1</sub>, forced expiratory volume in 1 second; FVC, forced vital capacity; and ellipses, not available.

†The referent group is the children in the lowest tertile of cotinine exposure.

‡Adjusted for race/ethnicity, socioeconomic status, parental history of asthma, family size, and sex.

§Adjusted for age, race/ethnicity, socioeconomic status, parental history of asthma, family size, and sex.

‖Lung function was not available for 7-year-old children.

versely, the covariates of low socioeconomic status and prenatal smoke exposure were much higher among children in the highest cotinine tertile than among those in the lowest tertile (Table 2).

Logistic regression models with outcome measures of current asthma, ever asthma, 3 or more wheezing episodes in the past year, wheezing apart from cold in the past year, 6 or more school absences in the past year varied by age group, with only the latter 2 outcomes being significant in all the children (Table 3).

Any level of prenatal maternal smoking was reported among 20.0% of children aged 4 through 6 years and 19.4% of children aged 7 through 11 years (Table 1). In our models, which included cotinine tertiles both with and without prenatal maternal smoking, among children aged 4 through 6 years, a high cotinine level was associated with an increased prevalence of current asthma and wheezing, regardless of the presence or absence of prenatal maternal smoking (Figure 1). A similar analysis in 7- through 11-year-old children yielded no significant results (data not shown).

Children with the highest cotinine levels were more likely to have an FEV<sub>1</sub>/FVC ratio of less than 0.8 than were children with the lowest cotinine level (Table 3). After adjustment for all covariates, the FEV<sub>1</sub>, the FEV<sub>1</sub>/FVC ratio, and the MMEF were significantly decreased in all children (Table 4). Environmental tobacco smoke exposure was also significantly associated with lung function decrements among 8- through 11-year-olds without prenatal maternal smoking (Table 4). The distribution of FEV<sub>1</sub>/FVC ratios was shifted toward a smaller ratio among children in the highest cotinine tertile (Figure 2).

School absence days, after adjustment for age, race/ethnicity, sex, socioeconomic status, family size, and parental history of asthma, were significantly increased among children in the highest cotinine tertile (mean increase in annual days absent, 2.5; 95% confidence interval [CI], 1.1-3.9). The odds of missing 6 or more days of school were significantly increased among all children (odds ratio [OR], 2.0; 95% CI, 1.4-2.8).

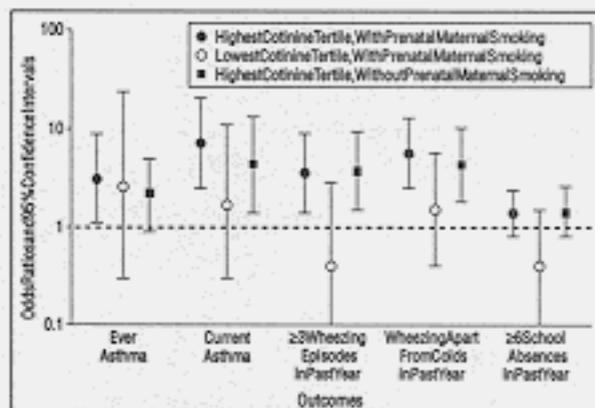


Figure 1. Odds ratios and 95% confidence intervals for 4- to 6-year-old children in the highest cotinine tertile with prenatal maternal smoking, in the lowest cotinine tertile with prenatal maternal smoking, and in the highest cotinine tertile with no prenatal maternal smoking, compared with those in the lowest cotinine tertile with no prenatal maternal smoking, using data from the Third National Health and Nutrition Examination Survey, 1988 through 1994. Model is adjusted for race/ethnicity, socioeconomic status, parental history of asthma, and family size. Children in the middle cotinine tertile were excluded to simplify the figure.

## COMMENT

We analyzed NHANES III data to determine the effects of ETS exposure on the respiratory health of children. Rather than depending on parental reports of exposure, which have been the basis of most analyses looking at the health effects of ETS exposure,<sup>1,2</sup> we used serum cotinine levels to classify children and compared those with the highest exposure with those with the lowest exposure. As is consistent with other studies, our strongest effects were in the youngest children, with ETS exposure being associated with an increased risk for ever and current asthma and wheezing.<sup>4,12,13</sup> Among older children, though, we found significant associations between ETS exposure and increased school absence and low lung function. Our data suggest that recent ETS

Table 4. The Mean FEV<sub>1</sub>, FVC, FEV<sub>1</sub>/FVC Ratio, MMEF, and, From Linear Regression Models, the Estimate of the Effect of Environmental Tobacco Smoke Exposure and Prenatal Maternal Smoking\*

Age Group, y	Parameter	Mean	Partially Adjusted,† Mean Effect, % (95% CI)	Fully Adjusted,‡ Mean Effect, % (95% CI)
Regression Among 3294 Children				
8-16	FEV <sub>1</sub> , mL	2477	-2.1 (-3.6 to -0.6)	-1.8 (-3.2 to -0.4)
	FVC, mL	2844	-0.7 (-2.3 to 0.9)	-0.3 (-1.6 to 1.2)
	FEV <sub>1</sub> /FVC, %	87.5	-1.4 (-2.1 to -0.7)	-1.5 (-2.2 to -0.8)
	MMEF, mL	2901	-5.7 (-8.2 to -3.2)	-5.9 (-8.1 to -3.4)
Regression Among 324 Children With Prenatal Maternal Smoke Exposure				
8-11	FEV <sub>1</sub> , mL	1901	0.0 (-3.3 to 3.3)	0.8 (-2.8 to 4.4)
	FVC, mL	2208	1.2 (-2.0 to 4.8)	1.3 (-2.1 to 4.7)
	FEV <sub>1</sub> /FVC, %	86.3	-1.4 (-3.4 to 0.6)	-0.8 (-2.6 to 1.0)
	MMEF, mL	2201	-1.2 (-10.3 to 7.9)	1.8 (-7.0 to 9.7)
Regression Among 1363 Children Without Prenatal Maternal Smoke Exposure				
8-11	FEV <sub>1</sub> , mL	1966	-1.9 (-3.5 to -0.3)	-1.7 (-3.5 to 0.1)
	FVC, mL	2250	-0.6 (-2.4 to 1.2)	0.0 (-1.8 to 1.8)
	FEV <sub>1</sub> /FVC, %	87.8	-0.9 (-1.7 to -0.1)	-1.3 (-2.3 to -0.3)
	MMEF, mL	2351	-6.6 (-11.0 to -2.2)	-7.4 (-11.4 to -3.4)

\*CI indicates confidence interval; FEV<sub>1</sub>, forced expiratory volume in 1 second; FVC, forced vital capacity; and MMEF, maximal midexpiratory flow.

†Adjusted for age, sitting height, sex, and race/ethnicity.

‡Adjusted for age, sitting height, sex, race/ethnicity, socioeconomic status, parental history of allergy or asthma, and family size.

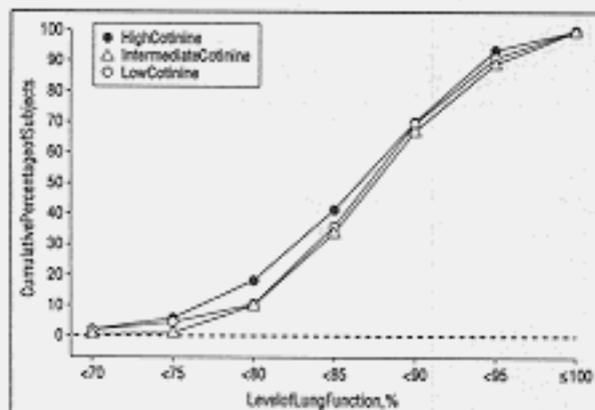


Figure 2. Level of lung function, as determined by the forced expiratory volume in 1 second to forced vital capacity ratio, among children aged 8 through 16 years, stratified by cotinine tertile, using data from the Third National Health and Nutrition Examination Survey, 1988 through 1994.

exposures are important even among children without prenatal smoke exposure.

Cotinine is an accurate marker not only for ETS exposure<sup>7</sup> but also for particulate air pollution exposure.<sup>14</sup> The relation between mean serum cotinine level (P), in nanomolar per liter (nmol/L), and the 24-hour average particulate matter 2.5  $\mu$ m in diameter or less (PM<sub>2.5</sub>) contributed by ETS exposure, in micrograms per cubic meter ( $\mu$ g/m<sup>3</sup>), above a non-ETS background air pollution level, is given by the formula PM<sub>2.5</sub> ( $\mu$ g/m<sup>3</sup>) = 12.0 P (nmol/L).<sup>15,16</sup> Thus, children aged 4 through 6 years with a mean cotinine level of 6.25 nmol/L had a mean PM<sub>2.5</sub> exposure in excess of 70  $\mu$ g/m<sup>3</sup>, which would be expected to be associated with adverse health outcomes.<sup>17-19</sup>

Our primary findings are consistent with those reported in the literature. We found an increased risk for current asthma (OR, 5.3; 95% CI, 2.2-12.7) and wheez-

ing apart from colds (OR, 4.8; 95% CI, 2.4-9.9) among children aged 4 through 6 years. Although current and ever asthma were not significantly associated with high cotinine levels in the overall group (OR, 1.5; 95% CI, 0.8-2.7 and OR, 1.3; 95% CI, 0.8-2.2, respectively), these results are comparable with the recent pooled estimate of parental smoking on asthma, yielding an OR of 1.5 (95% CI, 1.3-1.7) and, for wheeze, yielding an OR of 1.5 (95% CI, 1.1-1.9).<sup>12</sup>

The findings from our analysis of lung function showed significant decrements related to ETS exposure for MMEF and FEV<sub>1</sub>/FVC ratio for children aged 8 through 16 years (Table 4). A recent review of lung function decrements related to ETS exposure showed a pooled estimated decrement of 0.9% for FEV<sub>1</sub> and 4.8% for MMEF,<sup>20</sup> which is very similar to the results obtained from our final models (Table 4). Children with prenatal maternal smoking had lower mean levels of lung function (Table 4), suggesting that in utero exposure to smoke may have long-term effects on lung growth.

Interpretation of these data is subject to several potential limitations. Cotinine, which has a half-life of 3 or 4 days, accurately measures recent exposure to ETS or occasional active smoking in older children, but not remote exposure. Current exposure is more likely to indicate "lifetime exposure" in younger children (because of their being alive fewer years, with less opportunity for movement of smokers into or out of the household), thus potentially explaining why an association with asthma and asthma symptoms is strongest in this group. The questionnaire data were not validated by a review of medical records or physician interview. This may be a particular problem with regard to prenatal smoking, which could have been underreported or misclassified. Although this was a large sample, the analysis may have lacked power to detect small increases in the OR for some of the outcomes. An additional limitation is that children may

change their behavior on the basis of symptoms. Children, particularly older ones, bothered by smoke may avoid it, resulting in lower cotinine levels. Furthermore, all children may not uniformly process inhaled ETS. For example, racial differences in cotinine levels, for similar levels of tobacco intake, has previously been described.<sup>21</sup>

Proxy reports (typically by the parent) were used for children. While this may adequately report symptoms in younger children (where they are more likely to be observed by the parents), the potential exists for over-reporting or under-reporting once the children reach school age.<sup>22</sup> This may contribute, in part, to our not detecting significant effects on asthma or symptoms among 7- through 16-year-old children.

## CONCLUSIONS

We found that children of all ages exposed to ETS had health effects potentially related to this exposure, and that recent exposure to ETS is important whether or not children had exposure to prenatal maternal smoking. The observed effects were, generally, stronger in the younger children, for whom the ETS exposure is greater and parental reporting is more accurate. Environmental tobacco smoke exposure is potentially preventable by restricting or eliminating smoking in the home and in public places that children visit.

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## REFERENCES

1. Health effects of exposure to environmental tobacco smoke: California Environmental Protection Agency. *Tob Control* 1997;6:346-353.
2. 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;54:357-366.
3. 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. Available at: <http://www.pediatrics.org/cgi/content/full/101/2/e8>. Accessibility verified October 26, 2000.
4. Strachan DP, Cook DG. Health effects of passive smoking, 6: parental smoking and childhood asthma: longitudinal and case-control studies. *Thorax* 1998;53:204-212.
5. Strachan DP, Cook DG. Health effects of passive smoking, 1: parental smoking and lower respiratory illness in infancy and early childhood. *Thorax* 1997;52:905-914.
6. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the US population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988 to 1991. *JAMA* 1996;275:1233-1240.
7. Benowitz NL. Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol Rev* 1996;18:158-204.
8. Plan and operation of the Third National Health and Nutrition Examination Survey, 1988-94, series 1: programs and collection procedures. *Vital Health Stat F* 1994;32:1-407.
9. Standardization of spirometry—1987 update: statement of the American Thoracic Society. *Am Rev Respir Dis* 1987;136:1285-1298.
10. SAS Language: Reference, Version 6. Cary, NC: SAS Institute Inc; 1990.
11. Shah BV, Barmwell BG, Bieler GS. *SUDAAN User's Manual, Release 7.5*. Research Triangle Park, NC: Research Triangle Institute; 1997.
12. Cook DG, Strachan DP. Health effects of passive smoking, 3: parental smoking and prevalence of respiratory symptoms and asthma in school age children. *Thorax* 1997;52:1081-1094.
13. Mannino DM, Siegel M, Husten C, Rose D, Etzel R. Environmental tobacco smoke exposure and health effects in children: results from the 1991 National Health Interview Survey. *Tob Control* 1996;5:13-18.
14. Klepels NE, Ott WR, Repace JL. The effect of cigar smoking on indoor levels of carbon monoxide and particles. *J Expo Anal Environ Epidemiol* 1999;9:622-635.
15. Repace JL, Lowrey AH. An enforceable indoor air quality standard for environmental tobacco smoke in the workplace. *Risk Anal* 1993;13:463-475.
16. Repace JL, Jinot J, Bayard S, Emmons K, Hammond SK. Air nicotine and saliva cotinine as indicators of workplace passive smoking exposure and risk. *Risk Anal* 1998;18:71-83.
17. Dockery DW, Pope CA. Acute respiratory effects of particulate air pollution. *Annu Rev Public Health* 1994;15:107-132.
18. Gold DR, Damokosh AI, Pope CA, et al. Particulate and ozone pollutant effects on the respiratory function of children in southwest Mexico City. *Epidemiology* 1999;10:8-16.
19. Pope CA, Dockery DW. Acute health effects of PM10 pollution on symptomatic and asymptomatic children. *Am Rev Respir Dis* 1992;145:1123-1128.
20. Cook DG, Strachan DP, Carey IM. Health effects of passive smoking, 9: parental smoking and spirometric indices in children. *Thorax* 1998;53:884-893.
21. Caraballo RS, Giovino GA, Pechacek TF, et al. Racial and ethnic differences in serum cotinine levels of cigarette smokers: Third National Health and Nutrition Examination Survey, 1988-1991. *JAMA* 1998;280:135-139.
22. Theunissen NC, Vogels TG, Koopman HM, et al. The proxy problem: child report versus parent report in health-related quality of life research. *Qual Life Res* 1998;7:387-397.