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

Peter J. Gergen, MD, MPH*; Jean A. Fowler, MS‡; Kurt R. Maurer, PhD§; William W. Davis, PhD‡; and Mary D. Overpeck, DrPH‖

ABSTRACT. *Objective.* **To measure the effect of environmental tobacco smoke (ETS) on respiratory health in a national sample of young children.**

Methods. **The study evaluated children 2 months through 5 years of age participating in the Third National Health and Nutrition Examination Survey, 1988 to 1994. The group was a representative sample of the US population (N = 7680). A parental report of household smoking or maternal smoking during pregnancy ascertained ETS exposure. Respiratory outcomes were based on parental report of wheezing, cough, upper respiratory infection, or pneumonia in the last 12 months and chronic bronchitis or physician-diagnosed asthma at any time. Logistic regression was used to adjust for age, sex, race/ethnicity, birth weight, day care, family history of allergy, breastfeeding, education level of head of household, and household size.**

Results. **Approximately 38% of children were presently exposed to ETS in the home, whereas 23.8% were exposed by maternal smoking during pregnancy. ETS exposure increased chronic bronchitis and three or more episodes of wheezing among children 2 months to 2 years old and asthma among children 2 months to 5 years old. For household exposure, a consistent effect was seen only at ≥20 cigarettes smoked per day. Adjusted odds ratios for increased risk (95% confidence interval) for household exposures (≥20 cigarettes smoked per day vs none smoked) and maternal prenatal exposure (prenatal smoking vs no smoking), respectively, for children 2 months to 2 years old were chronic bronchitis, 2.5 (1.6, 4.1); 2.2, (1.6, 3); three or more episodes of wheezing, 2.7 (1.7, 4.2), 2.1 (1.5, 2.9); and for children 2 months to 5 years old were asthma, 2.1 (1.4, 3.2); 1.8 (1.3, 2.6). Reported use within the past month of prescription medications for asthma (β-agonists or inhaled steroids) was not different between those with asthma reporting ETS exposure and those reporting no exposure; percent of patients with asthma reporting use of medication by household exposure was 0, 25.7%; 1 to 19 cigarettes smoked per day, 32.9%; and ≥20 cigarettes smoked per day, 23.1%; percent of patients with asthma reporting use of medication by maternal smoking during pregnancy was no, 28.9%; yes, 22.7%. Among children 2 months to 2 years of age exposed to ETS, 40% to 60% of the cases of asthma, chronic bronchitis, and three or more episodes of wheezing were attributable to ETS exposure. For diagnosed asthma among children 2 months through 5 years old, there were 133 800 to 161 600 excess cases. Among exposed children 2 months through 2 years of age, there were 61 000 to 79 200 excess cases of chronic bronchitis and 126 700 to 172 000 excess cases of three or more episodes of wheezing.**

Conclusions. **ETS exposure is common among children in the United States. The reported prevalence of asthma, wheezing, and chronic bronchitis was increased with ETS exposures. No statistically significant increase in the prevalence of upper respiratory infection, pneumonia, or cough was associated with ETS exposure. ETS exposure has little effect on the respiratory health of children between 3 and 5 years of age, with the exception of asthma. ETS appears to increase the prevalence of asthma rather than the severity as measured by medication use. These findings reinforce the need to reduce the exposure of young children to ETS.** *Pediatrics* **1998;101(2). URL: http://www.pediatrics.org/cgi/content/full/101/2/e8;** ***environmental tobacco smoke (ETS), asthma, wheeze, chronic bronchitis, children, attributable risk.***

ABBREVIATIONS. ETS, environmental tobacco smoke; NHANES III, the Third National Health and Nutrition Examination Survey; CHS, Child Health Supplement; NHIS, National Health Interview Survey; URI, upper respiratory infection.

Environmental tobacco smoke (ETS) is a highly prevalent respiratory irritant. A recent meta-analyses concluded that ETS plays an important role in the health of children.[1] However, in regard to respiratory health, the report noted there were gaps in the literature. Many of the samples studied were small and may not have been suitable to generalization. Few studies have data on the many factors that contribute to respiratory health in the first 5 years of life (eg, birth weight, socioeconomic status, family history of atopy, day care attendance, and so forth), thus making it impossible to identify the independent contribution of ETS. The effect of ETS appears to change depending on the age stud-

From the *Center for Primary Care, Agency for Health Care Policy and Research, Rockville, Maryland; ‡Klemm Analysis Group, Inc, Hyattsville, Maryland; §National Center for Health Care Statistics, Centers for Disease Control and Prevention, Hyattsville, Maryland; and ‖National Institute of Child Health and Human Development, National Institutes of Health, Bethesda, Maryland.

This article does not represent the policy of the Agency for Health Care Policy and Research (AHCPR). The views expressed are those of the authors, and no official endorsement by AHCPR is intended or should be inferred.

Received for publication May 1, 1997; accepted Oct 17, 1997.

Reprint requests to (P.J.G.) Agency for Health Care Policy and Research, CPCR, Room 502, 2101 E Jefferson St, Rockville, MD 20852.

ied. Much of the work in young children has focused on the first year of life, when a strong effect has been reported consistently.[2–4] The few studies that have attempted to evaluate the change in the effect of ETS over the first 5 or so years of life have found the effect of ETS is limited primarily to the first several years of life.[5,6]

The Third National Health and Nutrition Examinations Survey (NHANES III), a representative sample of the US population, offered a unique dataset in which information on ETS exposure, respiratory health, and cofactors was collected. The objectives of these analyses are to evaluate the effect of ETS exposure on the reported respiratory health of children 2 months through 5 years of age in the United States while taking into account the relevant cofactors and to calculate the excessive number of cases of the various respiratory conditions attributable to ETS exposure.

METHODS

Study Population and Sample Design

NHANES III was conducted from 1988 to 1994 by the National Center for Health Statistics of the Centers for Disease Control and Prevention, Atlanta, GA.[7] A stratified multistage clustered probability design was used to select a sample of the civilian noninstitutionalized US population ≥2 months of age. A total of 81 locations were selected for inclusion in the final sample. The protocol included a home interview followed by a physical examination in a mobile examination center. The study was approved by the National Center for Health Statistics' Institutional Review Board.

Demographic Characteristics

Age at the time of interview is reported as age in years at the last birthday, except for children <1 year of age, for whom age in months is reported. Self-reported race/ethnicity was categorized as non-Hispanic white, non-Hispanic black, Mexican-American, or other. The education level of the head of the household was the highest grade completed. Household size was based on the number of persons living in the house regardless of relationships.

Variable Definition

Parents were asked whether the child was ever breastfed or fed breast milk and the age when the child stopped breastfeeding completely. Children who were breastfed until at least 1 month of age were classified as ever breastfed. A family history of allergies was the self-report of asthma or hay fever at any age in either biologic parent. Day care was defined as attendance at a day care center or nursery school where there were six or more children for at least 10 hours per week for 1 or more months. Birth weight was derived from birth certificates (87.2%) and maternal report at the time of the household interview (12.8%), when the birth certificate was not available. Birth weight was used as a categoric variable in the analyses: <2500 g, ≥2500 g. Medication use was defined as the self-reported use of β-agonists or inhaled steroids within the month before the household interview. Asthma and chronic bronchitis required the self-report of a physician's diagnosis at any time in the past. The number of self-reported episodes of wheezing or whistling in the chest, cough, and upper respiratory infection (URI) during the past 12 months were categorized into two groups: none to two, and three or more. Self-reported pneumonia during the past 12 months was categorized as none, one or more, because of its infrequent occurrence.

ETS Exposure

Reported ETS exposure was classified in two different ways: current household exposure and maternal smoking at any time during the pregnancy. Current household exposure was defined as the total number of cigarettes smoked by household members in the house per day. Smoking by visitors or guests was not asked. Three categories were defined: none, 1 to 19 cigarettes smoked per day, and ≥20 cigarettes smoked per day. Previous analyses from NHANES III reported that household exposure was significantly associated with serum cotinine levels and accounted for most of the explained variance in the regression models among participants ≥4 years of age.[8]

It was not possible to identify the mother among the reported smokers in the household. However, information was collected on the biologic mother's smoking during pregnancy. Maternal smoking during pregnancy was used as a surrogate for current maternal smoking, because a high percent of women who smoking during pregnancy continue smoking through at least the first 5 years of the child's life.[9,10]

Statistical Methods

All estimates were calculated using the sampling weights to represent children 2 months through 5 years of age in the United States. The sampling weights were created to adjust for unequal probabilities of selection and adjusted to account for nonresponse and poststratified to the US population as estimated by the Bureau of the Census. Statistical analyses were performed using SUDAAN,[11] a program that adjusted for the complex sample design when calculating variance estimates. All confidence intervals shown are at the 95% level.

Modeling and Attributable Risk

The association between exposure and outcome was evaluated by analyzing stratified two-way contingency tables, testing the hypothesis of independence versus general association. Linear contrasts were used to evaluate differences in outcome proportions between groups exposed to ETS and those not exposed. Logistic regression models were then developed to account for other factors that might affect the association between ETS exposure and each respiratory outcome. Separate models were developed for the age groups 2 months through 2 years, 3 years through 5 years, and 2 months through 5 years, which included main effects other than exposure only when they or interactions between them were significant at $P < .05$.

Finally, the proportion of the prevalence of each outcome among exposed groups that can be attributed to ETS was calculated from the logistic model parameters.[12] The resulting proportions were multiplied by the estimates of the exposed population to obtain the number of excess cases attributable to ETS. Totals for observations with missing values were distributed among the categories in proportion to the distributions of outcome by exposure for the sample of observations with complete information.

Response Rate of the Interview Sample

Among white, black, and Mexican-American children 2 months through 5 years of age ($N = 8238$) selected for the NHANES III sample, 94.2% ($n = 7763$) participated in the interview. Of those participating in the interview, 98.9% ($n = 7680$) had complete information on ETS exposure. The nonresponse for all other variables used in the analyses within the group with complete data on ETS exposure was <2.1%.

RESULTS

Exposure to ETS varied by the measure evaluated. Based on current household exposure, 61.6% of the children lived in homes with no smoke exposure, whereas 23.9% lived in homes where 1 to 19 cigarettes were smoked per day and 14.5% in homes where ≥20 cigarettes were smoked per day. Smoking during pregnancy was reported by 23.8% of the biologic mothers. Mexican-American children were exposed to the lowest levels of ETS, whereas non-Hispanic white children were exposed to the highest levels. Little difference was seen by age or sex.

Among all children, diagnosed asthma, chronic bronchitis, and three or more episodes of wheezing increased with increased ETS exposure, regardless of which measure of exposure was used (Table 1). For household exposure, the effect was seen most con-

TABLE 1. Percent of Children 2 Months to 5 Years of Age With Respiratory Outcomes by ETS Exposure

	Household Exposure: Number of Cigarettes Smoked in the Home per Day			Smoking During Pregnancy	
	None	1–19[a]	≥20[b]	No	Yes[c]
Diagnosed asthma	5.1 (4743)	5.7 (2006)	9.2** (929)	5.1 (6114)	8.3** (1564)
Chronic bronchitis	3.0 (4743)	3.9 (2006)	5.6* (929)	3.2 (6114)	4.8* (1564)
In the last 12 months					
Three or more episodes of wheezing	7.5 (4734)	10.6** (2004)	12.6* (928)	7.8 (6105)	12.8** (1561)
Three or more episodes of cough	14.4 (4731)	14.7 (2002)	13.8 (928)	14.0 (6101)	15.6 (1560)
Three or more episodes of upper respiratory infection	40.1 (4742)	36.0 (2000)	33.3* (929)	38.4 (6111)	37.3 (1560)
One or more episodes of pneumonia	3.0 (4745)	3.8 (2006)	4.2 (929)	3.3 (6116)	3.7 (1564)

[a] Contrast, 1–19 vs zero. [b] Contrast, ≥20 vs zero. [c] Contrast, yes vs no.
0 = Sample size; * $P < .05$; ** $P < .01$.

sistently when ETS exposure was ≥20 cigarettes per day. Exposure to ≥20 cigarettes appeared to be protective for URI, whereas smoking during pregnancy had no effect. The negative association of ETS with URI disappeared after adjusting for breastfeeding, day care, and race (data not shown). Pneumonia and three or more episodes of cough showed no significant association to ETS and were excluded from additional analyses.

The effect of ETS varied by age (Table 2). When the sample was divided into two age groups (children 2 months to 2 years of age and children 3 years to 5 years of age), ETS was associated with diagnosed asthma, chronic bronchitis, and three or more episodes of wheezing in the younger age group. In the older age group, only the association between ETS and diagnosed asthma remained statistically significant.

Logistic regression analyses were performed to evaluate the role of potential confounders on the relationship observed between ETS and respiratory outcomes (Table 3). Models were created for the all-ages group of children, for children 2 months to 2 years of age, and for children 3 years to 5 years age groups separately, using both definitions of ETS exposure. When ETS was defined as household exposure, the strongest effect was seen consistently in the 2 months to 2 years age group among children living in households where ≥20 cigarettes were smoked per day, with the exception of diagnosed asthma, for which the adjusted odds ratios were similar in the younger and older age groups. Similar results were seen when ETS was defined as smoking during pregnancy.

Additional analyses investigated whether exposure to ETS increased the use of asthma medication among children with asthma. Reported use within the past month of prescription medications for asthma (β-agonists or inhaled steroids) was not different between those with asthma reporting ETS exposure and those reporting none; percent of those with asthma reporting use of medication by household exposure: none, 25.7%; 1 to 19 cigarettes smoked per day, 32.9%; and ≥20 cigarettes smoked per day, 23.1% ($P > .4$); percent of those with asthma reporting use of medication by maternal smoking during pregnancy: no, 28.9%; yes, 22.7% ($P > .30$).

Attributable Risk

The excess number of cases attributable to ETS exposure after adjustment for the relevant confounders was calculated. The findings were similar regardless of the measure of ETS. The attributable risks ranged from 0.4 to 0.6 among children exposed to ETS at age 2 months to 2 years (Fig 1); that is, among children exposed, 40% to 60% of the cases of asthma, chronic bronchitis, and three or more episodes of wheezing were attributable to ETS exposure. For diagnosed asthma among children 2 months through 5 years old, there were 133 800 to 161 600 excess cases, whereas among children 2 months through 2 years old, there were 61 000 to 79 200 excess cases of

TABLE 2. Percent of Children With Respiratory Outcomes by Age and ETS Exposure

	Household Exposure: Number of Cigarettes Smoked in the Home per Day			Smoking During Pregnancy	
	None	1–19[a]	≥20[b]	No	Yes[c]
Diagnosed asthma					
2 months to 2 years	4.0 (2733)	4.3 (1130)	7.1* (556)	3.9 (3486)	6.6* (933)
3–5 years	6.1 (2010)	7.2 (876)	11.1* (373)	6.3 (2628)	9.8* (631)
Chronic bronchitis					
2 months to 2 years	2.6 (2733)	3.8* (1130)	7.4** (556)	2.7 (3486)	6.3** (933)
3–5 years	3.4 (2010)	4.0 (876)	3.9 (373)	3.7 (2628)	3.4 (631)
≥3 Episodes of wheezing (past 12 months)					
2 months to 2 years	7.6 (2729)	12.2** (1129)	15.7** (555)	8.0 (3482)	15.8** (931)
3–5 years	7.4 (2005)	8.8 (875)	9.9 (373)	7.6 (2623)	9.9 (630)

[a] Contrast, 1–19 vs zero. [b] Contrast, ≥20 vs zero. [c] Contrast, yes vs no.
0 = Sample size; * $P < .05$; ** $P < .01$.

TABLE 3. Adjusted Odds Ratios[a] and 95% Confidence Interval for ETS Exposure, Ages 2 Months to 5 Years

	Household Exposure: Zero Is the Referent Group		
	Total	2 Months–2 Years	3–5 Years
Diagnosed asthma			
1–19	1.1 (0.8, 1.6)	0.9 (0.6, 1.5)	1.2 (0.7, 2.1)
≥20	2.1 (1.4, 3.2)	2.0 (1.1, 3.4)	2.1 (1.2, 3.5)
Chronic bronchitis			
1–19	1.2 (0.8, 1.7)	1.3 (0.8, 1.9)	1.2 (0.7, 2.1)
≥20	1.8 (1.1, 3.0)	2.5 (1.6, 4.1)	1.3 (0.6, 2.9)
In the last 12 months 3 or more episodes of wheezing			
1–19	1.4 (1.1, 1.9)	1.7 (1.2, 2.5)	1.2 (0.8, 1.8)
≥20	1.9 (1.2, 3.1)	2.7 (1.7, 4.2)	1.2 (0.6, 2.4)
	Smoking During Pregnancy: Not Exposed Is the Referent Group		
	Total	2 Months–2 Years	3–5 Years
Diagnosed asthma	1.8 (1.3, 2.6)	1.7 (1.1, 2.6)	1.7 (1.1, 2.8)
Chronic bronchitis	1.5 (1.1, 2.0)	2.2 (1.6, 3.0)	1.0 (0.6, 1.8)
In the last 12 months 3 or more episodes of wheezing	1.8 (1.4, 2.4)	2.1 (1.5, 2.9)	1.3 (0.8, 2.0)

[a] Adjusted for age, sex, race/ethnicity, birth weight, day care, family history of allergy, breastfeeding, education of head of household, household size.

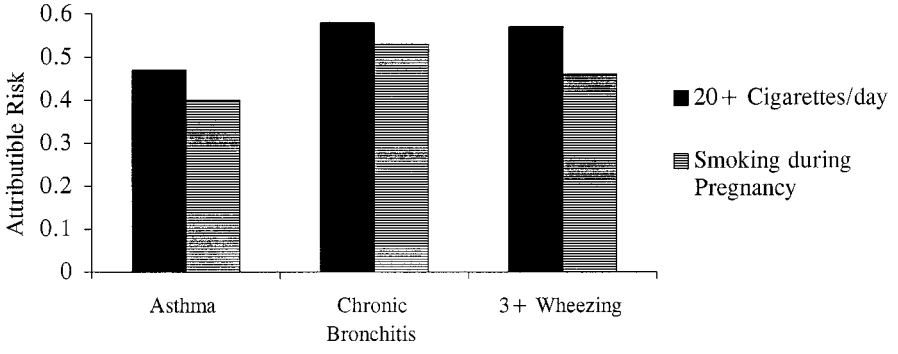

Fig 1. Attributable risk among children 2 months through 2 years of age exposed to ETS.

chronic bronchitis, and 126 700 to 172 000 excess cases of three or more episodes of wheezing.

DISCUSSION

ETS exposure in the home is widespread among US children <6 years of age (38%), and it plays an important, independent role in the respiratory health of children ≤2 years of age. The levels of exposure reported here are lower than those from the 1988 Child Health Supplement (CHS) of the National Health Interview Survey (NHIS), in which 42% of children ≤5 years of age were currently living in a household with a smoker.[13] Part of the difference may be attributable to the ~1.2% decrease in smoking levels between 1988 and 1992 among women >18 years of age in the United States.[14]

There is no ideal method to quantify a child's exposure to ETS. Comparison of self-report with cotinine measures have found that both give equivalent results when classifying children in regards to ETS exposure.[15–18] At the first well-child visit (6 to 8 weeks of age), urine cotinine levels ≥10 μg/L were found in 8% of nonsmoking homes, 44% in homes where someone other than the mother smoked, 91% where only the mother smoked vs 96% where the mother and another household member smoked.[19] The authors concluded that a screening questionnaire about household smoking was sufficient to define a group of infants with urine cotinines ≥10 μg/L. Previous analyses on Phase I (1988 to 1990) of NHANES III showed that among children 4 to 11 years of age, serum cotinine levels were higher in children with reported household exposure versus those with none reported.[8]

Reported smoking during pregnancy has been shown to be an excellent surrogate measure for current maternal smoking during at least the first 5 years of life. A telephone follow-up study performed on participants from the 1985 NHIS reported that 70% of women who quit smoking during any part of their pregnancy resumed smoking within 1 year of delivery, with >93% of those resuming in the first 6 months.[10] Data from the 1970 Child Health and Education Study in the United Kingdom, a birth cohort of all children born from April 5 to 11, 1970, found that >90% of the women who smoked during pregnancy were still smoking when their child reached 5 years of age.[9]

The number of smokers in the house is highly correlated with cotinine levels among children.[20,21] However, maternal smoking has been shown to be the chief source of ETS exposure for children in the home.[17,21] Maternal smoking behavior influences the smoking behavior of others. In a study of 433 infants at age 18 days, 75% of smoking mothers smoked near their infant and the amount smoked near the infant by the mother was correlated with the amount smoked by others near the infant.[22]

Day care does not seem to be a significant source of ETS exposure. Among English school children 5 to 7 years of age, only 8.7% of the children received child care from a smoker, and in 66% of the cases, the parent was also a smoker.[21] Among children with asthma 8 months through 13 years of age studied in an allergy clinic, reported ETS exposure in day care was not an important determinant of the child's cotinine level once household exposure was accounted for.[16] However in a nationwide study, day care has been shown to be an important factor contributing to the number of respiratory infections experienced by children 6 weeks through 17 months of age.[23]

In addition to the potential misclassification with respect to ETS exposure, a number of other issues may influence the findings of this study. Self-report was used to classify individuals in regards to their respiratory problems. Misunderstanding of the questions or use of unfamiliar terms may have resulted in nondifferential misclassification, which would tend to obscure any underlying association. Conversely, the effect of smoking on ETS may be attributable to an overreporting of symptoms by parents who smoke. Lebowitz and Burrows[24] reported that the association between ETS and smoking exposure was no longer significant after controlling for the respiratory symptoms of the parents. Other studies have reported that the association of ETS on respiratory symptoms is not eliminated when taking into account parental respiratory symptoms.[5,25] Finally, either the low prevalence of some of the respiratory effects or a small true effect of ETS on a particular respiratory outcome would reduced the power of this study to detect any true difference between the exposed and unexposed groups.

The NHANES III data add to the growing literature that with the exception of asthma, the respiratory effects of ETS are limited to the first few years of life among children ≤5 years of age. The reported incidence of pneumonia and bronchitis was influenced by ETS only during the first year of life in a birth cohort of >2200 children in England followed to age 5 years.[5] Maternal smoking was associated with an increased rate of medical consultations for bronchitis\pneumonia and maternal report of lower respiratory symptoms only in the first 2 years of life after adjustment for confounders in a birth cohort of ~1100 children followed annually from birth to 6 years of age in the Christchurch, New Zealand Child Development Study.[6] Although pneumonia was not statistically significantly related to ETS in the NHANES III, even in the 0 to 2 years age group, there appeared to be a tendency for pneumonia to increase with increasing ETS exposure.

Similar results are seen from analyses of studies focusing on active wheezing or wheezy bronchitis. In a case–control study of children 2 months to 16 years of age presenting to an emergency room with wheezing, ETS exposure (salivary cotinine ≥10 ng/mL) was found to be associated only with wheezing in children <2 years of age.[26] A case–control study of children 4 months to 4 years of age hospitalized for the first time for wheezy bronchitis reported that parental smoking, especially maternal, was an important factor in children <18 months of age.[18] Data from the 1987 National Medical Expenditure Survey showed that maternal smoking was significantly associated with asthma and wheezing combined only in the 0 to 2 years age group but not in the 3 to 18 years age group.[27]

Only diagnosed asthma is associated with ETS exposure during the first 5 years of life. Interestingly, wheezing, the major symptom of asthma, was not associated with ETS in the older group. Other studies have reported previously that ETS exposure approximately doubles the prevalence of asthma in children.[28,29] However, studies linking ETS to the incidence of asthma have found conflicting results. In Arizona, ETS exposure was associated only with the development of asthma if the mother had <12 years of education, but not among the children of more highly educated women.[30] In Canada, only children with atopic dermatitis exposed to ETS developed asthma, whereas others exposed to ETS did not develop asthma[31] and in New Zealand, ETS was not associated with the development of asthma.[32]

The role of ETS exposure on asthma severity is unclear. A previous report from the 1981 CHS of the NHIS reported that ETS was associated with the greater use of physician-prescribed asthma medication in the last 2 weeks,[29] whereas no association was seen in the NHANES III. The variation in defining medication use may explain the differences. In the 1981 CHS, a parent had to report the use of any medication prescribed by a physician for asthma. This form of the question does not eliminate the reporting of over-the-counter medications such as decongestants or cough suppressants. The NHANES III asked specifically about the name of the medication; the parent could either show the bottle or report the name verbally. In our analyses, we looked specifically at β-agonists and inhaled steroids, which are specific therapies for asthma. It is also possible that a sufficient number of sample persons whose asthma was aggravated by ETS avoided exposure to ETS and thus remove any association between ETS and severity in this cross-sectional study.

Studies using other measures of severity have reported conflicting results. Lower pulmonary functions and more reported acute exacerbations were found with ETS exposure among patients with asthma attending an allergy clinic.[16] Among low-income urban children with asthma in New York City (average age, 9 years), passive smoking was associated with emergency room visits for asthma, but not with hospitalizations or abnormalities in pulmonary function.[33] The effects of ETS on severity of asthma varied with age among patients evaluated at a Canadian allergy clinic. In the youngest group (1 to 6 years of age), no significant effect was found with exposure to smoke; in the middle age group (7 to 11 years of age), the children of smokers had an increase in their asthma score; and in the oldest group (12 to 17 years of age), the asthma symptom score was increased and the pulmonary functions were decreased in the children exposed to smoke.[34] In a recent report on 22 children (mean age, 5 years) followed for 1 month after being hospitalized for

asthma, children living with a smoker reported 1.9 fewer days of daytime symptoms than those not living with a smoker ($P < .05$); however, no statistically significant difference was found in the mean number of nighttime symptoms.[35] Other studies reporting changes in ETS exposure did not find an association with acute attacks of asthma.[36,37]

A previous study used a national dataset to estimate the number of excess cases of disease from ETS. Approximately 38 000 excess cases of asthma and wheezing in children <18 years of age were reported from the 1987 National Medical Expenditure Survey.[27] This probably is an overestimate because ETS was statistically significantly associated with ETS exposure only in children 0 to 2 years of age. However, the nonsignificant association in the older age group was included in their calculation.[27] Our findings indicate ~13 400 to 16 200 excess cases of asthma among children 2 months through 5 years old. Among children 2 months through 2 years old, there were ~61 000 to 79 000 excess cases of chronic bronchitis and 127 000 to 172 000 cases of three or more episodes of wheezing.

SUMMARY

ETS exposure is common among children in the United States. The reported prevalence of asthma, wheezing, and chronic bronchitis was increased with ETS exposures. No statistically significant increase in the prevalence of URI, pneumonia, or cough was associated with ETS exposure. The exposure has little effect on the respiratory health of children 3 years through 5 years of age with the exception of asthma. ETS appears to increase the prevalence of asthma rather than the severity, as measured by medication use. These findings reinforce the need to reduce the exposure of young children to ETS.

REFERENCES

1. DiFranza JR, Lew RA. Morbidity and mortality in children associated with the use of tobacco products by other people. *Pediatrics.* 1996;97:560–568
2. Harlap S, Davies AM. Infant admissions to hospital and maternal smoking. *Lancet.* 1974;1:529–532
3. Leeder SR, Corkhill R, Irwig LM, Holland WW. Influence of family factors on the incidence of lower respiratory illness during the first year of life. *Br J Prev Soc Med* 1976;30:203–212
4. Pedreira FA, Guandolo VL, Feroli EJ, Mella GW, Weiss IP. Involuntary smoking and incidence of respiratory illness during the first year of life. *Pediatrics.* 1985;75:594–597
5. Colley JRT, Holland WW, Corkhill RT. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. *Lancet.* 1974;2:1031–1034
6. Fergusson DM, Hons BA, Horwood LJ. Parental smoking and respiratory illness during early childhood: a six-year longitudinal study. *Pediatr Pulmonol.* 1985;1:99–106
7. National Center for Health Statistics. Plan and operation of the Third National Health and Nutrition Examination Survey, 1988–1994. *Vital Health Stat.* 1994;1:32
8. 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, 1988–1991. *JAMA.* 1996;275:1233–1240
9. Taylor B, Wadsworth J. Maternal smoking during pregnancy and lower respiratory tract illness in the early life. *Arch Dis Child.* 1987;62:786–791
10. Fingerhut LA, Kleinman JC, Kendrick JS. Smoking before, during, and after pregnancy. *Am J Public Health.* 1990;80:541–544
11. Shah BV, Barnwell BG, Bieler GS. *SUDAAN User's Manual*. Release 7.0. Research Triangle Park, NC: Research Triangle Institute; 1996
12. Kleinbaum DG, Kupper LL, Morgenstern H. *Epidemiologic Research Principles and Quantitative Methods*. Belmont, CA: Lifetime Learning Publications; 1982:160
13. Overpeck MD, Moss AJ. *Children's Exposure to Environmental Cigarette Smoke Before and After Birth: Health of Our Nation's Children, United States, 1988*. Hyattsville, MD: National Center for Health Statistics; 1991. Advance data from Vital and Health Statistics publication 202
14. National Center for Health Statistics. *Health, United States, 1994*. Hyattsville, MD: US Public Health Service; 1995
15. Greenberg RA, Haley NJ, Etzel RA, Loda FA. Measuring the exposure of infants to tobacco smoke. Nicotine and cotinine in urine and saliva. *N Engl J Med.* 1984;310:1075–1078
16. Chilmonczyk BA, Salmun LM, Megathlin KN, et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. *N Engl J Med.* 1993;328:1665–1669
17. Bakoula CG, Kafritsa YJ, Kavadias GD, et al. Objective passive-smoking indicators and respiratory morbidity in young children. *Lancet.* 1995;346:280–281
18. Rylander E, Pershagen G, Eriksson M, Bermann G. Parental smoking, urinary cotinine, and wheezing bronchitis in children. *Epidemiology.* 1995;6:289–293
19. Chilmonczyk BA, Knight GJ, Palomaki GE, Pulkkinen AJ, Williams J, Haddow JE. Environmental tobacco smoke exposure during infancy. *Am J Public Health.* 1990;80:1205–1208
20. Jarvis MJ, Strachan DP, Feyerabend C. Determinants of passive smoking in children in Edinburgh, Scotland. *Am J Public Health.* 1992;82:1225–1229
21. Cook DG, Whincup PH, Jarvis MJ, Strachan DP, Papacosta O, Bryant A. Passive exposure to tobacco smoke in children aged 5–7 years: individual, family, and community factors. *Br Med J.* 1994;308:384–389
22. Greenberg RA, Bauman KE, Glover LH, et al. Ecology of passive smoking by young infants. *J Pediatr.* 1989;114:774–780
23. Hurwitz ES, Gunn WJ, Pinsky PF, Schonberger LB. Risk of respiratory illness associated with day-care attendance: a nationwide study. *Pediatrics.* 1991;87:62–69
24. Lebowitz MD, Burrows B. Respiratory symptoms related to smoking habits of family adults. *Chest.* 1976;69:48–50
25. Weiss ST, Tager IB, Speizer FE, Rosner B. Persistent wheeze. Its relation to respiratory illness, cigarette smoking, and level of pulmonary function in a population sample of children. *Am Rev Respir Dis.* 1980;122:697–707
26. Duff AL, Pomeranz ES, Gelber LE, et al. Risk factors for acute wheezing in infants and children: viruses, passive smoke, and IgE antibodies to inhalant allergens. *Pediatrics.* 1993;92:535–540
27. Stoddard JJ, Miller T. Impact of parental smoking on the prevalence of wheezing respiratory illness in children. *Am J Epidemiol.* 1995;141:96–102
28. Gortmaker SL, Walker DK, Jacobs FH, Ruch-Ross H. Parental smoking and the risk of childhood asthma. *Am J Public Health.* 1982;72:574–579
29. Weitzman M, Gortmaker S, Walker DK, Sobol MA. Maternal smoking and childhood asthma. *Pediatrics.* 1990;85:505–511
30. Martinez FD, Cline M, Burrows B. Increased incidence of asthma in children of smoking mothers. *Pediatrics.* 1992;89:21–26
31. Murray AB, Morrison BJ. It is children with atopic dermatitis who develop asthma more frequently if the mother smokes. *J Allergy Clin Immunol.* 1990;86:732–739
32. Horwood LJ, Fergusson DM, Hons BA, Shannon FT. Social and familial factors in the development of early childhood asthma. *Pediatrics.* 1985;75:859–868
33. Evans D, Levison MJ, Feldman CH, Clark NM, Wasilewski Y, Levin B, Mellins RB. The impact of passive smoking on emergency room visits of urban children with asthma. *Am Rev Respir Dis.* 1987;135:567–572
34. Murray AB, Morrison BJ. Passive smoking by asthmatics: its greater effect on boys than on girls and on older than on younger children. *Pediatrics.* 1989;84:451–459
35. Abulhosn RS, Morray BH, Llewellyn CE, Redding GJ. Passive smoke exposure impairs recovery after hospitalization for acute asthma. *Arch Pediatr Adolesc Med.* 1997;151:135–139
36. Ehrlich R, Kattan M, Godbold J, et al. Childhood asthma and passive smoking. Urinary cotinine as a biomarker of exposure. *Am Rev Respir Dis.* 1992;145:594–599
37. Ogborn CJ, Duggan AK, DeAngelis C. Urinary cotinine as a measure of passive smoke exposure in asthmatic children. *Clin Pediatr.* 1994;33:220–226

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

Peter J. Gergen, Jean A. Fowler, Kurt R. Maurer, William W. Davis and Mary D. Overpeck

Pediatrics 1998;101;e8
DOI: 10.1542/peds.101.2.e8

PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

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

Peter J. Gergen, Jean A. Fowler, Kurt R. Maurer, William W. Davis and Mary D. Overpeck

Pediatrics 1998;101;e8
DOI: 10.1542/peds.101.2.e8