Impact of Environmental Tobacco Smoke on Children With Asthma, United States, 2003–2010

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ABSTRACT

OBJECTIVE: Given widespread interventions to reduce environmental tobacco smoke (ETS) exposure and improve asthma control, we sought to assess the current impact of ETS exposure on children with asthma.

METHODS: We analyzed 2003–2010 data for nonsmoking children aged 6 to 19 years with asthma from the National Health and Nutrition Examination Survey. Outcomes (sleep disturbance, missed school days, health care visits, activity limitation, and wheezing with exercise) were compared between ETS exposed children (serum cotinine levels 0.05 to 10 ng/mL) and unexposed children (<0.05 ng/mL) using ordinal regression adjusted for demographic characteristics. We also assessed whether associations were observable with low ETS exposure levels (0.05 to 1.0 ng/mL).

RESULTS: Overall, 53.3% of children aged 6 to 19 years with asthma were ETS exposed. Age-stratified models showed associations between ETS exposure and most adverse outcomes among 6- to 11-year-olds, but not 12- to 19-year-olds. Even ETS exposure associated with low serum cotinine levels were associated with adverse outcomes for 6- to 11-year-olds. Race-stratified models for children aged 6 to 19 years showed an association between ETS exposure and missing school, health care visits, and activity limitation due to wheezing among non-Hispanic white children, and disturbed sleep among non-Hispanic white and Mexican children. Among non-Hispanic black children, there was no elevated risk between ETS exposure and the assessed outcomes: non-Hispanic black children had high rates of adverse outcomes regardless of ETS exposure.

CONCLUSIONS: Among children with asthma 6 to 11 years of age, ETS exposure was associated with most adverse outcomes. Even ETS exposure resulting in low serum cotinine levels was associated with risks for young children with asthma.

KEYWORDS: asthma; environmental tobacco smoke

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WHAT'S NEW

National data show that despite population-based tobacco smoke exposure reduction efforts, long-standing recommendations for children with asthma to avoid environmental tobacco smoke, and available effective treatment to control symptoms, exposure remains common and is associated with adverse outcomes among young children with asthma.

ENVIRONMENTAL TOBACCO SMOKE (ETS) was highlighted as a health risk by the 1972 Surgeon General's report[1] and was the focus of a subsequent 1986 report, *The Health Consequences of Involuntary Smoking*.[2] ETS exposure (also called involuntary smoking, or passive or secondhand smoke exposure) is the combination of "sidestream" smoke given off by a burning tobacco product and "mainstream" smoke exhaled by the smoker.[1–3] Two decades later, with the publication of the 2006 Surgeon General's report on the same topic,[3] smoking rates among adults had declined from 30% to 20%,[4] and there were comprehensive smoke-free laws prohibiting smoking in public places in 25 states and the District of Columbia.[5] Yet ETS exposure declined more slowly among children compared to adults[6,7] and remained common: during the period 1999–2004, 60.5% of children ages 4 to 11 years and 55.4% of those aged 12 to 19 years of age were exposed to ETS.[7]

For children with asthma, ETS exposure poses specific risks such as potentiated impact of other airway irritants, increased asthma exacerbations, and greater asthma severity.[8–10] Despite long-standing recommendations in the National Asthma Education and Prevention Program Asthma (NAEPP) guidelines[11] to identify and avoid tobacco smoke exposure, in 2005–2010, 53% of children 4 to 19 years of age with asthma had evidence of ETS exposure.[12] Furthermore, while ETS exposure among children without asthma has continued to decline, progress among children with asthma recently stalled so that in 2007–2010, ETS exposure was significantly higher among children with asthma.[13]

Studies using national data from the early 1990s concluded that exposure to ETS is associated with increased symptoms and decreased lung function among children

with asthma.[9] From that time, there have been major advances to control asthma symptoms. During the 1990s, the NAEPP guidelines were introduced, which recommended multifaceted, ongoing management as the standard of care, and new medications became available (including more potent inhaled corticosteroids such as budesonide and fluticasone).[14] Our objective was to use recent nationally representative data to assess the current impact of ETS exposure for children with asthma to determine whether ETS exposure continues to pose a significant burden among this population. We also sought to examine whether subpopulations of children with asthma are similarly affected by ETS exposure given observed racial disparities in adverse asthma outcomes.[15] We analyzed 2003–2010 National Health and Nutrition Examination Survey (NHANES) data for children 6 to 19 years of age with asthma.

METHODS

The National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC) conducts NHANES on a continuous schedule. A complex, stratified, multistage probability cluster design is used to obtain a nationally representative sample of the civilian noninstitutionalized population; data are released every 2 years.[16] NHANES respondents participate in in-person interviews at home, and health examinations and laboratory testing in mobile exam centers. Informed consent was obtained for persons 18 years and older. Parental consent was obtained for participants aged 17 years and under, and child assent was obtained for children aged 7 to 17 years. The NCHS institutional review board approved the NHANES survey protocols. In 2003–2006, non-Hispanic black and Mexican American persons, low-income persons, and children ages 12 to 19 years were oversampled. Beginning in 2007, the entire Hispanic population was oversampled. During the in-home interview, children 16 years of age and older provided survey responses, and a responsible adult responded in proxy for younger children.

We compiled 2003–2010 data to obtain an adequate sample of children aged 6 to 19 years with current asthma who were not active smokers. Younger children were not included because children under 6 years had a high rate of missing data for serum cotinine, and data on missed school days due to wheezing were not collected for this age group. Unweighted examination response rates for children over the period 2003–2010 ranged from 81% to 89%.[17] Current asthma status was assessed by affirmative responses to both of 2 questions: "Has a doctor or health professional ever told you that you have asthma?" and "Do you still have asthma?" Overall, 1,286 of 11,866 child respondents aged 6 to 19 years had current asthma.

We included covariates previously associated with increased risk of having asthma or adverse asthma outcomes, and/or with risk for ETS exposure. Age was categorized as 6 to 11 years and 12 to 19 years. Race/ethnicity was categorized as non-Hispanic white, non-Hispanic black, Mexican American, and other. Poverty status was categorized according to the poverty–income ratio, a ratio of family income to the federal poverty threshold adjusted for family size. Low income status was defined as poverty–income ratio of <1.85, which is the level of eligibility for federal programs such as reduced price school lunches. Records with unknown income ($n = 68$) were excluded, leaving 1218. Home ownership was dichotomized as family ownership of the home versus renting or other arrangement. This covariate was included as a proxy for residence in an apartment, where ETS exposure may occur from smoking in adjacent units versus a detached home, in which smoke exposure is more easily controlled.[18] Family structure has been associated with risk of having asthma and with adverse outcomes.[19,20] Although specific information about family structure was not available, household size has been used in previous analyses as a proxy.[9] Household size was dichotomized as 4 or fewer and 5 or greater persons. Preventive asthma medication (PAM) use was categorized as "yes" if the child was reported to have received any prescription medicines in the past 30 days, and at least 1 of these medications was categorized as a PAM (either as identified by the interviewer if the container was available or reported by the respondent if the container was not available). PAMs included long-term controller medications listed in the NAEPP guidelines[11]: inhaled corticosteroids, leukotriene receptor antagonists, long-acting β-agonists, mast-cell stabilizers, and methylxanthines. Combination medications were included if one component was classified as a PAM.

Serum cotinine levels were measured by the CDC's National Center for Environmental Health.[21] The estimated half-life of serum cotinine is approximately 16 to 19 hours and indicates exposure over the previous 1 to 2 days.[22] Records with missing serum cotinine values ($n = 183$, 15.0% of the unweighted sample) were excluded, leaving 1035 records. Smoking status was ascertained using 2 measures. First, children 12 to 19 years old were asked about use of products containing nicotine in the previous 5 days. Those reporting nicotine product use were excluded ($n = 80$), leaving a sample of 955. Second, children with serum cotinine values consistent with tobacco product use (>10.0 ng/mL, $n = 26$) were excluded, leaving 929 records.[22] Among nonsmoking children, those with serum cotinine levels 0.05 to 10.0 ng/mL were classified as ETS exposed, and those with levels <0.05 ng/mL were classified as unexposed.[7,9,22]

The relatively high rate of missing data for serum cotinine raises the possibility of nonresponse bias. Younger children in the sample were missing laboratory data more frequently than older children. Age-specific analyses were conducted to compare children with and without missing laboratory data. As described elsewhere,[23] there were few differences in sociodemographic factors, dietary factors, and body measures with the exception of a lower amount of missing laboratory data among Mexican Americans. An adjustment of the original examination sample weights[24] was performed to examine the potential impact

of nonresponse bias. There were only small differences in point estimates and standard errors; therefore, nonresponse to the laboratory component likely did not introduce significant bias.

Outcomes were defined using responses to a set of questions about wheezing asked during the in-home interview, including missed days of school or work due to wheezing during the past 12 months (none, 1 to 7 days, and 8 or more days), number of health care visits to a doctor's office or hospital emergency room in the past 12 months due to attacks of wheezing (none, 1 to 2 visits, and 3 or more visits), disturbed sleep due to wheezing in the past 12 months (none, <1 night per week, or ≥ 1 nights per week), limitation of usual activities due to wheezing in the past 12 months (none, a little, or fair/moderate/a lot of activity limitation), and wheezing with exercise or physical activity in the past 12 months (no or yes). Children missing responses for any of these outcomes were excluded ($n = 4$), leaving 925 records for the full analysis.

Statistical analysis was conducted using SAS-callable SUDAAN (SAS version 9.3, SAS Institute, Cary, NC; SUDAAN, version 10.0, RTI, Research Triangle Park, NC) to account for the complex survey design. NHANES examination weights were used to produce national estimates accounting for differential probability of selection, noncoverage, and nonresponse. Family-wide chi-square analysis was used to compare outcomes between ETS-exposed and unexposed children for each characteristic, and the Wald test was used to compare individual categories. To assess adjusted associations, ordinal regression models were used to compare the risk of ETS exposure versus no exposure for each of the ordered levels of each outcome variable. For all models, we report the conditional marginal proportions (adjusted percentage) and adjusted risk ratios (ARR).[25]

We adjusted for gender, age group, race/ethnicity, poverty status, home ownership, and household size. We tested for interactions between ETS exposure status and gender, age group, and race/ethnicity given that asthma severity, exposure and sensitivity to airway irritants, and mechanisms of ETS exposure may vary between categories of these characteristics. Because significant interactions with exposure status were identified for some outcomes for age and race/ethnicity, stratified models are presented for all 5 outcomes. However, the sample size was insufficient to stratify simultaneously by age and race/ethnicity. Therefore, the models were first stratified by age with race/ethnicity included as a covariate, then were stratified by race/ethnicity with age included as a covariate.

To determine whether the associations between ETS exposure and each of the 5 outcomes was still observable even with cotinine levels consistent with low levels of ETS exposure, we used additional models that assessed associations for children with a low level of serum cotinine (0.05 ng/mL to <1.0 ng/mL) versus no exposure. The upper bound of serum cotinine levels consistent with low exposure was chosen at 1.0 ng/mL on the basis of past studies.[6,22] For these sets of models, the interaction between exposure and age was significant for some outcomes, but the interaction between exposure and race/ethnicity and income was not significant for any outcomes. Therefore, only age-stratified models are presented.

We introduced PAM use as a covariate in all models as a sensitivity analysis to attempt to control for differences in asthma severity. The NAEPP guidelines state that for population-based evaluations, "asthma severity can be inferred after optimal therapy is established by correlating levels of severity with the lowest levels of treatment required to maintain control."[11] Using PAM within the past month is a rough indicator of having asthma of at least persistent severity. However, it could also be argued that PAM use could be considered an outcome (an adverse outcome reflecting severity) or that it lies along causal pathway (ETS exposure increases symptoms and necessitates PAM, which in turn modulates observed outcomes). Therefore, these sensitivity models were not presented as main models but explored as a way to control for underlying asthma severity.

RESULTS

The characteristics of the analytic sample, weighted to estimate national percentages, are shown in Table 1. Exposure to ETS differed significantly by age, race/ethnicity, poverty status, and home ownership status. Differences between ETS-exposed and unexposed children for each of 5 outcomes related to wheezing in the past 12 months were assessed (Table 2). Although the observed percent with adverse outcomes was higher among ETS-exposed children for all outcomes, only the relationship with health care visits due to wheezing reached statistical significance.

In ordinal regression models, interactions between ETS exposure status and age and race/ethnicity were present. Because sample size was insufficient to stratify by both variables simultaneously, one set of models was stratified by age and another set by race/ethnicity. Conditional marginal proportions (adjusted percentages) and adjusted relative risks (ARR) of ETS exposure compared to no exposure for age-stratified models are presented in Table 3. The crude relative risks are similar to the ARR; therefore, only the ARR are presented. The risk of adverse outcomes was apparent among ETS-exposed younger children (greater percentages missed more school and had sleep disturbances, activity limitations, and wheezing with exercise) compared to unexposed 6- to 11-year-olds, but for children ages 12 to 19 years with ETS exposure, there was no observed increased risk for any of the adverse outcomes assessed.

When the model including the entire sample age range from 6 to 19 years was stratified by race/ethnicity (Table 4), only non-Hispanic white and Mexican American children had increased risk of adverse outcomes with ETS exposure. Non-Hispanic white children with asthma with ETS exposure had increased risk of missing more school days, having more health care visits, having disturbed sleep, and having an activity limitation due to wheezing compared to those with no exposure. Mexican American

Table 1. Sample Characteristics and ETS Exposure Status for Children Aged 6 to 19 Years With Asthma, United States, 2003–2010*

Characteristic	Unweighted Number	Weighted %	ETS Exposed, % (SE)†	*P* Value‡
Total no. of subjects	925	100	53.3 (3.2)	
Age group				
6–11 y	364	40.5	58.6 (4.2)	.05
12–19 y	561	59.5	49.7 (3.7)	
Sex				
Male	506	53.4	51.6 (3.8)	.45
Female	419	46.6	55.2 (4.2)	
Race/ethnicity				
Non-Hispanic white	235	55.9	52.2 (5.2)	<.001
Non-Hispanic black	354	20.6	67.9 (3.3)	
Mexican American	202	10.2	37.5 (3.3)	
Other	134	13.3	41.1 (6.4)	
Poverty status				
<1.85 PIR	508	42.3	72.0 (2.6)	<.001
≥1.85 PIR	417	57.7	39.5 (4.0)	
Home ownership				
Owns home	529	68.4	43.6 (4.3)	<.001
Rents/other	396	31.6	74.2 (3.2)	
Household size				
4 or fewer	490	58.3	53.8 (3.6)	.77
5 or more	435	41.7	52.6 (4.1)	
Preventive asthma medication				
Yes, in past month	248	31.0	48.5 (5.8)	.18
No	677	69.0	55.4 (2.9)	

ETS = environmental tobacco smoke; SE = standard error; PIR = poverty–income ratio.

*Excludes smokers (children with serum cotinine level >10 ng/mL or 12- to 19-year-olds who reported use of tobacco products in the previous 5 days). Current asthma is defined as reporting ever receiving a diagnosis of asthma from a health professional and having asthma at the time of the health survey.

†Defined as serum cotinine level 0.5 to 10.0 ng/mL.

‡*P* value for family-wide chi-square test.

children with asthma had increased risk of sleep disturbance due to wheezing with ETS exposure. Although non-Hispanic black children did not have increased ARR of adverse outcomes with ETS exposure, the percentages with adverse outcomes was generally higher for unexposed children compared with children of other race/ethnic groups. For example, among children unexposed to ETS, 7% of non-Hispanic white children, 13% of Mexican children, and 21% of non-Hispanic black children had 1+ nights/week of disturbed sleep due to wheezing after adjusting for other covariates.

In an additional set of models, the exposure variable was limited to low serum cotinine levels (0.05 to <1.0 ng/mL) and compared no exposure to assess if associations with

Table 2. Frequency of Outcomes Due to Wheezing Among Children With Asthma, 6 to 19 Years of Age, United States, 2003–2010

Characteristic	ETS Exposed, % (SE)* (n = 520)	Unexposed, % (SE) (n = 405)	*P* Value †
Missed school days past 12 mo			
0 d	67.5 (2.9)	71.3 (3.0)	.14
1–7 d	22.7 (2.8)	23.9 (2.9)	
8+ d	9.8 (2.1)	4.9 (1.0)	
Health care visits past 12 mo			
0	60.0 (3.3)	65.6 (3.4)	.02
1–2 visits	25.2 (2.4)	26.9 (2.9)	
3+ visits	14.7 (2.1)	7.5 (1.9)	
No. of nights disturbed sleep past 12 mo			
0	60.2 (3.1)	65.3 (3.6)	.11
<1 night per week	21.4 (2.7)	23.0 (2.7)	
1+ nights per week	18.5 (2.6)	11.6 (2.4)	
Activity limitation in past 12 mo			
None	64.7 (3.2)	68.9 (2.9)	.12
A little	20.3 (2.9)	21.5 (2.5)	
Fair/moderate amount/a lot	15.1 (2.1)	9.6 (1.8)	
Wheezing during exercise past 12 mo			
No	52.4 (2.7)	58.3 (3.4)	.10
Yes	47.6 (2.7)	41.7 (3.4)	

ETS = environmental tobacco smoke; SE = standard error.

*Defined as serum cotinine level 0.5 to 10.0 ng/mL.

†*P* value for family-wide chi-square test.

Table 3. Adjusted Percentage and Risk Ratio (ARR) of ETS Exposed Versus Unexposed Children With Asthma for Each of 5 Outcomes Due to Wheezing, Stratified by Age Group, United States, 2003–2010*

Characteristic	6–11 Years (n = 364)			12–19 Years (n = 561)		
	ETS Exposed (adj %)	Unexposed (adj %)	ARR (95% CI)	ETS Exposed (adj %)	Unexposed (adj %)	ARR (95% CI)
Missed school days in past 12 mo						
0 d	47.9	65.2	0.7 (0.6, 1.0)†	80.0	78.9	1.0 (0.9, 1.2)
1–7 d	37.3	27.0	1.4 (1.0, 1.9)†	16.1	17.0	1.0 (0.6, 1.5)
8+ d	14.7	7.8	1.9 (1.1, 3.3)†	3.9	4.1	0.9 (0.5, 1.7)
Health care visits in the past 12 mo						
0	47.0	57.9	0.8 (0.6, 1.1)	67.2	73.2	0.9 (0.8, 1.1)
1–2 visits	33.5	28.6	1.2 (0.9, 1.5)	24.4	20.4	1.2 (0.9, 1.6)
3+ visits	19.6	13.6	1.4 (0.8, 2.5)	8.4	6.4	1.3 (0.8, 2.1)
Nights w/disturbed sleep past 12 mo						
0	45.2	69.0	0.7 (0.5, 0.8)†	68.3	68.1	1.0 (0.9, 1.2)
<1 night per week	28.7	19.5	1.5 (1.1, 1.9)†	20.7	20.8	1.0 (0.8, 1.3)
1+ nights per week	26.1	11.6	2.3 (1.4, 3.6)†	11.0	11.1	1.0 (0.7, 1.5)
Activity limitation past 12 mo						
None	54.5	71.9	0.8 (0.6, 1.0)†	69.1	72.2	1.0 (0.8, 1.1)
A little	27.9	19.0	1.5 (1.0, 2.1)†	19.5	17.8	1.1 (0.8, 1.5)
Fair/moderate amount/a lot	17.6	9.1	1.9 (1.1, 3.4)†	11.4	10.0	1.1 (0.7, 1.8)
Wheezing during exercise past 12 mo						
No	43.5	70.2	0.6 (0.5, 0.8)†	57.7	53.4	1.1 (0.9, 1.3)
Yes	56.5	29.8	1.9 (1.3, 2.9)†	42.3	46.6	0.9 (0.7, 1.2)

ARR = adjusted risk ratio; ETS = environmental tobacco smoke; CI = confidence interval; adj = adjusted; SE = standard error.

*Adjusted for sex, race/ethnicity and poverty status, household ownership, and household size. Conditional marginal proportions (adjusted percentages) and ARRs were estimated by ordinal regression models for the ordered levels of the outcome variables.

†The 95% CI excluded 1.0 before rounding.

adverse outcomes were still observable even for this lower range of ETS exposure. Because this analysis showed significant risk of ETS exposure only for children aged 6 to 11 years of age, results for 12- to 19-year-olds are not shown in Table 5. The associations observed for low levels of serum cotinine and adverse outcomes were similar to those seen for the entire range of ETS exposure (Table 3): the ARRs were significantly elevated for missed school days, disturbed sleep, and activity limitation due to wheezing, and to wheezing with exercise.

Introducing PAM use into the models presented in Tables 3, and 5, 4 did not change the pattern of results, and an interaction term for PAM use and ETS exposure was not significant. The risk of the most adverse outcome category generally increased, but the overall interpretation the same as those observed in models that did not include PAM use (data not shown).

DISCUSSION

Although it has been well established that ETS exposure is harmful to children with asthma, this analysis of nationally representative data shows that despite long-term, widespread efforts to reduce exposure and promote effective treatment, ETS exposure remains associated with increased risk of adverse outcomes among children with asthma, primarily among younger children aged 6 to 11 years and non-Hispanic white children aged 6 to 19 years. This increased risk is particularly important to recognize now that recent data demonstrate that children with asthma have higher rates of ETS exposure compared with children without asthma.[13] When we examined associations between outcomes and a lower level of exposure among 6- to 11-year-old children, the associations remained significant. These results suggest that even low levels of ETS exposure pose risks for young children with asthma and that it remains important to support recommendations to eliminate exposures,[3,5,11,26] including direct (eg, in-home smoking by household members) and indirect (eg, smoking in adjacent units in multifamily housing).

Young children, more so than adolescents, spend much of their time indoors and encounter the majority of tobacco smoke exposure in residential locations.[27] Although the proportion of adults who smoke and children exposed to ETS continues to decline,[4,28] the proportion of ETS-exposed children is still substantial at about 50%.[13] In addition to the well-documented risks of ETS exposure for younger children, the results of this analysis suggest that the association between ETS exposure and adverse asthma outcomes exists mainly among younger children with asthma. These different findings by age group may be associated with smaller airways in younger children (ie, greater sensitivity to small amounts of inflammation), or they could be due to differences in the chronicity of exposure between older and younger children. That is, given the relatively brief half-life of serum cotinine, it is possible that on a population level, similar cotinine levels reflect different patterns of exposure. These patterns may differ systematically by age. Levels in younger children may represent more chronic exposure due to proximity to smoking household members or other residential exposures,[18] whereas for older children, similar serum levels may indicate periodic exposure due to smoking peers or in nonresidential environments.

Table 4. Adjusted Percentage and Risk Ratio (ARR) of ETS-exposed Versus Unexposed Children With Asthma for Each of 5 Outcomes Due to Wheezing, Stratified by Race/Ethnicity, United States, 2003–2010*

	Non-Hispanic white (n = 235)			Non-Hispanic black (n = 354)			Mexican (n = 202)		
Characteristic	ETS-exposed (adj %)	Unexposed (adj %)	ARR (95% CI)	ETS exposed (adj %)	Unexposed (adj %)	ARR (95% CI)	ETS exposed (adj %)	Unexposed (adj %)	ARR (95% CI)
Missed school days past 12 mo									
0 d	65.7	81.1	0.8 (0.7, 1.0)†	66.5	57.9	1.2 (1.0, 1.4)	68.5	68.7	1.0 (0.8, 1.2)
1–7 d	26.8	15.4	1.7 (1.2, 2.6)†	24.8	30.1	0.8 (0.7, 1.0)	23.8	23.6	1.0 (0.7, 1.5)
8+ d	7.5	3.5	2.1 (1.2, 3.7)†	8.7	12.1	0.7 (0.5, 1.1)	7.8	7.7	1.0 (0.6, 1.9)
Health care visits past 12 mo									
0	57.0	72.0	0.8 (0.6, 1.0)†	62.1	58.3	1.1 (0.9, 1.3)	61.0	62.9	1.0 (0.8, 1.2)
1–2 visits	30.3	21.0	1.4 (1.0, 2.0)†	27.4	29.6	0.9 (0.7, 1.2)	26.7	25.6	1.0 (0.8, 1.4)
3+ visits	12.7	7.0	1.8 (1.1, 3.1)†	10.5	12.1	0.9 (0.6, 1.4)	12.3	11.5	1.1 (0.6, 1.8)
Nights with disturbed sleep past 12 mo									
0	55.4	74.1	0.8 (0.6, 0.9)†	61.9	54.9	1.1 (0.9, 1.4)	46.9	64.1	0.7 (0.6, 1.0)†
<1 night per week	29.2	18.5	1.6 (1.1, 2.2)†	21.6	24.2	0.9 (0.8, 1.1)	29.6	22.7	1.3 (1.1, 1.6)†
1+ nights per week	15.5	7.4	2.1 (1.3, 3.5)†	16.5	20.9	0.8 (0.5, 1.2)	23.5	13.2	1.8 (1.1, 2.9)†
Activity limitation past 12 mo									
None	55.9	77.9	0.7 (0.6, 0.9)†	73.4	62.4	1.2 (1.0, 1.4)	63.7	63.7	1.0 (0.8, 1.3)
A little	27.3	15.3	1.8 (1.3, 2.5)†	14.8	19.4	0.8 (0.6, 1.0)	25.7	25.7	1.0 (0.7, 1.5)
Fair/moderate/lot	16.8	6.8	2.5 (1.5, 4.2)†	11.8	18.2	0.7 (0.4, 1.0)	10.6	10.6	1.0 (0.5, 2.0)
Wheezing during exercise past 12 mo									
No	49.1	61.3	0.8 (0.6, 1.0)	54.2	54.1	1.0 (0.8, 1.3)	51.1	56.6	0.9 (0.7, 1.2)
Yes	50.9	38.7	1.3 (1.0, 1.8)	45.8	45.9	1.0 (0.7, 1.4)	48.9	43.4	1.1 (0.8, 1.6)

ARR = adjusted risk ratio; ETS = environmental tobacco smoke; adj = adjusted; CI = confidence interval.

*Adjusted for sex, race/ethnicity and poverty status, household ownership, and household size. Conditional marginal proportions (adjusted percentages) and ARRs were estimated by ordinal regression models for the ordered levels of the outcome variables.

†The 95% confidence interval excluded 1.0 before rounding.

Table 5. Adjusted Percentage and Relative Risk (ARR) of Outcomes Due to Wheezing Among Children With Asthma 6 to 11 Years of Age, by ETS Exposure Among Children With Serum Cotinine Levels <1.0 ng/mL, United States, 2003–2010*

Characteristic	Low Exposure (adj %) (n = 161)	Unexposed (adj %) (n = 137)	Low Exposure Versus Unexposed, ARR (95% CI)
Missed school days past 12 mo			
0 d	42.8	64.9	0.7 (0.5, 1.0)†
1–7 d	42.3	28.5	1.5 (1.1, 2.0)†
8+ d	15.0	6.7	2.3 (1.2, 4.4)†
Health care visits past 12 mo			
0 visits	43.4	56.9	0.8 (0.6, 1.0)
1–2 visits	37.1	30.7	1.2 (1.0, 1.5)
3+ visits	19.5	12.4	1.6 (0.9, 2.7)
Nights with disturbed sleep past 12 mo			
0	42.7	69.3	0.6 (0.5, 0.8)†
<1 night per week	27.5	18.4	1.5 (1.2, 1.9)†
1+ nights per week	29.8	12.3	2.4 (1.5, 3.9)†
Activity limitation past 12 mo			
None	48.9	72.0	0.7 (0.5, 0.9)†
A little	33.2	20.5	1.6 (1.1, 2.4)†
Fair/moderate amount/a lot	17.9	7.5	2.4 (1.3, 4.5)†
Wheezing during exercise past 12 mo			
No	43.2	69.2	0.6 (0.5, 0.8)†
Yes	56.8	30.8	1.8 (1.3, 2.7)†

ARR = adjusted risk ratio; ETS = environmental tobacco smoke; adj = adjusted; CI = confidence interval.

*Only results for children ages 6 to 11 years are shown; all 95% CI for children 12 to 19 years included 1.0. Conditional marginal proportions (adjusted percentage) and ARRs were estimated by ordinal regression models for the ordered levels of the outcome variables. Adjusted for sex, race/ethnicity, poverty status, home ownership and household size. Low ETS exposure was defined as serum cotinine levels 0.05 to <1.0 ng/mL. No ETS exposure was defined as serum cotinine levels <0.05 ng/mL.

†The 95% CI excluded 1.0 prior to rounding.

There are no straightforward explanations for the differences observed by race/ethnicity: ETS exposure was associated with adverse outcomes among non-Hispanic white children but not non-Hispanic black children with asthma. Among non-Hispanic black children without ETS exposure, the proportions of adverse outcomes were similar to or greater than all other groups (exposed non-Hispanic black children and exposed and unexposed children of other race/ethnic groups). One possibility is that race/ethnicity may encompass otherwise uncontrolled differences in disease severity and exposures. Our findings may suggest that for children facing a milieu of exposures typically associated with lower housing quality (mold, dander, pests, and traffic pollution), the benefits of reducing smoke exposure may be more difficult to demonstrate. It could also indicate selection bias among this group of apparently more severely affected children; caretakers of children with greater symptoms may make greater efforts to eliminate ETS exposure (ie, survivorship bias where the children with less severe asthma are more likely to remain among the exposed group).

We did not have measures of asthma severity equivalent to those specified by the NAEPP guidelines.[11] It is not clear that adjusting for asthma severity would be appropriate, given that the outcome measures used in this analysis often serve as direct or surrogate measures for asthma severity. However, we did perform a sensitivity analysis in which PAM use served as an indicator of underlying asthma severity.[11] The results were similar to the main analysis. However, given the disparities in PAM use,[29,30,31] it is likely that this variable only partially captures differences in underlying severity.

The use of NHANES data presents several advantages, including a nationally representative sample with a biologic measure of ETS exposure and the ability to control for demographic covariates, but also presents limitations. The recall period for all of the outcomes included in the analysis was 12 months. It is possible that more recent experiences were preferentially recalled and reported. There was a high rate of missing data for serum cotinine levels, which could have introduced nonresponse bias. However, an analysis using adjusted weights to account for item nonresponse suggested that no significant bias was introduced. Even with a laboratory measure of ETS exposure, misclassified exposure is another possible source of bias because of the relatively short half-life of serum cotinine. It is possible that at the time of the examination at the mobile exam center, children had unusually high or low serum cotinine levels compared to their usual baseline levels. Therefore, cotinine levels reflecting exposure in the recent past may not reflect actual exposure of the length of time of the measured outcomes (12 months). In addition, as with any analysis of cotinine levels as an ETS exposure indicator, our analysis measured exposure to nicotine and only indirectly measured exposure to other components of smoke. However, past studies have demonstrated that nicotine exposure reflects exposure to other constituents of ETS when exposure occurs over prolonged periods of time (several hours or days).[32] To assess whether reported exposure to a household smoker had patterns comparable to those for cotinine, we performed a sensitivity analysis using an exposure variable of reported household smoker rather than cotinine level. The results were similar (higher relative risk of poor outcomes among young children and

white children) but with wider confidence intervals as a result of the smaller sample with exposure to a household smoker.

Our analysis suggests that ETS exposure, even at levels consistent with low serum cotinine levels, continue to be associated with adverse outcomes among children with asthma. This association remains apparent in a nationally representative sample despite reductions in ETS exposure over recent decades and advances in preventive medication and asthma management. These current data indicate that the association between ETS exposure and many adverse outcomes exists for younger children ages 6 to 11 years, for non-Hispanic white children, and for Mexican American children for disturbed sleep due to wheezing. Although ETS exposure status was not associated with outcomes for non-Hispanic black children, this group had higher percentages of adverse outcomes among unexposed children compared to unexposed children of other racial/ethnic groups. Challenges remain in meeting recommendations to reduce ETS exposure and impact, and to better understand the different patterns observed between population subgroups.

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