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Association between adverse childhood experiences in the home and pediatric asthma

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ABSTRACT

Background: Numerous studies suggest that psychosocial factors could contribute to pediatric asthma.

Objective: To examine the relation between single and cumulative adverse childhood experiences (ACEs), a measurement of household dysfunction, on parent report of lifetime asthma in children.

Methods: This cross-sectional study used data from the 2011 to 2012 National Survey of Children's Health, a nationally representative sample of children 0 to 17 years old ($n = 92,472$). The main exposure was parent or guardian report of 6 ACE exposures (eg, witnessing domestic violence). The relation between ACE exposures and parent-reported diagnosis of childhood asthma was examined using multivariable logistic regression after controlling for demographic, socioeconomic, and behavioral covariates.

Results: Overall asthma prevalence was 14.6%. Exposure prevalence to any ACE was 29.2%. Increased number of ACE exposures was associated with increased odds of asthma. In the adjusted model, the odds of reporting asthma were 1.28 (95% confidence interval [CI] 1.14–1.43) for those reporting 1 ACE, 1.73 (95% CI 1.27–2.36) for those with 4 ACEs, and 1.61 (95% CI 1.15–2.26) for those with 5 or 6 ACEs compared with those with no ACE exposures. Effects were moderated by Hispanic ethnicity. Hispanic children exposed to 4 ACEs had a 4.46 times increase in lifetime asthma (95% CI 2.46–8.08); white children had a 1.19 times increase (95% CI 0.80–1.79) compared with those exposed to 0 ACE.

Conclusion: This study supports the growing evidence for the biopsychosocial model of asthma onset. Future studies should examine the association between ACEs and specific asthma-related health outcomes.

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Introduction

Asthma is one of the most common chronic childhood conditions, currently affecting 7 million children (9.5%) in the United States.^{1,2} Pediatric asthma is associated with significant morbidity and health care usage. In 2010, there were 641,000 emergency department visits attributable to asthma, representing 2.5% of all such visits by children 0 to 15 years of age.³ The direct medical costs and indirect costs from missed school and lost caregiver workdays are considerable.⁴

The biological risk factors for asthma onset and severity have been well established and supported by numerous studies. These include genetic factors, allergic rhinitis, allergens, tobacco smoke, air pollution, and respiratory infections.^{2,5–12} Research investigating the role of psychosocial stressors as a determinant of asthma

continues to increase. Psychosocial stress has been shown to alter susceptibility to infectious and systemic illnesses and enhance airway inflammation by modulating immune cell functions through complex hormonal and neural pathways.^{13–18} Studies have demonstrated that stress is a predictor of asthma morbidity in children and has been shown to predict the onset of asthma in children who are genetically at risk.^{18–21} Many studies have focused on neighborhood and urban-related stressors, such as family poverty, poor-quality housing, inadequate environmental control (eg, exposure to household allergens), and access to community resources.^{22–24} Recently, parental concern for neighborhood safety has been shown to correlate with asthma prevalence in children, suggesting exposure to neighborhood violence could be an important factor in the occurrence of childhood asthma.²⁵

Disruptive family relationships within the home can be a significant source of psychosocial stress for children. Many studies investigating the effects of adverse childhood experiences (ACEs) have shown strong graded relations between exposure to household dysfunction and abuse during childhood and the leading causes of adult morbidity and mortality, including coronary artery

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Table 1

Home adverse childhood experience questions, National Survey of Children's Health 2011

Did [the SC] ever
Live with a parent or guardian who was divorced or separated after [the SC] was born?
Live with a parent or guardian who died?
Live with a parent or guardian who served time in jail or prison after [the SC] was born?
Live with anyone who was mentally ill or suicidal or severely depressed for more than a couple of weeks?
Live with anyone who had a problem with alcohol or drugs?
See or hear any parents, guardians, or any other adults in [the SC's] home slap, hit, kick, punch, or beat each other up?

Abbreviation: SC, sample child.

disease, asthma, and mental illness.^{26–29} Recent work has investigated relations between ACEs and the cardiovascular health of children.³⁰ Studies also have examined the impact of exposure to household dysfunction during childhood on asthma development in specific populations.^{20,31,32} The present study investigated the association between parental report of lifetime asthma in children and specific ACEs pertaining to interpersonal stress within families. The authors hypothesized that children with increased numbers of ACE exposures would be more likely to have an increased prevalence of asthma. Recognition of this association might lead to better clinical care for children with these ACE risk factors. By investigating potential sources of childhood stress, clinicians also might gain a more complete understanding of the causes of increased asthma prevalence to design targeted interventions.

Methods

Study Population and Design

Data for this study were from the National Survey of Children's Health (NSCH), which is a cross-sectional telephone survey conducted over 16 months in 2011 to 2012 that gathered information from parents or guardians on the physical and emotional health of children 0 to 17 years old. This is a nationally representative sample of noninstitutionalized children in the United States. The NSCH is conducted by the Centers for Disease Control and Prevention's National Center for Health Statistics and primarily funded by the US Department of Health and Human Services. Households were sampled by random-digit dialing of landline and cellular telephone numbers. One child per household was randomly selected to be the subject of the detailed interview. The survey was conducted in English, Spanish, Mandarin, Cantonese, Vietnamese, and Korean. A full description of the survey can be found at www.cdc.gov/nchs/slaits/nsch.htm.³³

Of a sample of 187,422 reported age-eligible children, 95,677 had complete, detailed, child-level interviews (completion rate 51.0%). Those with missing information on reported lifetime asthma ($n = 84$) and ACEs ($n = 3,166$) were excluded, yielding a final analytic sample of 92,427 children, constituting 96.6% of the eligible population. No institutional review board approval was necessary because the study was based on a de-identified, publically available dataset.

Outcome

Asthma prevalence was measured using parent or guardian responses to the survey question, "Has a doctor or other health care provider ever told you that [sample child] had asthma?" expressed as a dichotomous yes-or-no variable.

Exposures

The NSCH provides parent- or guardian-reported data on 6 ACEs pertaining to interpersonal stress within households or families.

These questions, listed in Table 1, are expressed as dichotomous yes-or-no variables. As performed by previous researchers, the authors examined the cumulative effects of these ACEs by creating an ACE score.³⁴ Responses to each of the 6 ACE questions were summed (1 if "yes," 0 if "no") to produce the number of ACEs to which each participant was exposed. ACE scores of 5 and 6 were combined owing to small numbers in each of these categories and indicator variables were used in logistic regression analysis.

Covariates

Parents or caregivers provided demographic information, including the child's age, sex, race, family income, and highest level of household education obtained by anyone in the household. Household poverty level was calculated based on household size and reported income. Other previously identified risk factors for asthma, including prematurity,³⁵ smoking exposure,^{36–38} and neighborhood safety,²⁵ were analyzed. Household smoking exposure was defined as a dichotomous variable to the question, "Does anyone living in your household use cigarettes, cigars, or pipe tobacco?" Perceived neighborhood safety was used as a covariate because this perceived stress relates to the social context outside the home and has been examined in prior studies.²⁵ This was measured through the survey question, "How often do you feel [sample child] is safe in your community or neighborhood?" with possible responses of "never," "sometimes," "usually," or "always." The responses of "never" and "sometimes" were combined for the analysis because of small numbers.

Statistical Analysis

Statistical analyses were performed using STATA 12.1 (StataCorp LP, College Station, Texas). All analyses used the provided sampling weights to adjust for nonresponse and noncoverage and to obtain correct SEs, thus providing nationally representative estimates.³⁹ The descriptive statistics of demographics and covariates for children in the study were examined by categorizing the ACE score. Then, the unadjusted effect of the ACE score on the odds of reporting asthma were calculated and subsequently sequentially adjusted for demographic variables (sex, age, race or ethnicity, income, and household education), followed by other confounding variables associated with asthma severity (prematurity, household smoking, and exposure to neighborhood violence).

Results

The estimated lifetime prevalence of asthma in US children based on the study sample was 14.6%. Table 2 presents a comparison of demographic and health characteristics of children in the study by the number of ACE exposures. The study population included slightly more boys (51.2%) than girls (48.8%). Most children were identified as non-Hispanic white (52.7%), followed by "other" non-Hispanic (23.6%), non-Hispanic black (13.4%), and Hispanic (10.3%). There was a roughly equal distribution of ages in this study population. In general, older children (≥ 6 years) had an increased number of ACEs compared with younger children. As the number of ACEs increased, the percentage of households with an income below the 100% poverty level increased; 18.7% of those families with 0 ACE had a household income below the 100% poverty level compared with 35.7% of children with at least 5 ACEs. Similarly, household education (households in which ≥ 1 parent or guardian had more than a high school education) was lower in those families in which at least 1 ACE was reported, with 70.6% in the 0 ACE category compared with 62.7% in the category of at least 5 ACEs. Children with at least 1 ACE had greater smoking exposure than those exposed to 0 ACE, with 51.5% of those exposed to at least 5 ACEs living with someone who smoked compared with 18.0% of those with 0 ACE. Most respondents always believed themselves

Table 2
Distribution of selected demographic, socioeconomic, and health characteristics in children 0 to 17 years old by reported ACEs, National Survey of Children's Health 2011

Variable	0 ACE, %	1 ACE, %	2 ACEs, %	3 ACEs, %	4 ACEs, %	≥5 ACEs, %	Total, % (N)
Total population	68.7	17.7	7.1	3.8	1.9	0.93	100 (92,427)
Asthma ever							
Yes	12.3	18.2	18.7	23.2	24.5	25.4	14.6 (13,049)
No	87.7	81.8	81.3	76.8	75.5	74.6	85.4 (79,378)
Sex							
Boys	51.3	51.3	49.8	50.6	50.3	52.2	51.2 (47,542)
Girls	48.7	48.7	50.2	49.4	49.7	47.8	48.8 (44,786)
Age (y)							
0–5	39.2	21.3	18.7	13.8	13.2	15.6	32.9 (29,119)
6–11	31.7	35.7	38.0	35.1	35.4	32.1	33.1 (29,911)
12–17	29.1	42.9	43.4	51.2	51.3	52.2	34.0 (33,397)
Race							
NH white	52.9	51.7	49.8	55.7	58.0	57.9	52.7 (60,383)
NH black	11.9	17.6	17.0	15.8	12.7	10.3	13.4 (8,592)
Hispanic	10.5	9.4	9.4	10.8	10.8	12.6	10.3 (10,178)
Other	24.7	21.3	23.9	17.7	18.5	19.2	23.6 (12,407)
Prematurity							
No	88.1	87.9	88.0	90.5	87.5	84.4	88.5 (81,419)
Yes	11.9	12.1	12.0	9.5	12.5	15.6	11.5 (10,398)
Poverty level							
<100%	18.7	25.2	29.5	32.1	35.5	35.7	21.7 (12,720)
100–200%	20.2	23.8	27.1	28.5	28.0	27.4	21.9 (15,139)
200–400%	28.9	29.0	28.1	26.8	28.0	26.2	28.7 (26,231)
>400%	32.2	21.9	15.3	12.6	8.5	10.7	27.7 (30,982)
Household education							
<HS	11.5	11.2	13.8	13.2	14.0	12.5	11.7 (5,464)
HS graduate	18.0	24.3	25.9	22.3	27.3	24.9	20.0 (14,110)
>HS	70.6	64.5	60.3	64.5	58.7	62.7	68.3 (72,304)
Smoking exposure							
No	82.0	68.8	58.0	54.0	48.7	48.5	76.0 (70,925)
Yes	18.0	31.2	42.0	46.0	51.3	51.5	24.0 (21,457)
Neighborhood safety							
Never or ST	12.3	14.3	17.5	16.7	18.4	17.0	13.3 (8,637)
Usually	29.5	30.1	32.5	34.5	34.3	32.1	30.1 (28,524)
Always	58.3	55.6	49.9	48.8	47.2	50.9	56.6 (91,940)

Abbreviations: ACE, adverse childhood experience; HS, high school; NH, non-Hispanic; ST, sometimes.

safe in their neighborhood, but this generally decreased with an increased number of ACEs (Table 2).

Of all children in the sample, 31.3% were exposed to at least 1 ACE. Approximately 1% of children in the sample were exposed to at least 5 ACEs, with 105 children having reportedly experienced all 6 intrafamilial ACEs studied. The most common ACEs were living with a parent or guardian who was divorced or separated (19.7%), living with someone who had an alcohol or drug problem (10.3%), and living with someone who was mentally ill or suicidal (8.5%). The least common ACE was having a parent or guardian who died (3.0%). All ACE categories had a larger percentage of children with asthma compared with the overall study population. Of children with exposure to at least 5 ACEs, 25.4% of parents or guardians reported an asthma diagnosis compared with 12.3% with 0 ACE exposure.

There was a significant association of intrafamilial ACE exposures and the odds of reporting lifetime asthma in children (Table 3). In unadjusted models, children with 1 ACE had a 1.58 times increase in reporting an asthma diagnosis (95% confidence interval [CI] 1.43–1.75) compared with those with no ACE. The odds ratios for reporting lifetime asthma in children exposed to increasing numbers of ACEs increased with each additional ACE. Children with at least 5 ACEs had 2.42 times increased odds of reporting asthma (95% CI 1.79–3.27) compared with those with no ACE. This effect was slightly attenuated but still considerable after sequential adjustments for demographic and other covariates, thus decreasing the odds of reported asthma associated with at least 5 ACEs to 1.61 (95% CI 1.15–2.26).

Significant associations for the odds of lifetime asthma in the fully adjusted model also were found with sex, age, race or

ethnicity, poverty level, prematurity, and smoking exposure (Table 3). Boys had a higher likelihood of asthma, with 1.32 times the odds of reported asthma (95% CI 1.21–1.45) compared with girls. Children in the older groups had increased odds of a lifetime asthma diagnosis. Non-Hispanic black and Hispanic children were more likely to have an asthma diagnosis compared with non-Hispanic white children. Children with a family income lower than 100% of the federal poverty level were more likely to be reported to have asthma compared with children in households with an income higher than 400% of the poverty level. However, households with intermediate incomes of 100% to 400% of the poverty level had no significant increase in asthma diagnosis compared with the highest income category. Prematurity and smoke exposure were independently associated with increased odds of reporting asthma. Reported neighborhood safety was not associated with reported lifetime asthma.

As shown in Figure 1, the adjusted odds of reported lifetime asthma increased as the number of ACEs increased, up to 4 ACEs (test of linear trend, $P < .001$). Then, the odds stabilized for those children exposed to at least 5 ACEs.

A subanalysis stratified by age showed that younger children (0–5 years old) had a similar pattern to the entire study population, with a significant association of intrafamilial ACE exposures and the odds of reporting lifetime asthma in children (Table 4). Children no older than 5 years and exposed to 3 ACEs had 4.94 times increased odds of reporting asthma (95% CI 3.16–7.73) compared with those with no ACE. Children 6 to 11 years old showed a similar trend, but results were not significant, except for those children exposed to at least 5 ACEs who had 2.36 times increased odds of reporting asthma (95% CI 1.36–4.10) compared with those with no ACE. The

Table 3

Crude and adjusted odds of lifetime asthma by ACE exposure score and covariates, National Survey of Children's Health 2011

Characteristics	Unadjusted, OR (95% CI)	Model 1, ^a adjusted OR (95% CI)	Model 2, ^b adjusted OR (95% CI)
ACE exposures			
0	1.00 (ref)	1.00 (ref)	1.00 (ref)
1	1.58 (1.43–1.75) ^c	1.31 (1.17–1.47) ^c	1.28 (1.14–1.43) ^c
2	1.63 (1.40–1.90) ^c	1.31 (1.10–1.55) ^c	1.27 (1.06–1.51) ^c
3	2.15 (1.81–2.56) ^c	1.67 (1.37–2.03) ^c	1.63 (1.34–1.99) ^c
4	2.30 (1.73–3.07) ^c	1.75 (1.29–2.38) ^c	1.73 (1.27–2.60) ^c
≥5	2.42 (1.79–3.27) ^c	1.73 (1.26–2.38) ^c	1.61 (1.15–2.26) ^c
Sex			
Girls	1.00 (ref)	1.00 (ref)	1.00 (ref)
Boys	1.27 (1.17–1.37) ^c	1.32 (1.21–1.44) ^c	1.32 (1.21–1.45) ^c
Age (y)			
0–5	1.00 (ref)	1.00 (ref)	1.00 (ref)
6–11	2.07 (1.85–2.31) ^c	2.08 (1.84–2.35) ^c	2.10 (1.86–2.38) ^c
12–17	2.48 (2.23–2.77) ^c	2.47 (2.18–2.79) ^c	2.52 (2.22–2.84) ^c
Race			
NH white	1.00 (ref)	1.00 (ref)	1.00 (ref)
NH black	1.98 (1.78–2.20) ^c	1.88 (1.66–2.13) ^c	1.83 (1.62–2.08) ^c
Hispanic	1.17 (1.03–1.33) ^c	1.27 (1.11–1.45) ^c	1.23 (1.07–1.41) ^c
Other	1.05 (0.90–1.14) ^c	1.08 (0.93–1.25)	1.09 (0.94–1.27)
Poverty level			
<100%	1.48 (1.32–1.66) ^c	1.36 (1.18–1.56) ^c	1.26 (1.09–1.46) ^c
100–200%	1.23 (1.09–1.38) ^c	1.12 (0.98–1.28)	1.07 (0.94–1.23)
200–400%	1.11 (0.99–1.23)	1.06 (0.95–1.19)	1.05 (0.94–1.17)
>400%	1.00 (ref)	1.00 (ref)	1.00 (ref)
Household education			
<HS	1.09 (0.97–1.23)	0.93 (0.80–1.07)	0.92 (0.80–1.06)
HS graduate	1.11 (1.01–1.21) ^c	1.00 (0.91–1.10)	0.99 (0.90–1.09)
>HS	1.00 (ref)	1.00 (ref)	1.00 (ref)
Prematurity		—	
No	1.00 (ref)		1.00 (ref)
Yes	1.62 (1.45–1.81) ^c		1.67 (1.49–1.88) ^c
Smoking exposure		—	
No	1.00 (ref)		1.00 (ref)
Yes	1.37 (1.26–1.50) ^c		1.17 (1.06–1.30) ^c
Neighborhood safety		—	
Never or ST	1.27 (1.12–1.45) ^c		1.14 (0.98–1.32)
Usually	1.00 (0.92–1.10)		0.96 (0.87–1.05)
Always	1.00 (ref)		1.00 (ref)

Abbreviations: ACE, adverse childhood experience; CI, confidence interval; HS, high school; NH, non-Hispanic; OR, odds ratio; ref, reference; ST, sometimes.

^aModel 1 adjusted for demographic variables only (sex, age, race, poverty level, and household education).^bModel 2 adjusted for all variables listed in table.^cDenotes statistical significance.

results of those children 12 to 17 years old are complex, with no trend and variable statistical significance.

Another subanalysis stratified by race or ethnicity showed that children reported as Hispanic and non-Hispanic “other” had higher odds of lifetime asthma for the same number of ACEs

Table 4

Adjusted odds of lifetime asthma by ACE exposure score and age, National Survey of Children's Health 2011

ACE exposures	0–5 y, AOR (95% CI) ^a	6–11 y, AOR (95% CI) ^a	12–17 y, AOR (95% CI) ^a
0	1.00 (ref)	1.00 (ref)	1.00 (ref)
1	2.20 (1.70–2.85) ^b	0.96 (0.80–1.14)	1.25 (1.05–1.49) ^b
2	3.00 (1.86–4.83) ^b	1.07 (0.79–1.45)	1.07 (0.86–1.32)
3	4.94 (3.16–7.73) ^b	1.23 (0.89–1.71)	1.48 (1.13–1.95) ^b
4	3.13 (1.76–5.56) ^b	1.63 (0.92–2.87)	1.50 (0.96–2.23)
≥5	1.32 (0.48–3.60)	2.36 (1.36–4.10) ^b	1.26 (0.82–1.94)

Abbreviations: ACE, adverse childhood experience; AOR, adjusted odds ratio; CI, confidence interval; ref, reference.

^aAdjusted for all variables.^bDenotes statistical significance.

compared with non-Hispanic white and non-Hispanic black children (Table 5). Of Hispanic children, those exposed to 4 ACEs had a 4.46 times increase in lifetime asthma (95% CI 2.46–8.08) compared with those exposed to 0 ACE. Of non-Hispanic white children, those exposed to 4 ACEs had only a 1.19 times increase odds of lifetime asthma (95% CI 0.80–1.79) compared with those exposed to 0 ACE.

Discussion

In this large, population-based, nationally representative survey, a strong positive association was found between childhood intra-familial ACE exposure and reported lifetime asthma in children. Children exposed to even just 1 ACE had a 28% increased odds of reported lifetime asthma compared with those with no ACE. These odds increased with each additional ACE, up to 4 ACEs, with children exposed to 4 ACEs having a 73% increased odds of reported lifetime asthma compared with those with no ACE. The effects of cumulative ACE exposures have been studied in adults,^{26,34} but this is the first study to investigate specific ACEs experienced in the home and their cumulative impact on childhood asthma prevalence. The present study controlled for perception of neighborhood safety, a factor shown to be associated with asthma prevalence,²⁵ and therefore highlights the effects of exposure to ACEs within the home environment when controlled for some external environmental exposures.

The present findings support the growing literature on the biopsychosocial model of asthma development. Psychosocial stressors are known to activate the sympathetic nervous system and the hypothalamic–pituitary–adrenocortical axis, leading to secretion of catecholamines and cortisol, which affect the expression and activity of many immune cells. Transient increases in these hormones are protective, but excessively high or prolonged exposures can be harmful. Negative emotional responses can disturb the regulation of these axes, causing chronic under- or overactivity of this normally balanced system. Stress has been shown to lead to a shift in the immune response toward a T-helper cell type 2 cytokine phenotype, leading to subsequent recruitment of inflammatory cells that can initiate or potentiate atopic inflammation characteristic of asthma.¹⁵ Animal studies have suggested that social stress induces corticosteroid insensitivity and can cause mast cell degranulation, which are responsible for many symptoms of atopic diseases such as asthma.^{13,40} There is also strong evidence that the genes involved in stress and inflammatory responses can affect the expression of asthma phenotypes.¹⁴

Many studies have suggested that stressful life events can increase the onset of asthma.^{41,42} Prenatal and early postnatal maternal stress has been significantly associated with early childhood wheezing.⁴³ One study showed that children born to mothers who experienced stressful life events during pregnancy, including

Adjusted Odds of Asthma Prevalence by ACE Score

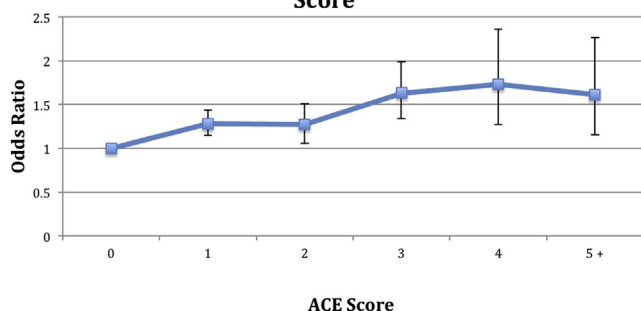


Figure 1. Adjusted Odds of Asthma Prevalence by ACE Score. Error bars indicate the confidence interval around the odds ratios.

Table 5

Adjusted odds of lifetime asthma by ACE exposure score and race/ethnicity, National Survey of Children's Health 2011

ACE exposures	NH white, AOR (95% CI) ^a	NH black, AOR (95% CI) ^a	Hispanic, AOR (95% CI) ^a	Other, AOR (95% CI) ^a
0	1.00 (ref)	1.00 (ref)	1.00 (ref)	1.00 (ref)
1	1.09 (0.94–1.26)	1.03 (0.81–1.32)	2.23 (1.56–3.20) ^b	1.56 (1.13–2.15) ^b
2	1.12 (0.90–1.35)	0.90 (0.59–1.38)	1.72 (1.15–2.56) ^b	1.71 (1.09–2.67) ^b
3	1.46 (1.13–1.88) ^b	1.04 (0.64–1.68)	3.26 (2.08–5.12) ^b	1.93 (1.11–3.35) ^b
4	1.19 (0.80–1.79)	1.60 (0.71–3.62)	4.46 (2.46–8.08) ^b	2.22 (0.93–5.29)
≥5	1.31 (0.80–2.13)	1.92 (0.74–4.97)	1.97 (0.84–4.61)	1.57 (0.72–3.44)

Abbreviations: ACE, adverse childhood experience; AOR, adjusted odds ratio; CI, confidence interval; NH, non-Hispanic; ref, reference.

^aAdjusted for all variables, including poverty level.^bDenotes statistical significance.

divorce, mourning, or loss of a job, had 1.71 times the odds of having asthma later in childhood.⁴⁴ A large, population-based study of adults found that high exposure to stressful life events was associated with a 2-fold increased risk of subsequent asthma development.⁴⁵

In addition to affecting the onset of asthma, exposure to stress has been shown to predict higher asthma morbidity in children.^{21,24,46} A subanalysis of children with reported current asthma in the present study failed to show a significant association between reported severity of asthma and ACE exposure (results not shown). The NSCH had only 1 question relating to asthma severity (“Would you describe [sample child’s] asthma as mild, moderate, or severe?”) asked to those whose child currently had asthma. Parents or guardians experiencing a higher level of household stress might tend to under-report their child’s severity of asthma if other life stressors are more severe than their child’s health. Therefore, subjective measurement of asthma severity is not a precise indicator for this variable.

The authors stratified by age to examine a possible relation between ACE exposures and asthma at different developmental levels. For children in the youngest group (0–5 years of age), the adjusted odds of reported lifetime asthma increased as the number of ACEs increased, up to 4 ACEs. A similar trend was seen in the middle age group (6–11 years old), but did not reach statistical significance. The results in the oldest group (12–17 years old) are difficult to interpret. This oldest group is one in which there has been more time to accumulate ACE exposures. The cross-sectional nature of this study is most limiting in this group because an asthma diagnosis might have been made before ACE exposure.

Approximately 5% of surveys were completed in a language other than English. Owing to the concern that interpretations of survey items could vary by language, the authors ran the model restricted to English only and the results were very similar (results not shown).

The observed race or ethnicity group differences in reported asthma prevalence are important in light of what is known about pediatric asthma disparities. In this study, children identified as non-Hispanic black or Hispanic had 1.83 and 1.23 (95% CI 1.62–2.08 and 1.07–1.41, respectively) times the odds of reporting asthma compared with non-Hispanic white children after controlling for all variables, including income level. This supports previous literature that urban African-American and Latino children are more likely to be diagnosed with asthma compared with non-Latino whites.^{47,48} Analyses examining the relation between intrafamilial ACEs and reported asthma prevalence by race or ethnicity showed interesting results (Table 4). Hispanic children or those identified as “other” race or ethnicity were found to have a statistically significant association between ACEs and asthma prevalence. Most notably, Hispanic children exposed to 4 home ACEs had 4.46 (95% CI 2.46–8.80) times the odds of asthma compared with those exposed to no home ACE. However, in general, the association was not supported in children identified as non-Hispanic white or non-Hispanic black. These findings implicate the possibility of other

culturally specific stressors or differences in home environments that could contribute to asthma development in the face of other ACE exposures in the home in these minority populations. Indeed, some cultural risks, such as discrimination and acculturative stress, are known to contribute to asthma morbidity in African-American and Latino children.⁴⁹ Therefore, these stressors also could contribute to asthma development. In addition, aspects of the home environment, such as housing deterioration and allergen exposure, and family routines and schedules differing among these ethnic groups, have been shown to affect asthma management and likely contribute to asthma development.^{50,51}

The use of the NSCH, a large, population-based, nationally representative survey, generalizable to noninstitutionalized children in the United States, is a clear strength of this study. However, there are several limitations to this study that warrant consideration. First, the NSCH is based entirely on parent and guardian subjective reports, which are vulnerable to recall bias. Childhood asthma in the NSCH is based on a single question, rather than objective data, such as spirometry or physician diagnosis. Over- or under-reporting of asthma in this study is possible. The prevalence estimates observed in this study are higher than other national estimates, possibly indicating over-reporting of asthma.² Second, ACEs are sensitive questions subject to social desirability reporting bias that can lead to misclassification of risk exposure. Respondents could have been reluctant to admit that their child was exposed to ACEs in the home, such as exposure to domestic violence or living with someone with an alcohol or drug problem. Third, owing to the nature of the study, the authors cannot rule out confounding by various unobserved confounders, such as family history of atopy, allergen exposures, and living conditions. In addition, parents or guardians might not be aware of their child’s smoking status, which is a known risk factor for asthma development. Fourth, this cross-sectional survey does not specify timing of ACE exposure or asthma diagnosis. Therefore, the outcome of interest could have occurred before ACE exposure. Similarly, the authors cannot rule out reverse causation. One could postulate that having a child with asthma might lead to stress, which might precipitate other home ACEs, such as parental separation or divorce.

The findings of this study further deepen the understanding of the psychosocial risk factors of asthma development in children. To the authors’ knowledge, this is the first study to show an association between home ACEs and childhood lifetime asthma. The cumulative effects of ACEs are particularly interesting and underscore the need for close monitoring of these particularly “high-risk” children, especially those of Hispanic or “other” race or ethnicity. By exploring potential sources of childhood stress, such as the specific ACEs explored in the present study, clinicians might have a more complete understanding of the complex causes of asthma and might be able to better target preventative medications and other interventions accordingly. Future studies should examine the association between home ACEs and specific asthma-related health outcomes, such as physician office visits, emergency department visits, or hospitalizations. In addition, intervention and prevention

efforts that target these stressors, through decreased stress perception or increased capability of handling stressors, should be studied to determine the effect on asthma prevalence and outcomes.

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