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Stress and the City: Housing Stressors are Associated with Respiratory Health among Low Socioeconomic Status Chicago Children

Kelly Quinn, Jay S. Kaufman, Arjumand Siddiqi, and Karin B. Yeatts

ABSTRACT *Asthma disproportionately affects non-whites in urban areas and those of low socioeconomic status, yet asthma's social patterning is not well-explained by known risk factors. We hypothesized that disadvantaged urban populations experience acute and chronic housing stressors which produce psychological stress and impact health through biological and behavioral pathways. We examined eight outcomes: six child respiratory outcomes as well as parent and child general health, using data from 682 low-income, Chicago parents of diagnosed and undiagnosed asthmatic children. We created a continuous exposure, representing material, social and emotional dimensions of housing stressors, weighted by their parent-reported difficulty. We compared the 75th to the 25th quartile of exposure in adjusted binomial and negative binomial regression models. Higher risks and rates of poor health were associated with higher housing stressors for six of eight outcomes. The risk difference (RD) for poor/fair general health was larger for children [RD=6.28 (95% CI 1.22, 11.35)] than for parents [RD=3.88 (95% CI −1.87, 9.63)]. The incidence rate difference (IRD) for exercise intolerance was nearly one extra day per 2 weeks for the higher exposure group [IRD=0.88 (95% CI 0.41, 1.35)]; nearly one-third extra day per 2 weeks for waking at night [IRD=0.32 (95% CI 0.01, 0.63)]; and nearly one-third extra day per 6 months for unplanned medical visits [IRD=0.30 (95% CI 0.059, 0.54)]. Results contribute to the conceptualization of urban stress as a "social pollutant" and to the hypothesized role of stress in health disparities. Interventions to improve asthma outcomes must address individuals' reactions to stress while we seek structural solutions to residential stressors and health inequities.*

KEYWORDS *Asthma, Chronic disease, Psychological stress, Stressors, Health disparities, Environmental health, Inner-city health, Low socioeconomic status and health*

INTRODUCTION

Asthma, one of the most common chronic diseases of childhood in the United States, disproportionately affects non-whites in urban areas and those of low socioeconomic status (SES).[1,2] Chicago's asthma prevalence and morbidity and mortality rates are among the highest in the nation, and within the city, they are typically

Quinn and Yeatts are with the Department of Epidemiology, University of North Carolina Gillings School of Global Public Health, Chapel Hill, NC, USA; Kaufman is with the Department of Epidemiology, Biostatistics, and Occupational Health, McGill University, Montreal, Quebec, Canada; Siddiqi is with the Department of Health Behavior and Health Education, University of North Carolina Gillings School of Global Public Health, Chapel Hill, NC, USA.

Correspondence: Kelly Quinn, Department of Epidemiology, University of North Carolina Gillings School of Global Public Health, Chapel Hill, NC, USA. (E-mail: kaquinn@email.unc.edu)

highest in neighborhoods with the lowest SES.[3–8] Chicago's burden, and the national epidemic, are not well-explained by known risk factors. Not all urban communities have excess asthma prevalence and morbidity though they may share low-SES and environmental exposures with high-risk urban areas.[3,9–11] Wright and Subramanian call for attention to "social and physical factors that covary with lower SES and minority-group status (e.g., differential environmental exposures, residential segregation, psychological stress, housing quality, and social capital) that mediate the effects of living in low-SES neighborhoods" to contextualize asthma and understand its social patterning.[11]

Psychological stress has been conceptualized as a "social pollutant that when 'breathed into the body' may disrupt biological systems through inflammatory processes".[10,12] Evidence demonstrates that acute and chronic stressors have psychological effects that influence psychologic and physiologic functioning as well as behavior.[13] Studies link stress to asthma onset, exacerbation and phenotypes through hypothesized "dysregulated immunity" mechanisms.[10,12,14–22] Stress experienced by children or their parents may also have indirect effects on asthma by causing health-compromising behaviors and comorbidities that adversely affect disease management.[23–34] Caregiver stress, negative life events, problematic family relationships, parenting difficulties, critical attitudes of one's mother and violence exposure have been related to wheeze, asthma onset, and/or adverse asthma outcomes among infants and youths.[14,15,20–22,29–32,35–41]

The housing and health relationship has long been acknowledged, not surprising given that water, warmth, air, shelter, and safety are fundamental human needs.[42] US children spend as much as 80–90% of their time indoors.[43] Urban-dwelling children may be kept at home because of concerns about safety outdoors and may thus experience increased risk of health-compromising indoor exposures and behaviors. Housing stressors may directly harm respiratory health, for example, when utilities are not in service, leading to poor ventilation, dampness, and mold. The focus of this paper, however, is psychological distress caused by stressors.

Low-SES and minority populations often have increased exposure to environmental and psychosocial stressors.[35,44–46] and may be more strongly affected by stressors due to already-compromised psychological health, social supports, coping resources, and lack of control over individuals' lives.[44,47–52] Just as material exposures act on biology, social environments may "get under the skin"[53] and become "biologically embedded"[54] to influence health.[10,11] Researchers posit that: (1) shelter is a fundamental necessity; (2) housing units create an infrastructure for group life, important for well-being; and (3) housing provides homes, with psychological importance as objects of attachment and sources of identity.[55] We hypothesize that stressors stemming from housing security, mobility, comfort, safety, finances, dynamic household membership, and relationships with neighbors and landlords cause stress, which influences immunological and behavioral pathways to health for parents and children.

The ongoing burden of asthma on inner-city populations demands more thoughtful investigation of determinants of risk. This study, cross-sectional in design and limited to Chicago, does not incorporate all known risk factors or test causation. Instead, it aims to advance asthma scholarship by incorporating novel exposure variables into a theoretical framework and furthering our understanding of how psychosocial factors become biologically embedded and influence health through stress pathways. We use unique data not collected in large-scale health studies to create a multidimensional measure of policy-relevant stressors reflecting

material, social and emotional aspects of housing for a low-income, urban, racially/ethnically heterogeneous sample of parents of young children. We examine associations between the number of housing stressors experienced by families, weighted by parent-reported difficulty, and health outcomes, with emphasis on childhood respiratory health under the hypothesis that a higher stressors score is associated with increased risk of poorer health outcomes.

METHODS

Study Population and Design

The Institutional Review Board of the University of North Carolina at Chapel Hill approved this study. We used a cross-sectional study design to characterize the relationship between parents' experience of housing stressors and eight parent-reported outcomes: six child respiratory health outcomes as well as parent and child general health.

We used survey data collected in 2002–2004 for an observational investigation of childhood asthma disparities among low-income Chicago families with children ages 5–13 years. The main study surveyed for respiratory problems in schools with a parent-completed tool, validated in English and in Spanish, with good sensitivity and specificity for identifying children with undiagnosed asthma (any one of four signs/symptoms but no diagnosis was classified as "undiagnosed asthma".[56,57] The first 15 public elementary schools that agreed to participate and met the following eligibility criteria were surveyed: more than 75% of enrollment qualified as low-income; no single racial/ethnic group comprised more than two-thirds of enrollment; and only local residents were enrolled. For the main study, eligible families (n= 1,244) were randomly selected from among the students surveyed in the schools for a longitudinal study with 3 data collection phases over 12 months in English or Spanish. Our current study used data from a total of 682 parents: 351 parents of children with diagnosed asthma and 331 parents of children with undiagnosed asthma obtained during the baseline telephone survey. Children with no asthma diagnosis and no respiratory symtoms were excluded since the focus of our analyses was child respiratory health outcomes (rather than onset of asthma).

Analytic Strategy

We used SAS 9.1 (SAS Institute Inc., Cary, North Carolina) for estimation of risk differences (RDs) and incidence rate differences (IRDs) by binomial and negative binomial regression, respectively, STATA 10.1 (StataCorp, College Station, Texas) for graphical data displays, and the Microsoft Excel program "Episheet" (version of June 11, 2008) written by Kenneth J. Rothman for additional tabular analyses.

Regression Analyses

Univariate examination of dichotomous outcomes defined as any sign, symptom, utilization or absence vs. none, or unfavorable (i.e., poorer general health or control) vs. favorable group revealed that these outcomes were common (16–47%). Odds ratios would therefore overestimate relative risks. We estimated absolute measures of effect and 95% confidence intervals (CIs) for associations between housing stressors and outcomes for cases with complete data. Binomial regression estimated RDs for binary outcomes (SAS GENMOD specifying identity link function and binomial distribution) while negative binomial regression to address overdispersion

caused by the large proportion of zero counts estimated IRDs for count outcomes (log likelihood function modeled in SAS NLMIXED). Bivariate analyses estimated crude risk and rate differences.

We assessed effect measure modification (EMM) by Mantel–Haenszel chi-squared tests of homogeneity (binary outcomes) and stratification and comparison of RD estimates and CIs (count outcomes) and did not observe consistent or strong evidence of modification for variables including: child asthma diagnosis, race/ethnicity, age and sex; parent asthma diagnosis, age, nativity, marital status, education, depressive symptoms and smoking; presence of household smoker; home ownership; housing type; overcrowding; and household recipient of Temporary Assistance to Needy Families (TANF).

We first identified potential confounders in directed acyclic graph (DAG) analyses;[58] therefore, we did not quantitatively assess variables on causal pathways, variables that were not associated with both outcome and exposure, and variables that had hypothesized bi-directional associations with other variables as confounders. We then assessed all potential confounders with a change-in-estimate strategy[59] and adjusted for them in multivariate models created by backward elimination, with removal from the full model of covariates that changed the magnitude of association by <10%. We evaluated the predictive importance of the exposure by the magnitude of the RDs and IRDs and the width of the CIs.

Variables

Main Exposure. We created a variable representing the number and parent-rated difficulty of housing stressors experienced in a 6-month period capturing material, social, and emotional dimensions of housing. We identified 13 survey items representing potential sources of psychological stress caused by housing issues (Table 1). We computed a continuous score by summing the stressors reported by each parent, each weighted by the parent-reported difficulty of the experience on a scale of 1 (not at all) to 4 (a lot). In regression modeling, goodness-of-fit tests supported the square root transformation of this variable.

Health Outcomes. Each of eight outcomes was modeled separately. We recoded three parent-reported health outcomes into binary variables after determining that ordinal coding was not appropriate due to violation of the proportional odds assumption; the more favorable outcome served as the referent category. Child and parent general health were recoded as poor or fair vs. good or very good or excellent. Controllability of child's asthma was recoded as not at all or somewhat vs. quite or extremely controllable. Five outcomes specific to children's asthma or breathing problems were included in models as counts: exercise intolerance, waking at night, school absences and rescue medication were reported for a 2-week period, and the number of unplanned visits to an emergency department, physician's office or clinic were reported for the previous 6 months. Analyses for rescue medication included only diagnosed asthmatics since undiagnosed children might not have access to prescribed asthma medications.

Sociodemographic Covariates. Since we identified potential confounders conceptually in DAG analyses and then quantitatively assessed a subset with the 10% change-in-estimate criterion, each model had a potentially unique set of adjustment variables. Covariates included: parent education (did not complete high school vs. high school diploma or beyond), marital status (single vs. married or cohabiting),

TABLE 1 Descriptives for parent-reported housing stressors occurring in the previous 6 months and their difficulty level used to create weighted main exposure ($n = 682$)

Housing stressor	Event reported by parent number (%)	Parent-reported difficulty[a] mean (SD)[b]
Did the utility or phone company threaten to cut off service because you couldn't pay bills?	205 (30.1)	3.1 (1.0)
Did you miss a rent or mortgage payment because you couldn't pay for it?	136 (19.9)	3.4 (0.9)
Did you go without furniture because you did not have the money to pay for it?	103 (15.1)	2.5 (1.2)
Did rats, mice or insects bother you in your home?	102 (15.0)	3.1 (1.1)
Was your telephone, electricity or gas turned off?	95 (13.9)	3.2 (1.1)
Did you go without appliances because you did not have the money to pay for them?	63 (9.2)	2.9 (1.1)
Did you move?	60 (8.8)	2.4 (1.4)
Did a relative or friend move into your home?	58 (8.5)	2.2 (1.1)
Did a relative or friend move out of your home?	54 (7.9)	2.3 (1.2)
Did you have trouble with your neighbors?	46 (6.7)	3.1 (1.1)
Did you have trouble with your landlord?	42 (6.2)	3.4 (0.9)
Did you lose your housing?	19 (2.8)	3.8 (0.6)
Were you a victim of a crime while you were in your own home?	13 (1.9)	3.1 (1.1)

[a]Parents who reported that the event occurred in the previous 6 months rated its difficulty on a four-point scale: not at all (1), a little bit (2), medium (3), a lot (4); difficulty ratings were used to weight individuals' housing stressors score

[b]SD: standard deviation

and nativity (foreign-born vs. not); child race/ethnicity (non-Hispanic black, non-Hispanic white, Hispanic); home ownership (rent vs. own); housing type (multi-unit building vs. single-family home); overcrowding (1.01 or more persons per room vs. <1.01); and TANF (household recipient vs. none).

RESULTS

Univariate Descriptives

Sample. The baseline survey achieved a 64% response rate for the randomly selected study population. Table 2 presents descriptive statistics for 351 diagnosed and 331 undiagnosed asthmatic children and their families. Nearly half of children were identified as Hispanic, 39% were non-Hispanic black and 13% were non-Hispanic white. Only 7% of children but 34% of parents were foreign-born, and 23% of parents spoke Spanish for the survey. Slightly more boys than girls participated, but the majority of parent respondents were female. Over half of parents were married or cohabiting, 27% had not completed high school, and 28% of households received TANF. Thirty-one percent of families owned their home, 40% were single-family structures, and 32% were overcrowded.

Exposure. Thirty-seven percent of parents reported no housing stressor during the previous 6 months, and no parent reported more than nine (of 13 possible). For the

TABLE 2 Descriptive statistics for parents and children in study population, main exposure, and health outcomes ($n=682$)

Sociodemographics	Number (percent)	Mean	Med[a]	SD[a]	Min–max[a]
Race/ethnicity[b]					
Non-Hispanic white	90 (13.2)				
Non-Hispanic black	263 (38.6)				
Hispanic	329 (48.2)				
Foreign-born					
Child	45 (6.6)				
Parent	229 (33.6)				
Spanish language used for survey (vs. English)	157(23.0)				
Female					
Child	291 (42.7)				
Parent	639 (93.7)				
Did not complete high school education	185 (27.3)				
TANF recipient in household[c]	192 (28.4)				
Parent married/cohabiting partner	390 (57.3)				
Homeownership	210 (30.8)				
Housing type (single family, not multi-unit)	274 (40.4)				
Overcrowded household[d]	216 (31.7)				
Child health insurance					
Public	321 (47.2)				
Private	273 (40.1)				
Uninsured	86 (12.6)				
Asthma diagnosis					
Child[e]	351 (51.5)				
Parent	106 (15.6)				
Smokers in household					
Parent	180 (26.4)				
Any smoker	290 (42.5)				
Age (years)					
Child		8.9	8.9	1.9	4.6–12.7
Parent		35.3	34.0	7.8	20.0–73.0
Months at current address		55.5	36.0	59.0	0.0–456.0
Parent CES-D score[f]		14.7	12.0	11.3	0.0–55.0
Main exposure					
Housing stressors (continuous, untransformed total)					
Unweighted		1.5	1.0	1.7	0.0–9.0
Weighted by parent-reported difficulty		4.2	2.0	5.8	0.0–32.0
Count health outcomes[g]					
Exercise intolerance		1.5	0.0	3.2	0.0–14.0
Waking at night		1.4	0.0	2.4	0.0–14.0
Unplanned medical visits		1.3	0.0	2.2	0.0–18.0
School absences		0.4	0.0	1.3	0.0–10.0
Rescue medication use[h]		2.2	0.0	3.8	0.0–14.0
Binary health outcomes[i]					
Fair/poor general health					
Child	111 (16.3)				
Parent	158 (23.2)				

TABLE 2 (continued)

Sociodemographics	Number (percent)	Mean	Med[a]	SD[a]	Min–max[a]
Controllability of asthma	142 (21.6)				
Exercise intolerance	247 (37.0)				
Waking at night	312 (46.0)				
Unplanned medical visits	301 (44.4)				
School absences	122 (18.1)				
Rescue medication use[i]	165 (47.4)				

[a]*Med* median, *SD* standard deviation, *min* minimum value, *max* maximum value

[b]Parent-reported race/ethnicity for child is considered an imperfect proxy for parent; $n=5$ non-black, non-Hispanic, "other" subjects were categorized as white

[c]*TANF* Temporary Assistance for Needy Families

[d]Overcrowding defined as unit occupied by 1.01 persons or more per room excluding bathrooms

[e]Proportion of children with diagnosis is influenced by recruitment and does not reflect prevalence in the target population

[f]*CES-D* Center for Epidemiologic Studies—Depression; possible score ranges from 0–60

[g]Five outcomes included as counts in models; 2-week reference period except for unplanned medical visits (6 months)

[h]Includes diagnosed asthmatics only; $n=351$

[i]General health was a five-response item recoded as fair or poor versus excellent or very good or good (referent); controllability was a four-response item recoded as not at all or somewhat vs. quite or extremely controllable (referent); no reference time periods specified; five other outcomes coded as continuous variables in models; any occurrence vs. none frequencies presented for descriptive purposes

unweighted total housing stressors score, the mean was 1.5 (median=1.0, standard deviation (SD)=1.7). For the total housing stressors score weighted by parent-reported difficulty, the mean was 4.2 (median=2.0; SD=5.8).

Outcomes. More parents reported their health as fair or poor compared to their children's health (23% vs. 16%). Twenty-two percent of parents reported their child's asthma as not at all or somewhat controllable. Prevalence of undesirable child asthma outcomes was high; the proportion experiencing the outcome at least once during the reference period ranged from 18% (school absences) to 47% (rescue medication).

Adjusted Risk and Rate Differences

Adjustment decreased the magnitude of the RD or IRD compared to the crude estimate in most models (Table 3). Adjusted estimates are discussed below and graphically represented in Figs. 1 and 2. Table 3 and the text present RDs and IRDs for the 75th compared to the 25th quartiles of the exposure (higher versus lower housing stressors score). For every outcome except for school absences and rescue medication use, the direction of the effect was as expected; that is, the risk or rate of the unfavorable outcome was larger when associated with a higher housing stressors score than with a lower score.

Binary Outcomes. The RD for the association of general health and housing stressors was larger and slightly more precise for children [RD=6.28 (95% CI 1.22, 11.35)] than for their parents [RD=3.88 (95% CI −1.87, 9.63)]. The RD for

TABLE 3 Crude and adjusted risk and incidence rate differences and 95% confidence intervals for the association of housing stressors with parent and child general health and child respiratory outcomes ($n=682$)

	Crude RD/IRD (95% CI)[a]	Adjusted[b] RD/IRD (95% CI)[a]
Binary outcomes[c]		
RD is the excess cases per 100 persons at risk of the unfavorable outcome associated with increased housing stressors (75th v. 25th percentile of exposure)		
Parent general health	7.51 (1.95, 13.08)	3.88 (−1.87, 9.63)
Child general health	10.20 (5.23, 15.17)	6.28 (1.22, 11.35)
Controllability of asthma	6.93 (1.65, 12.21)	6.19 (0.85, 11.54)
Count outcomes[d]		
IRD is the excess number of days the unfavorable outcome occurred per 2 weeks[e] associated with increased housing stressors (75th v. 25th percentile of exposure)		
Exercise intolerance	0.81 (0.43, 1.20)	0.88 (0.41, 1.35)
Waking at night	0.43 (0.15, 0.72)	0.32 (0.01, 0.63)
Unplanned medical visits[e]	0.44 (0.19, 0.69)	0.30 (0.059, 0.54)
School absences	0.032 (0.12, 0.19)	−0.027 (−0.24, 0.19)
Rescue medication use[f]	0.087 (−0.62, 0.79)	0.092 (−0.61, 0.80)

[a]*RD* risk difference, *IRD* incidence rate difference, *CI* confidence interval

[b]Adjusted for up to eight covariates including: parent race, foreign-born status, marital status, and education; child race/ethnicity; home ownership; housing type; overcrowding and Temporary Assistance for Needy Families, based on 10% change-in-estimate procedure

[c]General health were five-response items recoded as fair or poor versus excellent or very good or good (referent); controllability was a four-response item recoded as not at all or somewhat vs. quite or extremely controllable (referent); no reference time periods specified

[d]Count outcomes were coded as continuous variables; 2-week reference period

[e]Unplanned medical visits was a continuous variable with a 6-month reference period; therefore, IRD is the excess number of days the unfavorable outcome occurred per 6 months

[f]Includes diagnosed asthmatics only; $n=351$

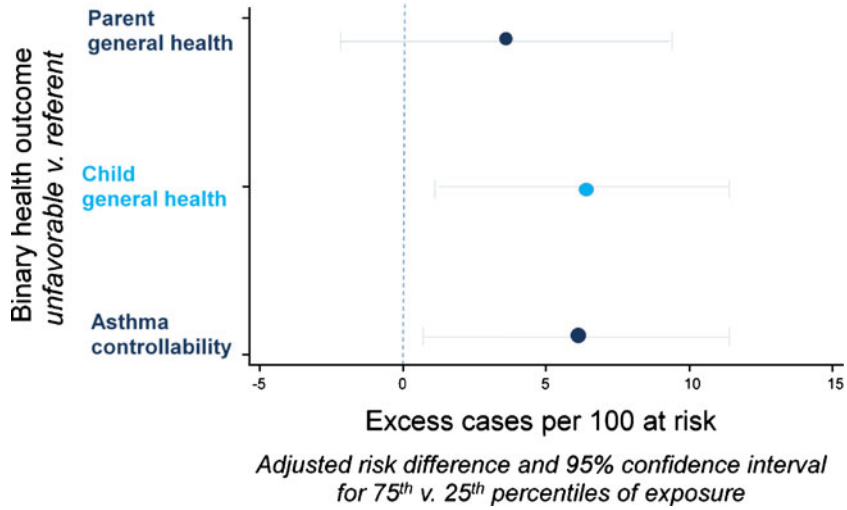

FIGURE 1. Associations of housing stressors and unfavorable binary health outcomes, $n=682$.

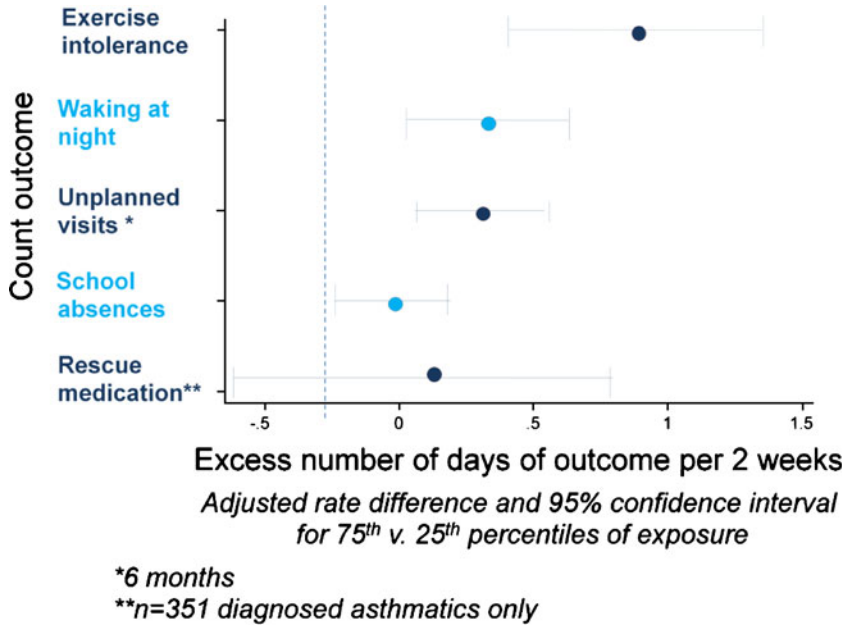

FIGURE 2. Associations of housing stressors and unfavorable count child respiratory outcomes, $n = 682$.

controllability changed little with adjustment and was of moderate magnitude [RD = 6.19 (95% CI 0.85, 11.54)].

Count Outcomes. Exercise intolerance had a moderately strong association with the exposure [IRD = 0.88 (95% CI 0.41, 1.35)]; nearly one extra day of exercise intolerance during a 2-week period was expected for those in the 75th vs. 25th quartiles of housing stressors. Almost one-third extra day of experiencing the outcome was expected for the higher exposure group for waking at night [IRD = 0.32 (95% CI 0.01, 0.63)] and unplanned visits [IRD = 0.30 (95% CI 0.059, 0.54)]. The IRDs for school absences [–0.027 (–0.24, 0.19)] and rescue medication [IRD = 0.092 (95% CI –0.61, 0.80)] were nearly null.

DISCUSSION

Results supported the study's hypothesis; exposure levels reflecting increased number and difficulty with housing stressors were associated with higher risks and rates for most of the undesirable health outcomes examined. This study adds to growing evidence that the social environment contributes to asthma burden in urban areas. Specifically, it furthers the conceptualization of psychological stress as a "social pollutant" that may be "breathed" into the body.[12,16] When stressors are many or difficult to experience and resources for dealing with them are few, as is often the case for low-income, urban families, psychological morbidity may occur and may in turn produce biological and behavioral changes that impact health.

Potential stress pathways by which acute and chronic housing stressors impact general and respiratory health are many. Stressors may compete with a parent's time, resources and motivation to manage a child's asthma and with childrearing in

general. People living in homes with frequent or ongoing stressors may be less likely to have visitors and be denied opportunities for social support and sharing knowledge. Changing household membership and residential mobility may leave adults and children without their usual stress-buffering relationships or expose them to problematic ones. Recurrent, negative thoughts about past events may produce ongoing stress and comorbidities,[60,61] and may influence problem-solving and perceived control.[62] There is evidence that parent stress may produce stress responses in children and cue children to adopt suboptimal coping strategies.[31] More emotional coping style among asthmatics has been associated with lower perceived control, rescue medication overuse, controller medication underuse, and increased emergency care and hospitalization.[63] Stress may lead to health-harming coping strategies such as smoking, overeating, and being sedentary.

The distinction between traditional "environmental" (e.g., allergens and irritants) and psychosocial determinants of disease may be key to understanding disparities, and their policy implications are likely to be different. Just as important, progress has been made over the centuries to improve basic human rights such as safe, secure housing; solutions can be found for those in need of improved social and emotional circumstances. Housing and safety are basic human needs; improvements in these realms are likely to affect a range of physical and mental health outcomes for individuals and potentially for communities. Many stressors are actionable through policies that affect, for example, where, how, and what kind of housing is provided for disadvantaged populations as well as rental management, home ownership, and participation in housing issues.[10,64] While we seek structural solutions to inequities, interventions can address individuals' reactions to stressors. Identifying vulnerable populations allows clinicians and others to recommend stress reduction to mitigate harmful immunologic responses and promote behaviors such as symptom awareness and management, allergen reduction in homes, smoking cessation, and parenting skills.

A strength of this study is the examination of several respiratory outcomes. A challenge for asthma researchers is that outcomes may be influenced by underlying disease severity and individual triggers as well as by disease management, which reflects an individual's attitudes and practices as well as societal factors such as access to care and diagnostic biases. A single composite measure may therefore obscure important relationships. For three of four outcomes coded as counts, the reporting period was 2 weeks and not likely subject to recall bias. Unplanned visits were obtained for a 6-month period, but these rare, major events were probably not difficult to recall. Bias might have resulted from the self-report nature of outcomes and exposures. Though most stressors were objective events, with important exceptions such as trouble with neighbors and landlords and being "bothered" by pests, their difficulty ratings were subjective by design and allowed us to weight the stressors rather than use a simple sum implying equal importance among stressors. Summarizing multiple stressors allows us to address the complex circumstances of low-income, urban families that are hypothesized to impact asthma. Unlike many single-risk factors, this exposure is robust to problems with single variables, for example, influences of secular, geographic, or seasonal trends.

Associations with most outcomes were moderately strong. Exercise intolerance was the count outcome with the largest IRD. The estimate is probably conservative as it is unlikely that all children consistently reported symptoms that occurred while away from their parents. Waking at night is a relatively objective outcome, often used in surveys of asthma control and quality of life. Waking from factors other than

breathing difficulties may have been reported, even though the question specified that only asthma-related night disturbances were of interest. Some outcomes, such as controllability and general health, are subjective, while others, such as school absences and unplanned visits, involve familial and societal influences and do not perfectly reflect disease activity. Misclassification bias may have obscured some associations.

Only rescue medication and school absences had no observed association with housing stressors. Power to detect an association for rescue medication was reduced by omitting undiagnosed children. The association for parent general health was weak, while for child general health it was moderately strong. We might expect that the influence of psychological stress through physiologic mechanisms (directly affecting a parent's health) would be stronger than through behavioral mechanisms (indirectly impacting disease management for their child). Also, parent-reported stressors might be considered proxies for stressors that also produce distress among their children, though we might expect that young children would be less aware or less concerned with housing stressors than adults. The observation of a weaker association for parent than for child general health does not support these theories, but general health is a vague outcome, and no time reference period was specified. Recent research has highlighted the important indirect role of psychosocial factors by demonstrating the association of negative life events and worse asthma-specific quality of life (AQoL) among adults across SES strata.[24] Within the lowest SES stratum, a similar relationship existed across strata of asthma severity, whereas negative life events did not influence the association between worse severity and worse .AQoL across SES strata. A comparison of parent and child asthma outcomes might illuminate contributions of biological and behavioral pathways.

EMM assessment informed the decision not to stratify analyses based on asthma diagnosis. Parents of undiagnosed asthmatic children may have been unfamiliar with symptoms, resulting in outcome misclassification. Nonetheless, interventions to address health-harming housing stressors would likely be targeted to low-income families generally and not only to those with diagnosed asthma, especially since underdiagnosis is a well-documented problem.[65,66] The average effect for all children with respiratory problems was desired since diagnosis is a sociological process based not only on underlying disease but on family and community resources and health attitudes and practices.

The main study's respiratory survey, from which study participants were recruited, captured 90% of the schools' enrollment ($n=12{,}699$), thereby adding to the generalizability of results to low-SES, urban populations. The baseline survey from which the study sample was randomly selected achieved a 64% response rate; therefore, selection bias is a possibility. The study sample was low-income by definition given the recruitment strategy, thereby minimizing confounding by SES. Parent education and TANF were assessed as potential covariates to further control for confounding by SES.

Physical risk factors (e.g., mold and cockroaches), well-documented in the biomedical literature, were not the focus of this study. Biological (e.g., cytokines and cortisol) and behavioral (e.g., smoking and allergen reduction) measures are hypothesized to be on causal pathways and were not included as covariates. Disentangling material and social risk factors for asthma is difficult because they are likely to co-occur as a result of distal determinants such as low-SES and segregation. Furthermore, individual-level mechanisms are influenced by community-level conditions and processes.[10,64,67,68] Risk factors may interact to affect health, as

shown by research on the synergistic effects of air pollution and exposure to violence in relation to asthma among urban children.[69] Future research must address psychological factors over the lifecourse, the relative importance of chronic and acute stressors,[70] the possibility that the asthma phenotype is programmed before birth,[17] and reverse causality, since stress and consequent problems may be caused or aggravated by having asthma or caring for someone with asthma.[70–73] In addition to an ecological perspective, longitudinal data and a multi-level approach are required to understand structural forces that influence the distribution of housing stressors—and as a result, psychological stress.

These findings emphasize the importance of addressing not only documented housing stressors, such as overcrowding and extreme temperature, but social and emotional stressors that may be moderators of the effects of low-SES on respiratory health. It was important to investigate this low-SES population not only because of documented excess asthma but because we expect excess *stressors*. Observing differential outcomes within this low-SES stratum supports the idea that stressors are moderators of the SES-health relationship. If stressors were only mediators, they would not explain additional variance in this sample nor would they explain why some urban areas do not have excess asthma despite similar low-SES and other exposures. In any case, all relevant risk factors must be identified if we are to understand causal mechanisms. Stress may be a crucial contributor to the burden of asthma and other illnesses experienced by urban populations. Sociodemographic and psychological factors are not necessarily easy targets of interventions, but recognizing which stressors are associated with asthma and which groups are most vulnerable to stress is necessary for effective public health and social policies and reduction of health disparities.

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