Published in final edited form as:

J Epidemiol Community Health. 2010 July ; 64(7): 636–642. doi:10.1136/jech.2008.082842.

Social and environmental stressors in the home and childhood asthma

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Abstract

BACKGROUND—Both physical environmental factors and chronic stress may independently increase susceptibility to asthma; however, little is known on how these different risks may interact. We examined the relationship between maternal intimate partner violence (IPV), housing quality and asthma among children in the Fragile Families and Child Wellbeing Study (N=2013).

METHODS—Maternal reports of IPV were obtained after the child's birth and at 12 and 36 months. At the 36 month assessment, interviewers rated indoor housing conditions, regarding housing deterioration (i.e., peeling paint, holes in floor, broken windows) and housing disarray (i.e., dark, cluttered, crowded or noisy house). At the same time, mothers reported on housing hardships (i.e., moving repeatedly, and hardships in keeping house warm). Maternal-report of physician-diagnosed asthma by age 36 months which was active in the past year was the outcome.

RESULTS—Asthma was diagnosed in 10% of the children. In adjusted analysis, an increased odds of asthma was observed in children of mothers experiencing IPV chronically (OR 1.8, 95% CI 1.0, 3.5) and in children experiencing housing disarray (OR 1.5, 95% CI 1.1, 2.0) compared to those not exposed to these risks. In stratified analyses, a greater effect of IPV on asthma was noted among children living in disarrayed or deteriorated housing or among children whose mothers were experiencing housing hardship.

CONCLUSIONS—IPV and housing disarray are associated with increased early childhood asthma. Exposure to cumulative or multiple stressors (i.e. IPV and poor housing quality) may increase children's risk of developing asthma more than a single stressor.

Keywords

intimate partner violence; asthma; housing quality; built environment

INTRODUCTION

Research documenting an association between psychological stress and asthma continues to grow. [1–4] Evidence from animal and human studies strongly suggests that early life adversity shapes stress neurobiology [5], resulting in disturbed regulation of endocrine and

autonomic processes [e.g., hypothalamic-pituitary-adrenal (HPA) axis, sympathetic-adrenal-medullary (SAM) system]. These disturbed patterns of stress regulation are hypothesized to subsequently modulate immune function, increasing susceptibility to asthma and related diseases. [6] Specifically, violence exposure, a chronic psychological stressor has been associated with increased asthma prevalence among children as young as 5 years of age. [7]

Housing quality has been associated with psychological distress and physical health conditions in a number of studies. [2, 8–10] Among New York City residents, Galea et al reported that, living in a neighborhood characterized by poor quality built environment (both internal and external) is associated with greater likelihood of depression even after accounting for individual level socio-demographic factors and neighborhood level income. [8] More specifically, increased noise levels in the home have been associated with higher perceived stress and increased cortisol levels [11], presence of mold, dampness and the inability to keep the house warm have all been associated with both asthma symptoms and distress outcomes [2, 12, 13] and housing instability has been associated with both increased allergen levels such as cockroach and dust and with higher distress symptoms. [9, 14] It's been hypothesized that these associations can be explained by lack of control over one's living environment. When one loses the ability to cope or to take control over one's life or environment, we perceive stress. [9] Thus the home environment may be a direct stressor and in addition may also modify the impact of other stressors on individual health. [15]

We previously reported on the prospective association between intimate partner violence and development of asthma among three year olds in the Fragile Families and Child Wellbeing study. [16] Our goal is to extend that work and consider the impact of other home stressors as well as the joint impact of multiple stressors present in one's household on child asthma. Specifically, we explore the potential for housing quality to impact child asthma by exploring, (1) associations between housing quality and child asthma independent of another key home stressor, i.e., IPV and (2) the potential for housing quality to modify the association between asthma and IPV. We will do this by focusing on three factors defining housing quality (i.e., housing deterioration, housing disarray and housing hardship).

METHODS

Study Population

Analyses were conducted using public-use data available from the Fragile Families and Child Wellbeing Study, a prospective birth cohort study that follows a sample of children from 20 large cities in the United States (US). Non-marital births were over-sampled relative to marital births in a ratio of 3 to 1. The study is a joint effort by Princeton University's Center for Research on Child Wellbeing (CRCW) and Center for Health and Wellbeing, Columbia University's Social Indicators Survey Center, and The National Center for Children and Families (NCCF) (<http://www.fragilefamilies.princeton.edu>). Details on the study design can be found in Reichman et al 2001. [17] In brief, 4789 women were recruited from 75 hospitals during the birth of the child in 20 US cities with populations over 200,000. Random samples of both married and unmarried births were selected until preset quotas were reached based on the percentage of non-marital births in the city that occurred at that hospital in 1996 or 1997. Several exclusions were made: mothers who planned to place the child for adoption, families in which the father of the baby was not living at the time of the birth, those who did not speak either English or Spanish well enough to complete the interview, mothers who were too ill to complete the interview or babies too ill for the mother to complete the interview and those whose baby died before the interview could take place. Among eligible mothers, 82% of those married and 87% of those unmarried agreed to participate. Mothers completed a baseline interview at delivery and participated in follow-up interviews when the children were approximately 12 and 36 months of age. At the time of

the 36-month in-home assessment, 3288 families remained in the study with 2119 completing the study at home. [17] Those who did not participate in the 36 month in home assessment differed significantly from those who responded based on race/ethnicity. In addition, 106 children were missing data on other covariates, leaving 2013 children for the current analyses.

Housing quality

Housing items were adapted from previous work regarding housing conditions demonstrating links to psychological distress [9] and asthma [18][19]. At the 36 month in-home follow-up trained interviewers conducted an assessment of indoor housing conditions. A total of 11 items regarding the integrity of the physical home environment were completed including whether the home unit contained broken windows or cracked windowpanes, open cracks or holes in walls or ceiling, holes in floor, broken plaster or peeling paint and whether any of the following hazardous conditions were observed: frayed electrical wires, presence of mice or rats, broken glass, falling plaster, broken stairs, peeling paint, and other hazards. Housing deterioration was characterized as responding yes to one or more of the above questions. In addition six interviewer-reported items regarding the organization of the home environment (i.e., is inside of home dark or crowded, cluttered or dirty/not reasonably cleaned, is house overly noisy-from noise in the house or from noise coming from the outside of the house) made up a second factor on housing disarray characterized as responding yes to one or more of the above items. Lastly mothers were asked three questions regarding housing hardship. Mothers were asked whether in the past 12 months their home had been uncomfortably cold or whether they had no running water for a period of 48 hours or more and whether they had moved two or more times since the child's first birthday. Mothers who responded yes to one or more of the above questions were categorized as experiencing housing hardship.

Confirmatory factor analysis using weighted least-squares method, the recommended method for models with categorical variables was used to test the three housing factors outlined above. The analyses confirmed three factors: one factor included the 11 items on housing deterioration, a second factor included the six items on housing disarray, and a third factor included the three items on housing hardship. Model fit was assessed using the comparative fit index (CFI) 0.97 (> 0.90 acceptable, > 0.95 excellent) and the root mean square error approximation (RMSEA) 0.03 (< 0.08 acceptable, < 0.05 excellent) indicating the data fit well by the assumed factors. All factor analyses were conducted in MPLUS. [20]

Intimate Partner Violence Assessment

Maternal IPV was assessed at baseline and at the 12- and 36-month assessments, using previously validated questions which varied slightly between baseline and follow-up assessments. [21, 22] At baseline, mothers were asked to think about their relationship with the baby's father and asked how often does "he hit or slap you when he is angry?" Mothers who responded, 'often' or 'sometimes' as opposed to 'never' were categorized as experiencing baseline IPV. At 12 and 36 months, mothers were asked to think about their relationship with the baby's father or current partner and were then asked: "How often does he slap or kick you?", "How often does he hit you with a fist or object that could hurt you?", and "Were you ever cut or bruised or seriously hurt in a fight with the baby's father or current partner?". Mothers who responded 'often' or 'sometimes' as opposed to 'never' to either of the first two questions or who responded 'yes' to the third question were categorized as experiencing IPV for the relevant follow-up period. Physical IPV was characterized as: (a) never experiencing IPV (b) occurring prior to 12 months only (endorsed at baseline and/or 12-month follow-up), (c) occurring between 12 and 36 months only

(endorsed at 36 months only), or (d) occurring both prior to 12 months and also between 12 and 36 months (chronic exposure).

Asthma Outcome

During the 36 month follow-up interview, mothers were asked whether a physician had diagnosed the child with asthma and whether the child had an asthma attack in the past 12 months, a standard approach with demonstrated reliability and validity. Parent-reported physician-diagnosed asthma is associated with more objective outcomes (e.g., airway hyperresponsiveness) and more severe disease. [23] Moreover, this definition is used by the National Center for Health Statistics in surveys of the US population to facilitate generalizability. [24]

Covariates

The baseline and follow-up questionnaires ascertained information on sociodemographic factors as well as maternal smoking and birth weight of the child. Racial/ethnic minorities as well as those of lower socioeconomic status (SES) experience higher rates of family violence as well as higher rates of asthma [25] than their white, higher SES counterparts. [26–28] Analyses were adjusted for maternal race/ethnicity, maternal education level and receipt of public assistance in the past year. Women who experience IPV are also more likely to have lower birth weight babies [29], a risk factor that has also been linked with increased childhood asthma risk. [30, 31] Birth weight was categorized as less than 2.5 kg compared to 2.5 kg and above. Furthermore, tobacco exposure was included as another potential confounder. Women who experience interpersonal violence and other stressors are also more likely to smoke [32] and environmental tobacco smoke exposure has been associated with early wheeze and asthma [33]. Mothers reported whether they smoked during pregnancy at the baseline assessment. During follow-up assessments, mothers were asked to report on their current smoking status and whether anyone in the household smoked. Child tobacco smoke exposure was categorized as follows: (a) no exposure; (b) postnatal tobacco smoke exposure only; and (c) both *in utero* and postnatal tobacco smoke exposure. There were few children with *in utero* tobacco exposure but no postnatal exposure; hence they were included in the *in utero* and postnatal tobacco smoke exposure category. Lastly we adjusted for maternal use of asthma medications as an indicator of maternal history of asthma.

Statistical Analyses

Summary measures of continuous covariates are reported as mean and standard deviation (SD) unless otherwise noted. Logistic regression analyses were conducted to estimate the association between housing quality and asthma while adjusting for IPV and potential confounders (child's gender, age, race/ethnicity, low birth weight, maternal education, marital status, public assistance, maternal use of asthma medications, and tobacco exposure). First, regression models including timing of occurrence of maternal IPV and housing quality indicators were examined. Second, three separate logistic regression models were examined regressing maternal IPV on child's asthma diagnosis, stratified by the three housing quality indicators: housing deterioration (model 1), housing disarray (model 2), and housing hardship (model 3). All analyses were conducted in SAS version 9.0 (SAS Institute, Cary, NC).

RESULTS

Among the 2013 children available for analyses, 52% are boys, 54% are Black, 27% are Hispanic, 10% had been diagnosed with asthma which was active in the past 12 months, and 17% lived in a household with maternal IPV at some time during follow-up (Table 1).

Fifteen percent of children lived in deteriorated housing, 35% lived in disarrayed housing and 24% were experiencing housing hardship. Housing deterioration, disarray and hardship had a low correlation, Pearson coefficients ranged from 0.05–0.38.

In bivariate analyses (results not shown), IPV was significantly associated with housing hardship (OR: 2.0, 95%CI: 1.6, 2.6). IPV was also positively associated with housing deterioration (OR: 1.2, 95%CI: 0.9, 2.7) and with housing disarray (OR: 1.2, 95%CI: 0.9, 1.5) albeit this was not statistically significant. Housing disarray was statistically significantly associated with asthma (Table 2).

In adjusted analysis, children of mothers experiencing IPV in only one time period, that is, prior to 12 months or at 36 months only, did not have a significant increased odds of asthma compared to children whose mothers reported never being exposed (OR: 0.8, 95%CI: 0.4, 1.4 and OR: 1.2, 95%CI: 0.7, 2.3). Those exposed to IPV during both time points (chronic exposure) had an almost 2-fold odds (OR: 1.8, 95%CI: 1.0, 3.5) of having an asthma diagnosis (Table 3). Home disarray was associated with asthma (OR: 1.5, 95%CI: 1.1, 2.0) independently of IPV (Table 3). An additional multilevel model further accounting for clustering at the city level did not change the results (data not shown).

In stratified analyses, children of mothers experiencing IPV chronically and whose homes were deemed to be deteriorated were at increased odds of asthma (OR: 4.6 95%CI: 1.0, 21.8) compared to those not exposed to IPV with home deterioration (Figure 1). No significant associations were noted among children of mothers experiencing IPV chronically but no home deterioration (OR: 1.6 95%CI: 0.8, 3.3) compared to those not exposed to IPV with no home deterioration. Children of mothers experiencing IPV chronically and with home disarray were at increased odds of asthma (OR: 2.3 95%CI: 1.0, 5.3) compared to those not exposed to IPV with no home disarray (Figure 2). Children of mothers experiencing IPV chronically with no home disarray did not have a statistically significant odds of asthma (OR: 1.4 95%CI: 0.5, 4.0) compared to those not exposed to IPV with no home disarray.

Children of mothers experiencing IPV chronically and experiencing housing hardship were at increased odds of asthma (OR: 4.4 95%CI: 1.6, 12.3) compared to those not exposed to IPV also experiencing housing hardship (Figure 3). Children of mothers experiencing IPV chronically with no housing hardship did not have a statistically significant increased odds of asthma (OR: 1.2 95%CI: 0.5, 3.0) compared to those not exposed to IPV and not experiencing housing hardship.

DISCUSSION

As others have shown, we found chronic exposure to IPV to be associated with childhood asthma in this prospective cohort. More notably, we found an indicator of housing quality, previously shown to be associated with psychological distress, to be associated with childhood asthma, independently of IPV and other confounders. We furthermore found suggestive evidence that other aspects of housing quality (deterioration, hardship and disarray) can potentially exacerbate the effect of IPV, a chronic stressor, on childhood asthma.

Our findings can be interpreted two different ways. One, housing quality is a marker for other environmental factors known to be associated with asthma. Numerous studies have shown associations between characteristics of the physical environment, such as dust and cockroach allergen, pollutants from traffic sources and indoor environmental factors such as mold, and childhood asthma. [25] Rauh et al has previously shown markers of housing

deterioration (i.e. holes in floor, broken plaster) and housing instability to be predictive of cockroach allergen in the home of asthmatics residing in New York City. [14]

As previously noted, social factors, such as chronic stress resulting from IPV, have been implicated in the development of asthma. Traditionally, the relationship between social and environmental factors has been treated as one of confounding, with each measure being treated as an independent risk factor. [34] Biologically, however, the effect of joint exposure may not be so simple. Both environmental exposures (i.e., traffic patterns, indoor and outdoor air quality, home allergens, environmental tobacco smoke, use of pesticides) and social stressors (i.e., racial segregation, employment rate, violence and crime, crowding, family relationships) are common among disadvantaged populations. [18] Within these populations, there appear to be individual or host differences in the body's response to similar levels and durations of stress. [35] If stress plays a role in complex disorders, such as asthma development, then a possible mechanistic role is that chronic stress modifies the response to environmental toxins. Animal studies have shown that stress can modify the body's response to environmental toxins. [36] In humans, only a few studies have examined how social factors can modify the effects of environmental pollutants. In a study by Clougherty et al, exposure to violence was shown to modify the relationship between traffic related air pollution and childhood asthma. An association between nitrogen dioxide, a marker of traffic pollution, and asthma was only noted among children with high levels of violence exposure. [37] In contrast, while Chen et al also noted an interaction between traffic pollution and asthma inflammatory markers among children, they noted higher inflammatory profiles among those with high chronic stress and low pollution levels. [38]

A second interpretation is that housing quality is a direct source of stress and thus a contributor to the stress pathway, and as such, influences the risk of asthma. When environmental demands are found to be taxing or threatening, and coping resources are viewed to be inadequate, individuals perceive themselves as being under stress. [39] This perception is presumed to result in negative emotional states. Behavioral and emotional changes that follow the effort to adapt to stressors are accompanied by complex patterns of neuroendocrine and immunologic alterations. [40–42] Psychological stress has been associated with the activation of the SAM system and the HPA axis. [43] Negative emotional responses disturb the regulation of the HPA axis and the SAM systems; that is, in the face of stress, physiological systems may operate at higher or lower levels than during normal homeostasis. It is the disturbed balance of these systems that is relevant to disease. Immune, metabolic, and neural defensive biological responses important for the short-term response to stress may produce long term damage if not checked and eventually terminated. [44] Poor housing conditions can be perceived as a lack of control over one's environment thereby causing stress. In a study of 8 European large cities Shenassa et al noted that perception of control over one's housing environment partially mediated the association between a damp or moldy residence and depression. [45] Among children, noise, crowded housing conditions and poor housing quality have all been associated with increased psychological distress. [46] Cumulative stress exposure, that is stress from both social and environmental factors, may have more of an impact on asthma development than a single stressor. Evans et al reported that the association between childhood poverty and stress dysregulation could partly be explained by cumulative risk exposure defined as exposure to multiple physical (e.g. substandard housing) and social (e.g. family turmoil) risk factors. [47] Multiple chronic stressors have also been shown to increase the risk of asthma attacks in response to negative life events. [48]

While these data have allowed us to explore prospective relationships between maternal IPV, housing quality and childhood asthma, there are limitations worth mentioning. While we adjust for a number of potential confounders no data was available on maternal history of

asthma, however we adjust for maternal use of asthma medication which did not have an effect on the associations previously noted. As is typical with longitudinal studies, there was a reduction in the sample available from the original cohort over time. While there was a difference based on race/ethnicity between those who completed the 36-month assessment and those who did not, there were no differences based, on maternal education, smoking status, low birthweight, or maternal IPV at baseline. It is also possible that differences in access to care or health care utilization may vary in lower socioeconomic groups based on race/ethnicity and may be a factor related to misclassification of receiving a diagnosis of asthma [49] this seems unlikely to explain our findings. Mothers experiencing IPV may have more difficulty scheduling and keeping routine health care visits for their child in which case we would expect to see lower rates of diagnosis in this group, and thus unlikely to account for the observed associations. Furthermore, only 4% of the children were reported as not having any regular checkups (doctor or nurse) in the past 12 months indicating similar access to care and utilization for the majority of the cohort. A lack of an association between housing deterioration and asthma could be potentially due to the fact that while interviewers were trained to assess the indoor home environment, the assessment was only done at one point in time at the end of a questionnaire assessment. While this is an objective measure of the indoor environment its possible the interviewers missed certain features of the home due to the short duration of the assessment. However, this potential misclassification of exposure is likely non-differential with respect to the outcome, and thus unlikely to account for the observed associations noted for other housing conditions. Similarly a lack of an association between housing hardship and asthma could be due to a potential underreport of hardship conditions by the mother. In addition, as both asthma and housing quality were assessed at the 36 month follow-up we are limited by the cross-sectional nature of the data. Because a number of families moved during study follow-up we conducted a sensitivity analysis, restricting our sample to only families who had not moved since the child's first birthday (i.e., in the past two years). The effect of housing disarray on asthma (1.2 95%CI 0.9–1.7) is qualitatively similar to that previously noted in the entire sample, furthermore the effect of chronic IPV exposure (1.8 95%CI 0.9, 4.0) on asthma is qualitatively similar as that seen for the entire sample. Finally, by characterizing asthma as, physician-diagnosed asthma by three years of age that has been active in the past year, we may be reducing the potential for misclassification of what is truly asthma. It is also possible that children previously diagnosed with asthma without an attack in the past year may represent children with less severe underlying disease and the effect of chronic underlying stress may influence disease severity. No other measures of asthma symptoms (e.g., nocturnal cough, wheezing, medications needed and other healthcare utilization indicators) were collected to assess whether housing conditions and IPV are related to asthma severity..

In this study IPV and poor housing quality were associated with early childhood asthma. Our results also suggest an interactive effect between IPV and housing quality, in that IPV was associated with asthma only among children who were living in poor housing quality environments. Intervention and prevention efforts that target both the social and physical environment in the homes of children may have better results in remedying asthma disparities among children.

Acknowledgments

Funding: Support was provided by the Robert Wood Johnson Foundation New Connections Initiative Award and NIH T32MH073122-04.

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Figure 1. Odds of child asthma by maternal intimate partner violence stratified by housing deterioration

Adjusted for child's age, gender, race/ethnicity, maternal education, marital status, public assistance, mother's use of asthma medication, tobacco exposure and low birth weight

Figure 2. Odds of child asthma by maternal intimate partner violence stratified by housing disarray

Adjusted for child's age, gender, race/ethnicity, maternal education, marital status, public assistance, mother's use of asthma medication, tobacco exposure and low birth weight

Figure 3. Odds of child asthma by maternal intimate partner violence stratified by housing hardship

Adjusted for child's age, gender, race/ethnicity, maternal education, marital status, public assistance, mother's use of asthma medication, tobacco exposure and low birth weight

Table 1

Demographics, Fragile Families and Child Wellbeing Study (N=2013)

Demographics	N	%	Mean	SD
Child's age (Months)			38.2	2.8
Child's Gender				
Male	1051	52.2		
Female	962	47.8		
Race/Ethnicity				
White	378	18.8		
Black	1090	54.2		
Hispanic/Other	545	27.1		
Mother's Highest Education Level Attained				
Some College	803	39.9		
High School Graduate or Less than High School	1210	60.1		
Mother's Marital Status				
Single	1441	71.6		
Married	572	28.4		
Income from Public Assistance/Welfare/Food Stamps	832	41.3		
Low Birth Weight (Less than 2.5 kg)	192	9.5		
Mother's use of Asthma Medication	53	2.6		
Environmental Factors				
Tobacco Exposure				
No in-utero or postnatal tobacco exposure	1139	56.6		
In utero and Post-natal Tobacco Smoke Exposure	405	20.1		
Post-natal Second Hand Smoke only	469	23.3		
Violence Exposure				
Intimate Partner Violence (IPV)¹				
IPV prior to 12 months	170	8.5		
IPV between 12 and 36 months	108	5.4		
IPV chronic	72	3.6		
Asthma				
Asthma diagnosis (active in the past 12 months)	197	9.8		
Housing Quality				
Housing Deterioration (one or more positive items)	300	14.9		
Housing Disarray (one or more positive items)	713	35.4		
Housing Hardship (one or more positive items)	485	24.1		

¹ Intimate Partner Violence defined as physical abuse as reported by the mother

Table 2

Housing Quality and Odds of Asthma diagnosis among 36 month old children (N=2013)

	N	%	Asthma OR 95% CI Unadjusted	Asthma OR 95% CI Adjusted
Housing Deterioration (one or more of the following)			1.0 (0.7–1.5)	0.9 (0.6–1.4)
Any of the following conditions: Frayed Electrical Wires, Mice or Rats, Broken Glass, Falling Plaster, Broken Stairs, Peeling Paint, Other Hazards	77	4.8		
Are there any broken windows or cracked windowpanes?	111	5.9		
Does the housing unit have open cracks or holes in walls or ceiling?	140	7.3		
Does the housing unit contain holes in floor?	36	1.9		
Does the housing unit contain broken plaster or peeling paint?	173	9.0		
Housing Disarray (one or more of the following)			1.6 (1.2–2.2)	1.4 (1.0, 1.9)
Is inside of home dark?	482	24.4		
Is inside of home crowded?	392	19.8		
Are all visible rooms noticeably cluttered?	442	22.6		
Are all visible rooms dirty or not reasonably cleaned?	421	21.4		
Is house overly noisy- from noise in the house?	219	13.0		
Is house overly noisy-from noise outside the house?	93	5.5		
Housing Hardship (one or more of the following)			1.1 (0.8–1.6)	1.1 (0.8, 1.6)
In the past 12 months has your home been uncomfortably cold for a period of 48 hours or more?	173	8.6		
In the past 12 months, did you ever have no running water for a period of 48 hours or more?	55	2.7		
Moving two or more times since the child's first birthday	309	15.4		

Adjusted for child's age, gender, race/ethnicity, maternal education, marital status, public assistance, mother's use of asthma medication, tobacco exposure and low birth weight

Table 3

Odds of asthma by intimate partner violence at 12 months of age and 36 months of age and housing factors

	Active Asthma Unadjusted	Active Asthma Adjusted	Active Asthma Adjusted plus Housing factors
	OR (95%CI)	OR (95%CI)	OR (95%CI)
IPV (prior to 12 months)	0.9 (0.5–1.6)	0.8 (0.4–1.4)	0.8 (0.4–1.4)
IPV (between 12 and 36 months)	1.4 (0.8–2.6)	1.3 (0.7–2.3)	1.2 (0.7–2.3)
IPV (chronic)	2.1 (1.2–4.0)	2.0 (1.3–3.9)	1.8 (1.0–3.5)
Housing Deterioration	--	--	0.8 (0.5–1.2)
Housing Disarray	--	--	1.5 (1.1–2.0)
Housing Hardship	--	--	1.1 (0.8–1.6)

Adjusted for child's age, gender, race/ethnicity, maternal education, marital status, public assistance, mother's use of asthma medication, tobacco exposure and low birth weight