AN ECOLOGICAL EXAMINATION OF PSYCHOLOGICAL STRESS AND ASTHMA AMONG LOW-INCOME FAMILIES IN CHICAGO: FAMILY, HOUSING AND NEIGHBORHOOD DETERMINANTS

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ABSTRACT

KELLY ANN QUINN: An Ecological Examination of Psychological Stress and Asthma among Low-income Families in Chicago: Family, Housing and Neighborhood Determinants (Under the direction of Jay S. Kaufman)

Objective. Asthma disproportionately affects non-white, urban, low- socioeconomic status populations, but trends and inequities are not well-explained by known risk factors. We hypothesized that disadvantaged populations experience housing and neighborhood stressors that produce psychological stress and impact child respiratory health through biologic and behavioral pathways. **Methods**. We examined relationships between material and social stressors and six child respiratory outcomes (measured as events/two weeks, except unplanned visits (six months) and controllability (time unspecified) and parent and child general health (GH) (time unspecified) in two cross-sectional studies using data from low-income, racially/ethnically heterogeneous Chicago families of children with respiratory problems. Adjusted binomial and negative binomial regression models produced risk differences (RDs), incidence rate differences (IRDs), and 95% confidence intervals (CIs). Housing Stressors (HS) was a continuous exposure representing number of stressors experienced in six months by 682 parents, weighted by parent-reported difficulty. Principal components analysis yielded two exposures representing 319 parents' perceptions of neighborhood collective efficacy (CE) and physical/social order (order); three-level exposures yielded low and mid vs. high (most favorable) contrasts. **Results.** HS was associated with nearly one extra day/two weeks of

exercise intolerance [IRD=0.88 (95%CI: 0.41, 1.35)], nearly one-third extra day/two weeks of waking at night [IRD=0.32 (95%CI: 0.01, 0.63)] and nearly one-third extra day/six months of unplanned visits [IRD=0.30 (95%CI: 0.06, 0.54)]. Controllability [RD=6.19 (95%CI: 0.85, 11.54)] and child GH [RD=6.28 (95%CI: 1.22, 11.35)] were moderately associated with HS; parent GH's association was weak. More negative neighborhood perceptions tended to be associated with poor outcomes, though results differed by exposure. Only waking at night was strongly associated with CE [$RD_{low\ v.\ high}$=16.7 (95%CI: 2.8, 30.6)] and order [$RD_{low\ v.\ high}$=22.2 (95%CI: 8.6, 35.8)]. Exercise intolerance [$RD_{low\ v.\ high}$=15.8 (95%CI: 2.1, 29.5)] and controllability [$RD_{mid\ v.\ high}$=12.0 (95%CI: 1.8, 22.3)] were moderately associated with order. Parent GH was strongly associated with CE [$RD_{low\ v.\ high}$ =20.8 (95%CI: 7.8, 33.9)]; child GH's association was weak. **Conclusions.** Findings add to the conceptualization of stress as a "social pollutant" that becomes "biologically embedded." Interventions must address physical and social dimensions of residence and mitigate individual-level stress while structural solutions to inequities are sought.

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LIST OF ABBREVIATIONS

AIC	Akaike Information Criterion
BIC	Bayesian Information Criterion
BPAS+	Brief Pediatric Asthma Screen Plus
CE	Collective Efficacy
CES-D	Center for Epidemiologic Studies-Depression
CHQ	Child Health Questionnaire
CI	Confidence Interval
CRISYS-R	Crisis in Family Systems-Revised
DAG	Directed Acyclic Graph
e.g.	For example
EMM	Effect Measure Modification
HS	Housing Stressors
i.e.	That is
IgE	Immunoglobulin E
IRD	Incidence Rate Difference
NIEHS	National Institute of Environmental Health Sciences
N	Number
PCA	Principal Components Analysis
PHDCN	Project on Human Development in Chicago Neighborhoods
RD	Risk Difference
SD	Standard Deviation
SES	Socioeconomic Status
SF-12	Short Form-12

CHAPTER 1
INTRODUCTION

> *"The profound question, initially raised in the context of cancer, is what gets asthma? Is it the cell, gene, organ, individual, household, population subgroup, or community? Indeed, the potential answers are rarely exclusive."*[1]

Though asthma prevalence among U.S. children seems to have recently plateaued at historically high levels,[2] increasing prevalence and morbidity worldwide across several decades has been documented,[3] and asthma's disproportionate burden on certain populations has been clarified to a great extent. It is one of the most common chronic diseases of childhood in the U.S.[2] Among U.S. children, non-whites living in urban areas and impoverished groups are most at risk of developing asthma, having more severe asthma, and having higher rates of hospitalization and morbidity.[2, 4, 5] Chicago's asthma mortality and hospitalization rates are among the highest in the nation.[6, 7] Underdiagnosis among children and suboptimal care among Chicagoans have been documented.[8-10] Within Chicago, prevalence, morbidity, and mortality rates vary by neighborhood and are highest in neighborhoods with the lowest socioeconomic status (SES).[6, 7, 9, 11-13] The association of SES and asthma prevalence, morbidity and mortality is graded in the U.S., though SES does not fully explain dramatic disparities by race and ethnicity.[1, 14]

Asthma epidemiology has become complex, and it is becoming clear that the epidemic is not explained by known risk factors, leading Wright and Subramanian to posit, "Is it simply asthma disparities or is it social disparities in asthma?"[1] They 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.[1]

Psychological stress has been conceptualized as a "social pollutant that when 'breathed into the body' may disrupt biological systems through inflammatory processes."[15, 16] Acute and chronic stressors are believed to have psychological effects that in turn influence psychologic and physiologic functioning as well as behavior.[17] Studies link psychological stress to asthma, including onset of disease, precedent phenotypes, and disease exacerbation, through hypothesized "dysregulated immunity" mechanisms.[15, 16, 18-26] Psychological stress experienced by children or their parents may also have indirect effects on asthma by causing health-compromising behaviors and co-morbidities that compromise disease management.[27-38]

Low-SES and minority populations often have increased exposure to environmental and psychosocial stressors.[39-42] and may be more strongly affected by stressors due to already-compromised psychological health, social supports, coping resources, and individuals' sense of lack of control over their lives.[40, 43-48] Just as material exposures act on biology, social environments may "get under the skin"[49] and become "biologically embedded"[50] to influence health.[1, 16] The housing and health relationship has long been acknowledged, not surprising given that water, warmth, air to breathe, shelter, and safety are fundamental human needs.[51] Children in the U.S. spend as much as 80-90% of their time indoors,[52] and urban-dwellers may experience increased risk of health-compromising indoor exposures and behaviors because of concerns about safety outdoors. Concentration of poverty may lead to deterioration of housing and physical conditions of neighborhoods as well as deterioration of social institutions and networks.

Despite widespread recognition that residence is important to health, much work is needed to expand the relevant dimensions of housing and neighborhoods and elucidate

causal mechanisms involving asthma outcomes, including psychological stress mechanisms. Not all urban communities have excess asthma morbidity though they may share low-SES and physical environmental exposures with high risk urban areas.[1, 6, 16, 53] The associations between many material housing and neighborhood factors and respiratory conditions have been documented. The pathways by which stress influences asthma and atopy are increasingly being clarified, but the ongoing burden of asthma on inner-city populations demands more thoughtful investigation of determinants of risk.

This research, cross-sectional in design and limited to Chicago families, 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 psychological stress pathways. Good interventions have multifaceted effects and address a breadth of interrelated factors. We used unique data not collected in large-scale health studies to create multidimensional measures of policy-relevant stressors, robust to problems with single variables, for example, influences of secular, geographic or seasonal trends. We used parent-rated difficulty to create a weighted measure of housing stressors and principal components analysis to summarize parents' perceptions of their neighborhoods for a low-income, urban, racially/ethnically heterogeneous sample of parents of young children. These exposure variables were used to investigate the conceptualization of psychological stress as a "social pollutant" that affects respiratory health among children.

A dataset collected in 2001-2005 for a National Institute of Environmental Health Sciences (NIEHS)-funded longitudinal cohort study of racial/ethnic disparities in childhood asthma among low-income families in Chicago provided data for two cross-sectional analyses of six parent-reported child asthma outcomes. I hypothesized that mechanisms

involving psychological stress potentially influence a variety of health outcomes; therefore, I also investigated parent and child general health. Research aims were divided into two groups by unique exposure variables and are discussed throughout this dissertation as follows:

Specific Aim 1*:* To investigate the association of housing stressors and child respiratory health and parent and child general health among low-income families in Chicago.

Hypothesis*:* Material and social aspects of housing including security, mobility, comfort, safety, finances, dynamic household membership, and relationships with neighbors and landlords cause psychological stress, which influences biological and behavioral pathways to health for parents and children. Increased exposure to housing stressors, reflecting the number of stressors and the difficulty of the experience(s), as reported by parents, is associated with increased risk of poor health outcomes for parents and their children.

Rationale: Low-SES populations have increased exposure to environmental and psychosocial stressors and may be more strongly affected by stressors. Acute and chronic housing stressors may cause psychological stress, which in turn influences respiratory and general health directly through immunologic pathways and indirectly by causing health-compromising behaviors and co-morbidities that compromise disease management for parents and their children. Identifying causes of stress among disadvantaged populations may illuminate determinants of health inequities in general and of asthma's social patterning in particular.

Specific Aim 2: To investigate the association of parents' perceptions of neighborhood stressors and child respiratory health and parent and child general health among low-income families in Chicago.

Hypothesis: Numerous aspects of neighborhoods, such as existence of physical hazards and lack of cohesion and collaboration among residents, cause psychological stress, which

in turn influences biological and behavioral pathways to health for parents and children. Poor parent perceptions of their neighborhoods are associated with increased risk of poor health outcomes for parents and their children.

Rationale: Low-SES populations have increased exposure to environmental and psychosocial stressors and may be more strongly affected by stressors. Parents' perceptions of their neighborhoods cause psychological stress, which in turn influences respiratory and general health directly through immunologic pathways and indirectly by causing health-compromising behaviors and co-morbidities that compromise disease management for parents and their children. Identifying causes of stress among disadvantaged populations may illuminate determinants of health inequities in general and of asthma's social patterning in particular.

Chapter 2 provides a critical review of risk factors for asthma and asthma disparities, in particular, social determinants and psychological stress. Chapter 3 details the research design and methodological strategies. Chapters 4 and 5, written as manuscripts for peer-reviewed publications, present the results of analyses addressing specific aims 1 and 2, respectively. Chapter 6 synthesizes research findings and discusses methodological issues, Public Health implications, and future research priorities.

CHAPTER 2
BACKGROUND AND SIGNIFICANCE

> *"Experts agree: the blight of poverty housing reaches beyond rotting roofs and insufficient sanitation systems. It casts low-income families into an unforgiving cycle of physical and emotional duress, compromising their health, academic achievement and sense of security. While adequate shelter is not the cure for every poverty ill, it does offer a haven for emotional and physical rest, and the stability found therein empowers families in their pursuit of a better quality of life. The importance of a decent place to live cannot be overstated, for with it come stability and promise, family unity, hope and a foundation from which individuals reach their full potential."*[54]

The Asthma Epidemic

Though asthma prevalence among U.S. children seems to have recently plateaued at historically high levels,[2] increasing prevalence and morbidity worldwide for the past three decades has been documented, and asthma's disproportionate burden on certain populations has been clarified to a great extent. It is one of the most common chronic diseases of childhood in the U.S.[2] Among children in the U.S., non-whites living in urban areas and impoverished groups are most at risk of developing asthma, having more severe asthma, and having higher rates of hospitalization and morbidity.[2, 4, 5] African American compared to white children with asthma have significantly greater emergency department and hospitalization rates and lower rates of ambulatory care visits.[2, 4] Among Medicaid patients, blacks receive fewer preventive services than whites.[55] Asthma mortality rose in recent decades despite improved medications and treatment plans; low-income minorities in urban settings experienced the largest death rate increases.[1, 2, 4-6, 13] There is variation within and across U.S. cities that is not fully explained by SES.[1, 6, 16] Furthermore, though

the association of SES and asthma prevalence, morbidity and mortality is graded in the U.S., SES does not fully explain disparities by race and ethnicity.[1, 14]

Genetic theories about race differences do not explain inequities in asthma since increasing incidence and disparities have occurred over only a few decades. Gene-environment interactions, however, have become a research priority. Other proposed explanations for asthma disparities include: limited implementation of asthma guidelines; lack of emphasis on asthma control; failure to evaluate effectiveness of evidence-based therapies on community/population indicators; the obesity epidemic in the U.S.; environmental factors; and poor access to quality care.[56] As studying asthma determinants becomes increasingly complex, it is becoming clear that the epidemic is not explained by known risk factors, leading Wright and Subramanian to posit, "Is it simply asthma disparities or is it social disparities in asthma?"[1] They 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.[1]

Chicago is one of two U.S. cities with the highest asthma mortality and hospitalization rates, and within Chicago, prevalence, morbidity, and mortality rates vary by neighborhood and are highest in neighborhoods with the lowest SES.[6, 7, 9, 11-13] Hospitalization rates in Chicago twice as high as in suburban Chicago and in the U.S. overall have been documented.[7] Studies of Chicago school children have shown that underdiagnosis is a substantial problem. Including respiratory symptoms consistent with asthma increases the total asthma burden in some schools to more than one-quarter of students.[8, 57] These studies also confirm dramatic racial and ethnic disparities: a total respiratory burden of more than one in three children was found for blacks and Puerto-

Ricans. Non-Hispanic whites (23%) and Hispanics of Mexican origin (22%) have lower but alarmingly high total potential asthma burdens.[8] For individuals with asthma, suboptimal disease management has been documented although interventions to improve asthma control have been a focus.[10, 13] Despite a decade of extraordinary efforts to increase asthma equity in Chicago, progress has been modest, and huge challenges remain for Chicagoans.[13]

Asthma Clinical Characteristics and Etiology

Asthma is a chronic airways disease characterized by bronchial hyperresponsiveness and a link between hyperresponsiveness and airways inflammation involving many cell types.[58] This may lead to recurrent wheeze, cough, breathlessness and chest tightness, most often reversible either spontaneously or with treatment. Multiple mechanisms involving environmental and genetic factors are thought to influence asthma initiation and exacerbation. Responses to exposures as well as to treatments may be modified by racial/ethnic variability in genetic polymorphisms.[59] Genetic theories about race differences do not explain trends or inequities in asthma, however, since increasing incidence and disparities have occurred over only a few decades. Also, genes are generally more likely to determine susceptibility than development of asthma.[1] Gene-environment interactions, however, may be crucial to understanding asthma risk and severity.

Hypersensitivity to aeroallergens has been associated with asthma in cross-sectional and prospective studies.[1] In the U.S. more than 80% of asthmatic children are also atopic, that is, sensitive to one or more allergens, though many children with atopy do not have asthma. Most allergy in asthmatic children is immunoglobulin E (IgE)-mediated, in which specific IgE antibody responses occur, while occupational asthma is often IgE-independent.[58] Half of asthmatics have multiple (three or more) sensitivities.[60, 61] Others

have non-allergic asthma, with symptoms caused by, for example, exercise, weather, and emotions, though inflammation and hyperresponsiveness are also the causes of symptoms in these individuals.

Most asthma is diagnosed in childhood, though diagnosis may be difficult in small children, who often have respiratory symptoms from colds, infections and allergies, and in the elderly, who may have symptoms due to heart disease and chronic obstructive pulmonary disease. Some children may outgrow asthma though it may recur in adulthood.[58] Diagnosis occurs later for females than males.[58] Prevalence is higher for boys than for girls, but post-puberty, incidence is higher for girls and ultimately prevalence, too, is higher for girls.[4]

Major Known Asthma Risk Factors

Allergens and irritants. Onset and severity of asthma and respiratory diseases have been associated with exposure to allergens, irritants and conditions including: cockroaches, dust mites, rodents, pets, dampness, cold temperature, mold, environmental tobacco smoke (ETS), air pollution, ozone, and volatile organic compounds.[58, 62-65] Lower-SES children may be more likely to be responsive to multiple allergens.[1] African American asthmatic children are more likely than whites to be sensitized to cockroach and dust mite allergens.[66, 67] Cockroach sensitivity is a predictor of severe asthma, and cockroach is the most common indoor allergen among inner-city black children.[67, 68] Other factors known to cause chronic persistent inflammation, such as viral respiratory infections, also reduce lung function and are linked to asthma.[69, 70]

Obesity. Obesity is independently associated with asthma prevalence, severity and control.[71, 72] Proposed physiologic pathways by which body mass index may influence asthma include lung dynamics, undiagnosed co-morbidities, and systemic pro-inflammatory

sequalae.[71, 72] Obesity is also associated with asthma medication use; asthma medical care utilization; asthma hospital and intensive care admissions; and asthma-related quality of life.[71, 73] Obesity may reduce responsiveness to therapies and may be associated with an asthma phenotype that is difficult to control.[73, 74] Prevalence and morbidity of both obesity and asthma have sharply increased in recent years, and these conditions highlight dramatic disparities.

Barriers to Care. SES and race as well as culture are associated with health beliefs and practices. Barriers to care in urban communities may include: variation in physician practice, referral patterns, patient disease knowledge, patient care preferences, transportation, and child care.[56] Subgroup beliefs about and/or distrust in traditional medical care and the U.S. healthcare system may interfere with asthma diagnosis and care. Asthma management plans are often very effective when adhered to, but acceptance of asthma as a chronic condition requiring regular use of controller medications may differ by subgroup due to healthcare inequities, knowledge of the disease, and/or culture. Asthma disparities may be influenced by health practices that lead to reluctance to use prescribed medications and inconsistent use of the same provider.[53] Blacks with asthma have higher rates of hospital-based visits while whites have higher rates of physician office visits, suggesting disparities in preventive care.[75] A study of asthmatic children enrolled in a state Medicaid program found that blacks were 64% less likely than whites to get timely follow-up care after an emergency department visit for asthma.[76] Racial disparities in asthma care, including daily corticosteroid use, self-management education, trigger avoidance information, and specialist care, have been noted in patients who were well-educated and privately insured, suggesting that discrimination may influence asthma outcomes.[77]

Stress and Asthma

"Stress," coined by Hans Selye in 1936, is defined as "the non-specific response of the body to any demand for change."[78] It is a highly subjective phenomenon, and while it is comprised of distress (negative) and eustress (positive), stress often has negative connotations. Selye conceptualized the physiology of stress as having two components: a set of responses which he called the "general adaptation syndrome", and the development of a pathological state from ongoing, unrelieved stress. Selye later coined "stressor" to distinguish impulse from response. Like stress, stressors are highly subjective. Stressors are defined in this research as the internal and external stimuli that produce a bodily reaction, that is, stress, and are believed to have a psychological impact and to affect psychological and physiologic functioning. In addition, psychological stress may affect health by causing health-compromising behaviors and co-morbidities that compromise disease management.[27-38]

Negative affect, emotion and psychological stress are believed to have a role in atopic disorders and asthma, both transiently and long-term, and their mechanisms are increasingly being clarified.[15, 16, 79-81] Increasing asthma prevalence in the West has been concurrent with increasing chronic stress in women, particularly in low-income urban areas where family stress levels are high.[82-85] Even short-term responses to stressors may produce long-term damage if stressors are not eliminated.[86]

Biologic pathways

Neuroendocrine and immune functioning, oxidative stress, and autonomic response help us to explore the mind-body connection.[15] Recent advances have illuminated mechanisms for the influence of psychological stress on asthma expression and development through "dysregulated immunity."[15, 16] Psychological stress may have direct effects that result in inflammation, fundamental to asthma pathophysiology, through

immune-mediated and neurogenic processes (hypothalamic-pituitary-adrenal axis function, proteases-antiproteases, oxidants-antioxidants, cortisol expression, etc.).[15, 87] Psychological stress likely influences the immune system, causing the release of neuroendocrine hormones and a type 2 cytokine response associated with an allergic phenotype. Theories about reciprocal relationships between neural, hormonal and immunological pathways have been posited[21] and are supported by studies showing that psychological stress influences the expression of cytokine patterns of asthmatics or those at-risk of developing asthma.[19, 88] Dysregulation of normal mechanisms from chronic psychological stress may result in hyper-arousal and/or hypersponsiveness and impact atopic disease expression.[16]

Psychological stress has been associated with altered immune expression in adolescent asthmatics[88] and in younger children.[19] Stress can reduce resistance to respiratory infections[89] which are related to asthma morbidity. Maternal distress, measured by their short-term and persistent co-morbidities, that persisted past the child's first year of life was related to child's increased asthma risk at age seven while maternal depression and anxiety limited to the child's first year was associated with subsequent asthma.[85] Chen's study of adolescents with asthma showed that those with higher psychological stress levels have higher levels of cytokines IL-5 and interferon-c, associated with type 2 and type 1 immune responses, respectively.[88] More recent work on asthmatic children showed that chronic psychological stress and threat perception represented statistically significant pathways between SES and immune responses, providing biopsychosocial evidence for the relation between low SES and adverse asthma outcomes.[90] Studies have shown that neuroimmune and genetic processes may lead to differential responses to therapies for asthma and allergy.[21, 23, 90] Research is needed to understand a possible mediating role of

psychological stress, for example, the ability of chronic stressors to mediate the effect of allergens or irritants on atopy and asthma through neuro-immune mechanisms.[15, 16]

Behavioral pathways

Influences of psychological stress on asthma may be mediated through behavioral pathways that expose people to asthma triggers. Psychosocial stress in adolescents has been linked to risk factors for asthma as well as other diseases including increased smoking rates;[30] other substance abuse;[91] lower medication adherence;[92] and adverse psychological symptoms.[33] Stressful events may cause symptoms of depression in adolescents[33] which may impact compliance with asthma treatment.[32] On the other hand, asthma morbidity could increase psychological stress levels. Asthma diagnosis has been associated with an increased number of stressful life events in young adults.[93] Studies have shown that the relation of race and asthma is weakened when quality of life is examined, leading to hypotheses that a person's perspective, which is influenced by family, social and behavioral factors, is important.[94] Though quality of life is not strongly associated with physiologic measures of asthma, it may influence health behaviors such allergen reduction in the home, care seeking and treatment adherence, which in turn influence asthma expression. Archea *et al.* found that individuals across all SES strata experiencing a greater number of negative life events reported worse asthma-specific quality of life (AQoL), as did people with more severe disease (regardless of number of negative life events). In the lowest income category, however, greater number of negative life events was associated with poorer AQoL across severity strata, highlighting the potential important indirect effects of stress.[28]

Pathways involving caregivers and others

Psychological stress may be indirectly associated with asthma expression through caregivers and others. Among infants and youths, exposure to violence, problematic family relationships, parenting difficulties, caregiver stress, critical attitudes of one's mother, negative life events and psychological stress have been related to wheeze, asthma onset and/or adverse asthma outcomes.[18, 19, 25, 33, 37, 39, 85, 93, 95, 96] For children with asthma, psychological stress experienced by parents may lead to impaired problem solving, influence reporting of symptoms, quality of life, and perceptions of asthma outcomes, and allow suboptimal disease management and healthcare utilization to occur.[27, 29, 31, 34, 36-38, 96, 97] A bi-directional relation between maternal stressors and depression and children's outcomes has been proposed.[96] It is possible that the development of asthma and wheeze in young children may result in dysfunctional family interaction, especially if the family encounters stressful life events.[98] Life stress, therefore, may contribute to asthma morbidity and be a consequence of asthma morbidity. A study of the social environment and adolescent asthmatics suggested that family factors may affect asthma through physiologic changes, such as inflammation, while neighborhood factors influence health behaviors, such as smoking, which impact asthma.[24] These studies lend empirical support for the importance of considering acute and chronic exposures and social context in both asthma development and expression at different stages of the lifecourse.[99]

Stress and gender and childhood

The impact of psychological stress and violence may be different for boys and for girls. Girls have been shown to have a greater propensity to become directly involved in parent conflict.[100] It is hypothesized that girls may be more susceptible to the adverse effects of family conflict because they are more sensitive to others or because of strong

identification with their mothers, often the abused.[87] Boys may have more aggressive responses to stressors and be more likely to confront situations alone, and thus may be more negatively impacted by community violence.[101] Girls, however, may exhibit depressive symptoms and seek help with stressful situations.[102] For boys and girls, there may be an effect of emotional development on the violence-stress-health outcomes pathway. Early life trauma and caregiving experiences have been shown to influence emotional understanding and expression.[103] Positive emotion was found to be protective in studies of lung function while negative emotion was associated with impairment.[79, 80, 104]

"Place" and Health

Residence and Public Health

The housing and health relationship has been acknowledged for centuries, not surprising given that shelter is a fundamental need. Infectious diseases such as tuberculosis, typhus and cholera have been fought with measures such as improved sanitation and ventilation, reduced crowding and intrusion by disease vectors, and slum clearance.[16, 64] Substantial decreases in morbidity and mortality rates during the 19th and 20th centuries were due in large part to housing improvements.[105] Chronic diseases, injuries, developmental disabilities, inadequate nutrition and poor mental health, too, have been linked to substandard housing through biological, chemical and physical hazards.[64] Industrialization, migration, economic crises, wars, social unrest, class conflict, and civil rights over the centuries have highlighted the importance of quality and affordability of housing for health and human rights.[64]

The first U.S. health and housing laws were enacted in 1866 in New York City. The American Public Health Association produced "Basic Principles of Healthful Housing" in 1938 and in 1971 identified the need "to understand and assess... better the relative effects

on humans of the various stresses that may exist in housing and its environment."[106] More recently, the Healthy Homes Initiative was established in 1999 by the U.S. Department of Housing and Urban Development.[106, 107] Among HUD strategic goals and objectives are: increasing the availability of decent, safe and affordable housing; reducing segregation of racial and ethnic minorities and low-income households; promoting housing stability; and increasing satisfaction with neighborhoods and safety.[107] Likewise, Healthy People goals address the reduction of substandard housing and environmental improvement.[108] Housing improvements -- including quality and affordability -- through policies, guidelines, codes, interventions, monitoring, and advocacy remain an avenue for public health researchers and practitioners to address an important social determinant of health driving health inequities.[64]

Housing

Understanding the health effects of housing is a priority since children spend as much as 80-90% of their time indoors.[52] More than 80% of U.S. residents live in cities or their adjacent communities, and about 75% of people worldwide are predicted to live in urban areas by 2030.[109] Urban-dwelling children may experience increased risk of health-compromising indoor exposures and behaviors because of concerns about safety outdoors. A recent estimate of the total annual costs for childhood diseases in the United States with an environmental component -- including asthma and respiratory diseases -- is $54.9 billion.[110]

It is clear that many asthma risk factors are associated with housing quality. Water intrusion and moisture, from leaks as well as overcrowding and poor ventilation, allow dust mites, cockroaches, viruses and molds to flourish.[63, 64] In one study, after central heating was installed, children's respiratory symptoms improved and fewer school days were missed due to asthma.[111] Materials used to manufacture flooring and wall coverings have been

associated with bronchial obstruction in early life.[64] Carpeting accumulates dust, allergens and toxic chemicals, which may cause respiratory illnesses, and substandard housing often has old, dirty carpeting. Multi-unit dwellings may have spaces which harbor pests, and deteriorating structures may allow pests to enter homes. Inadequate storage may attract pests when food and clutter are allowed to accumulate. Residentially unstable families were found to have higher cockroach allergen levels.[112] Exposure to tobacco smoke may be increased by poor ventilation, which may also expose people to nitrogen dioxide from combustion appliances. Housing quality and overall health were improved in one study when families moved to renovated, better housing, even while remaining in disadvantaged neighborhoods.[113] Moving from substandard housing was shown to improve mental health in another study,[114] while other relocation studies have shown mental health improvements after moving from high- to low-poverty neighborhoods,[115] highlighting the importance of disentangling housing and neighborhood effects.

Concentrated Disadvantage

Housing-related stressors may extend beyond the walls of one's home to include physical and social dimensions of the immediate neighborhood which may directly and indirectly cause psychological stress. The role of the inner-city environment in increasing asthma prevalence, morbidity and disparities has long been of interest. Because of high levels of residential segregation in many parts of the U.S., residence is correlated with SES and race and ethnicity. Residential segregation is hypothesized to influence health by contributing to socioeconomic inequalities and psychosocial stress at both the individual and neighborhood levels. Living in a racially segregated place may negatively impact health for those who are excluded from desirable resources and opportunities and exposed to hazardous physical and social environments. In the U.S., 71% of blacks and 58% of whites

live in counties that violate federal air pollution standards.[56] High traffic areas in urban communities may expose residents to hydrocarbons from vehicles. Cigarette smoking prevalence is high in urban populations as is passive exposure to ETS. However, while the presence of some allergens and irritants has been shown to differ by race/ethnicity, SES and urbanicity,[1, 116] neither trends in prevalence nor disparities in morbidity are explained by environmental exposures alone.[16, 58, 117]

Concentration of poverty may lead to deterioration of social institutions and networks. Frequent moves, overcrowded living conditions, abandoned housing, and homelessness may result from poor quality or unaffordable housing and may cause neighborhoods to become residentially unstable. Unaffordable housing may also result in depleted household resources for food, utilities, and health care. Social capital and social cohesion may allow residents to work together for mutual benefit and may influence investment in housing, residential stability, crime and poverty.[118, 119] A study of neighborhood-level variation in asthma and respiratory diseases in Chicago found that collective efficacy was protective while residential stability had an association with respiratory outcomes only when collective efficacy was controlled.[53] The authors hypothesized that collective efficacy may protect against respiratory diseases through: 1) social control of health-compromising behaviors; 2) access to health services; 3) management of physical hazards; and 4) promotion of psychosocial health by minimizing fear of being outside and engaging with community. In disadvantaged neighborhoods, social networks may increase risk of some exposures and conditions. Parents may feel the need to protect themselves and their children from neighborhood influences while at the same time have great need for stress buffers.[120] Single parenting may add to the burden of such circumstances.[121] Parents living in communities with high levels of violence may keep their children indoors, leading to

increased exposure to indoor allergens and depriving children of opportunities for exercise, social development, and support networks.

Violence

Trauma theory frames the notion that exposure to violence, a specific psychological stressor, is a cause of childhood morbidity, including respiratory disease.[122] Stressors are thought to cause negative affective states which directly influence biological processes and behaviors that lead to disease risk. Living with violence may cause individuals to view their lives and the world as unpredictable and out of their control. Living in chronic fear under perceived threat, in turn, predisposes people to adverse effects of stress[39, 122] and its psychological, behavioral and physiological sequelae. An estimated 6.2 million youth experience some form of assault or abuse each year; 2.8 million experience injury; and 250,000 require medical attention.[123] Inner-city, poor children are especially at risk of experiencing and witnessing violence in their communities.[123, 124] A study of inner-city Chicago children aged 7-13 found that 42% had seen a person shot; 37% had seen a person stabbed; and about half of boys (55%) and girls (47%) had witnessed violence.[125] Domestic violence also is a source of stress for children. Behaviors such as smoking, substance abuse and violent behavior have been associated with children growing up in chronic violence.[126]

It is possible that parents and children living in the same environment and similarly exposed to chronic violence may not report events to each other to protect each other from additional emotional pain. Also, psychological comorbidity resulting from a parent's violence exposure may influence their reactions to their children's exposure and resulting distress.[126] Caregiver stress may influence child neuroendocrine function during early development and may cue children to adopt less effective coping strategies.[35] A study of violence and feeling

unsafe among low-income, urban asthmatic children and their families found high violence exposure for children and parents, and for those families exposed, more asthma symptoms and less likelihood of seeing their primary care provider or seeking asthma care.[127] A study of urban, suburban and rural U.S. high school students with asthma showed that victimization and missing school because of feeling unsafe increased the odds of having an asthma episode.[128] Increased violence exposure predicted a higher number of symptom days and nights that caregiver lost sleep in a graded fashion, controlling for SES, housing and negative life events.[35, 120]

Housing, Neighborhoods and Asthma in a Biopsychosocial Context

According to Maslov's theory of the hierarchical importance of human needs, physiological needs, such as water, warmth and air to breathe, and then shelter and safety are of primary importance.[51] Thus, attention to so-called "environmental diseases" and to socioeconomic stratification not only has health and economic benefits but presents a moral imperative and may be crucial to the elimination of physical and mental health inequities. However, despite widespread recognition that housing quality and residence are important to health, much work is needed to expand the relevant dimensions and elucidate causal mechanisms involving asthma.

Various disciplines have explored the pathways through which housing and neighborhoods influence health through psychological stress, yet such pathways have rarely been applied in the asthma literature.[16] Just as material exposures act on biology to impact health, social conditions can be "biologically embedded."[16, 50] Recent literature has called for understanding how social environments 'get under the skin' to influence health and how psychological stress is a "social pollutant" that when 'breathed into the body' may disrupt biological systems through inflammatory processes."[16] Fullilove and Fullilove posit a

dynamic relationship between environmental conditions and health behavior; that is, individuals who practice health-compromising behaviors may also contribute to poor environmental conditions, and those poor conditions may contribute to those behaviors. They point out that: 1) shelter is a fundamental necessity; 2) housing units, in relation to one another, create an infrastructure for group life, which is also important for well-being; and 3) housing provides homes, with psychological importance as objects of attachment and sources of identity. Homes also provide a person's orientation to space and time.[129] Materiality, meaningfulness and spatiality are three important dimensions of housing that are, "…well known to shape power relations between social actors and groups, to influence the distribution of control over individuals' life circumstances, and to differentially shape social identity and confer social status."[130]

Residential Stressors

Substandard housing has been linked to poor health among children and adults. Various stressors may be linked to asthma through though biological and behavioral pathways. Housing characteristics including floor level, pests, dampness, noise, housing age, housing type, location, dampness, and temperature have been linked to mental and physical health outcomes[131-135] though few studies have explored causal mechanisms. Temperature, dampness, noise and crowding may impact asthma by causing irritability, social intolerance, anxiety, and depression, [133-135] which may interfere with disease management. There may also be a subjective emotional response to housing which can reflect social status, control and identity and may be positive or negative.[105] Pride in one's dwelling, homeownership, and presence of a tenants' structure were associated with good health status while problems meeting housing costs, forced moves, dislike of being at home, and dissatisfaction with the home's physical features were associated with poorer health

status in one study.[130] Poor quality housing may prevent people from having guests in their homes and lead to social isolation. Multi-unit buildings may lack common areas for people to meet and socialize and expose residents to crime, lack of privacy, and unsafe play for children.[105]

Cost of housing, residential satisfaction, residential instability and lack of control over housing are posited as possible determinants of asthma.[16] Concerns about affordability and lack of control over housing issues may reduce the level of attention paid to one's asthma or lead people to believe that they lack the means to move or remedy household hazards. Homeownership may promote a sense of security and control, and it has been independently associated with improved health[136] though for people on the margins of ownership (experiencing hardship due to this financial obligation or in jeopardy of losing housing), insecurity and mental health problems may develop.[137] Socioeconomically disadvantaged groups devote a higher percentage of income to rent and yet live in more substandard housing.[138] They also experience more negative life events, including those involving housing.[139] Homeownership may vary by subgroup in terms of meaningfulness.[140] Tenure is related to overall health, controlling for income and self-esteem.[141] Moving is considered a stressful event with harmful health effects, and relocation may result in loss of social networks. Perceived residential satisfaction may mediate the effects of objective housing factors on psychological status.[142]

A review of studies that measured changes in social outcomes after housing improvements found reduced sense of isolation and fear of crime, increased sense of belonging, feeling of safety, involvement in community affairs, greater recognition of neighbors, and improved view of the area as a place to live.[140] Neighborhoods may expose residents to social toxins in addition to material ones. Poor social capital and collective efficacy and high crime and violence rates, for example, may influence biology and

behavior. Deteriorated social institutions and informal networks may leave residents without important health and stress-buffering resources.

Why do perceptions matter?

Stressors are subjective; people may react differently to the same stressor, and an individual's reaction may change over time. For example, a household member moving in or out may be a negative or a positive event in a person's life and the difficulty of the experience depends on various individual characteristics and circumstances. Perceptions are important since stressors are internalized by individuals, and they are internalized differently by different individuals. The number of different stressors as well as their frequency of occurrence triggers the body's biological response, and the difficulty of the experiences also influences an individual's stress level. Stress has both psychological and biological aspects since the mind and body are not separable. Perception, thought, and emotion are hypothesized to produce biological changes that in turn impact asthma through "dysregulated immunity," and they may influence behaviors that influence immune system and lung functioning and, ultimately, asthma.

Both acute and chronic stressors and their impacts on disease expression by way of allostatic load have been hypothesized as ways that psychosocial exposures can "get into the body."[15, 35] The consequences of neighborhood-level deprivation are not uniform; rather, individuals' perceptions and reactions may modify the harmful effects of stress caused by specific events and general disadvantage. Stressful events may have a greater adverse impact if a deprived person observes inequities between the environment of her peers and that of others.[143] Economic inequity has been related to health outcomes in various populations reflecting social gradients in health.[144] Social hierarchies have profound effects on the psychological and physiological processes that influence biology and

disease.[143] Crime and violence rates may reflect a community's collective well-being and social cohesion[145] and chronic stress in disadvantaged neighborhoods may affect all residents regardless of individual risks or circumstances. Collective efficacy, reflecting the level of trust and attachment of community residents and their capacity to act for mutual benefit, may promote the ability of neighborhoods to provide health-relevant resources, eliminate environmental health hazards, and promote communication among residents which may enable dissemination of asthma-related information.[53] Though collective efficacy is usually measured and quantified as a contextual determinant, individuals' perceptions and feelings about one another are components of this construct and are important proxies of the burden of stressors on individuals.

Proposed Mechanisms by Which Residential Stressors Influence Health

The conceptual framework for this study of residential stressors and health is a psychological stress mechanism. There are numerous ways by which housing and neighborhood stressors may more directly harm respiratory health, without stress as an intermediary. Utilities not in service may lead to poor ventilation, extreme temperatures, dampness, and mold, or to the inability to access medical advice by telephone. Changing household membership may affect sharing of health knowledge and the level of resources available to manage an asthmatic child's disease. Overcrowding may interfere with sleep and impact immune functioning. Parent perceptions of neighborhoods as physically or socially unsafe may cause them to keep children indoors where exposure to allergens or harmful behaviors may exacerbate asthma. Many more scenarios by which residential factors may influence health can be imagined. The focus of this research and the common link among the stressors of interest in this study is that they are theoretically capable of producing a psychological stress reaction.

We conceptualize the following exposures as "social pollutants" that may be "breathed into the body" and harm health: 1) housing stressors (parent-reported number and difficulty of events and ongoing issues occurring in a 6-month period involving housing security, mobility, comfort, safety, finances, dynamic household membership, and relationships with neighbors and landlords) and 2) neighborhood stressors (parent perceptions of physical and social order and cohesion and collaboration among residents). A substantial body of literature demonstrates the association of life events or life stressors with depression[146-149] and with various physical and mental health outcomes.[147, 150-156] We hypothesize that acute and chronic residential stressors, relevant for low-SES, urban families, cause psychological stress which influences respiratory health and general health through biologic and behavioral pathways.

Biologic pathways. A growing number of studies links psychological stress to asthma, including onset, precedent phenotypes, and disease exacerbation.[15, 16, 18-21, 21-26] Recent advances have illuminated mechanisms for the influence of psychological stress on asthma through "dysregulated immunity."[15, 16] Psychological stress may have direct effects that result in inflammation, fundamental to asthma pathophysiology, through immune-mediated and neurogenic processes.[15, 87] Psychological stress likely causes the release of neuroendocrine hormones and a type 2 cytokine response associated with an allergic phenotype. Dysregulation of normal mechanisms from chronic psychological stress may result in hyper-arousal and/or hypersponsiveness and impact atopic disease expression.[16]

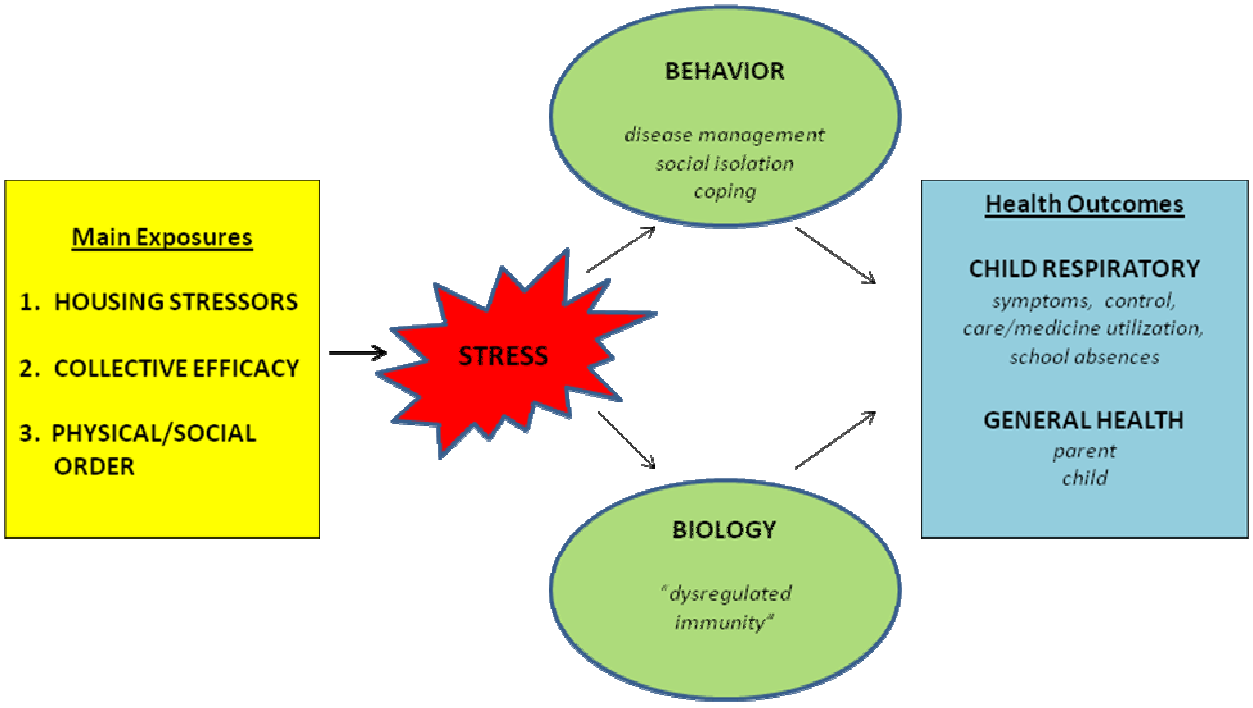
Behavioral pathways. Psychological stress experienced by children or their parents may affect asthma by causing health-compromising behaviors and co-morbidities that compromise disease management.[27-38] Stress may lead to smoking, becoming overweight and leading a sedentary lifestyle. Depression and anxiety may lead to poor adherence to treatment or poor disease management of children's asthma by their

caregivers. Neighborhood disadvantage may cause individuals to withdraw from the community, prevent people from accessing care or filling prescriptions, and keep their children indoors, exposing them to indoor allergens and robbing them of opportunities for development and social support. 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. There is also evidence that parent stress may produce stress responses in children and cue children to adopt suboptimal coping strategies.[35] 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.[157] Recurrent, negative thoughts about past events may produce ongoing stress and comorbidities.[158, 159] and may influence problem-solving and perceived control.[160]

Asthma is a complex condition with many interrelated causes as well as unknown and difficult-to-measure risk factors. Traditional "environmental" asthma risk factors, such as mold and cockroaches, are not the focus of this study as they have been well-documented in the biomedical literature. Other data were beyond the scope of the NIEHS study and do not exist in the dataset, for example, variables for genetics, air pollution and violence. Biological and behavioral variables are hypothesized to be on the causal pathways from stressors to health and are included in the theoretical model but not included as covariates in analyses. Figure 1 represents the proposed conceptual model of the influence of residential stressors on health. It is simplified to highlight the proposed mechanisms, that is, the production of psychological stress by the main exposures, and the

subsequent influence of stress on respiratory health through biological and behavioral changes. A variety of health outcomes are likely to be similarly influenced by stress pathways; therefore, general child and adult health are also examined.

Figure 2.1. Conceptual framework for the effect of residential stressors on health through psychological stress pathways



Study Rationale

Asthma disproportionately affects non-white, urban, and low-SES populations. Chicago's asthma prevalence and morbidity and mortality rates are among the highest in the nation, and within the city, they are typically highest in neighborhoods with the lowest SES. [6, 7, 9, 11-13] Despite a decade of extraordinary efforts to increase asthma equity in Chicago, progress has been modest.[13] Furthermore, Chicago's burden, and the national epidemic,

are not well-explained by known risk factors. Not all urban communities have excess asthma though they may share low-SES and environmental exposures with high-risk urban areas.[1, 6, 16, 53] Attention to social and physical factors that covary with lower SES is needed to contextualize asthma and understand its social patterning.[1] Recent literature has also called for understanding how social environments "get under the skin"[49] and become "biologically embedded"[50] to influence health.[1, 16] Acute and chronic stressors are believed to have psychological effects that in turn influence psychologic and physiologic functioning as well as behavior. Low-SES and other disadvantaged groups may experience increased stressors and be more strongly affected by them due to already-compromised health and coping resources.

The ongoing burden of asthma on inner-city populations demands more thoughtful investigation of determinants of risk. An important contribution of this study will be a better understanding of the *kinds of stressors* that produce stress and impact health. Residence-related stressors may be of utmost importance as housing is a basic human need which carries physical and emotional meaning and confers physical and emotional health. This study aims to advance asthma scholarship by incorporating novel exposure variables into a theoretical framework of how psychosocial factors become biologically embedded and influence health through psychological stress pathways. Survey research is often limited in its ability to uncover social mechanisms and help researchers to generate sound hypotheses for specific populations.[161] We use unique data not collected in surveillance activities or in typical asthma investigations to create multidimensional measures of policy-relevant stressors, robust to problems with single variables, for example, influences of secular, geographic or seasonal trends. It would be inappropriate to make inferences based on one variable without considering the remaining interrelated factors that contribute to stress. Good interventions have multifaceted effects and address a breadth of

interrelated factors. Identification of the interrelated factors that may covary with indicators of disadvantage may inform effective Public Health interventions by allowing us to address the complex circumstances of low-income families associated with onset and expression of disease. Attention to complex psychosocial issues may also illuminate new priorities for causal research. The rationale for this research has resulted in the following hypotheses:

Hypothesis 1: Housing Stressors. Material and social aspects of housing including security, mobility, comfort, safety, finances, dynamic household membership, and relationships with neighbors and landlords cause psychological stress, which influences biological and behavioral pathways to health for parents and children. Increased exposure to housing stressors, reflecting the number of stressors and the difficulty of the experience(s), as reported by parents, is associated with increased risk of poor health outcomes for parents and their children.

Hypothesis 2: Neighborhood Stressors. Numerous aspects of neighborhoods, such as existence of physical hazards and lack of cohesion and collaboration among residents, cause psychological stress, which in turn influences biological and behavioral pathways to health for parents and children. Poor parent perceptions of their neighborhoods are associated with increased risk of poor health outcomes for parents and their children.

CHAPTER 3
RESEARCH DESIGN AND METHODS

Specific Aims

Research aims are divided into two groups by unique main exposure variables and discussed throughout this dissertation as follows:

Specific Aim 1: Housing Stressors and Health. To investigate the association of housing stressors and child respiratory health and parent and child general health among low-income families in Chicago.

Specific Aim 2: Neighborhood Stressors and Health. To investigate the association of parents' perceptions of neighborhood stressors and child respiratory health and parent and child general health among low-income families in Chicago.

The study population is the same for both research aims, though aim 1 uses baseline data, and aim 2 uses survey 2 data (with participants lost to follow-up). The same 8 health outcomes are investigated across aims. These outcomes are investigated in separate models since they represent a mixture of disease severity, control, and care-seeking behavior and are not appropriate for measurement by one variable. Some covariates are the same across aims while others are included or excluded given their relationships to the exposure(s) and outcomes for each aim. Sections immediately below describe overall study design relevant to both research aims. Following sections describe methods particular to aim 1 or 2, with an emphasis on creation of exposure variables and model specification.

Overview of Study Design

We used survey data collected in 2001-2005 for a NIEHS-funded observational investigation of childhood asthma disparities among low-income Chicago families. With a

cross-sectional design we explored the associations of 1) housing stressors and 2) neighborhood stressors with eight parent-reported health outcomes. Six child respiratory health outcomes include: waking at night, exercise intolerance, school absences, rescue medication use, unplanned medical visits and controllability. Stress pathways are hypothesized to influence a variety of mental and physical health conditions; therefore, two additional outcomes are examined: parent and child general health. Regression analyses estimated absolute measures of effect -- risk (RD) and incidence rate (IRD) differences -- and 95% confidence intervals (CI).

Study Setting and Population

Recruitment of schools. The NIEHS study surveyed for asthma and respiratory problems in 15 low-income, public elementary schools that met the following recruitment strategy: 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 (no magnet schools were included so that a school's enrollment lived in the same community). Low-income was defined as coming from families that are receiving public aid, living in institutions for neglected or delinquent children, being supported in foster homes with public funds, or being eligible to receive free or reduced-price lunches. The schools were a convenience sample; the study surveyed the first 5 eligible schools per year in each of 3 years that agreed to participate.

Schools were surveyed with the Brief Pediatric Asthma Screen Plus (BPAS+) and the Spanish version of the BPAS+, validated, parent-completed respiratory questionnaires.[162, 163] The school surveys (n=13,139; 90% of the total enrollment surveyed) yielded three groups of children: 1) diagnosed asthma; 2) respiratory symptoms suggestive of asthma but no diagnosis (hereafter referred to as "undiagnosed"); and 3) no asthma

symptoms or diagnosis (hereafter referred to as “non-asthmatics”), from which families were recruited for a longitudinal study.

Recruitment of families. Families were randomly selected from among three race/ethnicity groups (with the goal of approximately equal numbers of Hispanic, non-Hispanic black and non-Hispanic white participants) and three asthma diagnosis groups (with the goal of approximately ¼ diagnosed, ¼ undiagnosed and half non-asthmatics). Eligibility criteria included: child 5-13 years of age; family residing in Chicago; and telephone number provided. Families with more than one eligible child were allowed to enroll only once. The longitudinal study included data collection at baseline and 12 months by telephone and at 6 months in families’ homes. Families chose whether to participate in English or Spanish. All three surveys elicited data on: child respiratory health (except for non-asthmatics), healthcare utilization and general health; parent physical and mental health, life stressors and social support; and family socioeconomic data. A total of 1244 (64% response at baseline) were enrolled, including families of 351 diagnosed, 331 undiagnosed, and 562 non-asthmatics. Parents of non-asthmatic children participated only at baseline. The diagnosed and undiagnosed groups provided the sample for specific aim1 (n=682 total) since the focus of these analyses is respiratory health. Of those 682 families, 319 (47%) also completed the 6-month survey (158 diagnosed and 161 undiagnosed) and provided the sample for specific aim 2. Data from the 12-month survey were not used in this research.

Analytic Strategy

We used SPSS 16.0 (SPSS Inc., Chicago, Illinois) for data reduction by Principal Components Analysis (PCA), SAS 9.1 (SAS Institute Inc., Cary, North Carolina) for descriptive statistics and regression analyses, 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. Univariate examination of dichotomous outcomes defined as any sign, symptom, absence or utilization vs. none, or unfavorable (poorer general health or control) vs. favorable group revealed that all outcomes were common in both waves of data (baseline and 6-month). Odds ratios would therefore overestimate relative risks. We estimated absolute measures of effect and 95% CIs for associations between main exposures (stressors scores) and health outcomes. Missing data were minimal; analyses were conducted for cases with complete data. Binomial regression estimated RDs for binary outcomes while negative binomial regression estimated IRDs for count outcomes. Bivariate analyses assessed exposure-outcome relationships and provided crude RD and IRD estimates. Multivariate models included sociodemographic confounding variables and produced adjusted RDs and IRDs used to evaluate the predictive importance of each main exposure. The strength of each exposure-outcome association was measured as the magnitude of the RD or IRD and its precision, measured as the width of the corresponding 95% CI.

Health Outcomes

Child respiratory outcomes. Six parent-reported child respiratory outcomes were investigated for each aim. Outcomes were developed by the main study's investigators, including a pediatric allergist, and based on their clinical and research expertise and review of the literature. A number of measures of asthma symptoms, therapies and control for children and adults exist (REFS), though time reference periods differ. Four outcomes were reported as the number of days the event occurred in a two-week period to minimize recall bias: exercise intolerance, waking at night, school absences and rescue medication use. Analyses of rescue medication use were restricted to diagnosed asthmatics since undiagnosed children probably do not have access to prescribed asthma medications.

Unplanned visits, defined as the number of visits to an emergency department and to a physician's office or clinic without having made an appointment at least one day in advance, were reported for a six-month period since these are typically rare occurrences. Controllability was a subjective parent rating with no time period specified and four response choices: not at all, somewhat, quite, or extremely controllable. All survey questions specified that parents were to report events pertaining to asthma/respiratory problems (as opposed to, for example, waking up at night from another disturbance or becoming fatigued for another reason).

Parent and child general health. Two parent-reported general health outcomes were obtained from the Child Health Questionnaire (CHQ) and the Short Form-12 (SF-12) (for parents). These instruments are widely-used, reliable measures validated in numerous languages with numerous populations.[164, 165] The global health question from each instrument does not specify a time period and has five response choices: excellent, very good, good, fair and poor.

Effect measure modification. We assessed effect measure modification (EMM) by stratification analyses: Mantel-Haenszel chi-squared tests of homogeneity for binary outcomes and comparison of IRD estimates and CIs for count outcomes. No strong or consistent evidence of EMM was observed in analyses for both aims; therefore, inclusion of interaction terms in statistical models and likelihood ratio tests comparing them to main effects models were not undertaken.

Confounding. We first identified potential confounders in directed acyclic graph (DAG) analyses;[166] 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[167] and adjusted for them in multivariate models created by backward elimination. We removed covariates from the full model singly beginning with the variable producing the smallest change in the exposure-outcome association. When removal changed the magnitude of association by <10%, the covariate was not retained. As a result, each model had a potentially unique sufficient set of adjustment variables.

Protection of human subjects. The Institutional Review Board of the University of North Carolina at Chapel Hill approved this dissertation research. This research is a secondary analysis of extant data originally collected for an NIEHS study conducted during 2001-2005 and did not involve contact with human subjects. Data collection activities for the NIEHS study received approval from the Institutional Review Boards of the investigators' academic institutions and from the Board of Education of the Chicago Public Schools. There were minimal risks associated with this research. The potential loss of privacy through the NIEHS study investigators' access to personal identifying information was minimized by providing the graduate student conducting this dissertation research with datafiles stripped of identifying information. The NIEHS study provided pizza lunches or cookie breaks to schools and $10 grocery store gift certificates to families as incentives for participating. Families (and schools, with parent permission) received the results of the respiratory surveys and health information and resources for asthma care. Indirect benefits to participating families and to the public include a better understanding of determinants of asthma among low-income, urban populations.

Methods for Specific Aim 1: Housing Stressors and Health

This analysis uses data from parents of children with diagnosed asthma (n=351) and undiagnosed asthma (n=331) obtained during the baseline survey; 562 children with no

diagnosis and no symptoms were not included in this analysis since the focus of this study is respiratory health among children with asthma or possible asthma.

Main Exposure: Housing Stressors

We created a variable representing the number and parent-rated difficulty of housing stressors experienced in a six-month period that captured material, social, and emotional dimensions of housing. We identified 13 survey items representing potential sources of psychological stress caused by housing issues (table 4.1). These items are from the Crisis in Family Systems-Revised (CRISYS-R), an index of contemporary life stressors.[149] Developers of the instrument note that traditional measures are dated, rigid, and culturally and socioeconomically biased.[147, 150, 152, 154, 155, 168, 169] The CRISYS-R includes items particularly relevant to, but not limited to, low-income populations. Stressors include daily hassles and "life events" that represent the population's experience and also allow individual variation. The development of the instrument blended "traditional" stressors, the researchers' knowledge of inner-city families encountered in the clinical setting, and feedback from target communities and community case managers who worked with low-income families of chronically-ill and disabled children.[96] Validation studies demonstrate good test-retest reliability and construct validity better than other researchers have found using life events measures to predict depression.[146, 147] Correlation with the Center for Epidemiologic Studies-Depression (CES-D) instrument of depressive symptomatology indicated that the number of CRISYS-R stressors accounted for 22% of the variance in CES-D scores in one study [148] and 19% in another study with a more socioeconomically and residentially diverse population.[149]

The main exposure variable attempts to reflect the multidimensional character of housing stressors. A large number of variables related to housing precludes modeling all of

them individually. Data reduction techniques, such as principal components analysis, were not ideal since the variables of interest are binary variables. Also, the *accumulation* of stressors is hypothesized to impact mental and physical health, therefore we desired to capture a breadth of relevant stressors as opposed to creating a scale of correlated variables measuring an underlying construct. The development of the CRISYS-R and its validity for low-income, urban populations makes its content well-suited for this study, though all 63 items are not included in this analysis since not all are housing-related. Stressors referring to neighborhood events and conditions were excluded in an effort to create a main exposure reflecting the housing experience of individual families.

Parents answered yes or no to having experienced each of 13 stressors in the six months prior to the survey. For each stressor experienced, a parent rated its difficulty on a four-point scale: not at all, a little bit, medium, or a lot (Table 4.1 presents average difficulty by stressor). This difficulty variable allowed us to weight the main exposure score rather than using a simple sum of stressors, which would imply equal importance in terms of their influence on psychological stress. We computed a continuous score by summing the stressors reported by each parent, each weighted by the parent-reported difficulty. A higher score reflects increased number and difficulty of stressors. We chose not to categorize the exposure variable to avoid losing valuable variation reflected in the continuous score. In crude and fully adjusted logistic regression models for three binary outcomes, Hosmer and Lemeshow goodness-of-fit tests supported the square root transformation of this variable when compared to: 1) the untransformed continuous variable; and 2) the square transformation of the continuous variable. The square root transformation was also supported by fully adjusted negative binomial models for five count outcomes. Pearson chi-square, deviance and log likelihood statistics for models with the untransformed continuous

exposure were very similar to those with the transformed exposure; the square root transformation was retained for consistency across all eight outcomes.

Thirty-seven percent of parents reported no housing stressor during the previous six months, and no parent reported more than nine. For the 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).

Health outcomes. We recoded three nominal outcomes into binary variables after determining that ordinal coding was not appropriate due to violation of the proportional odds assumption for controllability and child general health. The general health variables are commonly dichotomized in the literature; therefore, adult general health also was used as a binary variable for consistency and interpretability of results. The more favorable outcome served as the referent category for each. Child and parent general health were recoded as poor or fair vs. good or very good or excellent, consistent with their use in numerous research studies. Controllability of child's asthma was recoded as not at all or somewhat vs. quite or extremely controllable.

Five child respiratory outcomes were included in models as counts: exercise intolerance, waking at night, school absences, rescue medication use and unplanned visits. Univariate statistics identified outlier values for unplanned visits; four cases with values >=20 were excluded form analyses using this outcome (mean=1.3, SD=2.2, range 0-18). Univariate statistics highlighted the high proportion of cases with zero values (53-84%) for all count outcomes. Table 4.2 presents descriptive statistics for outcomes. Overdispersion is addressed in the discussion of model choice in the section on regression analyses.

Socio-demographic covariates. We assessed EMM by stratification analyses for the following variables: child age (<7.9, 7.9-10, >10 years); child sex (male vs. female); child

race/ethnicity (non-Hispanic black, non-Hispanic white, Hispanic); child asthma diagnosis (diagnosed vs. symptoms but no diagnosis); parent asthma diagnosis (diagnosed vs. not diagnosed); parent age (20-29, 30-39, >=40 years); parent education (less than high school, high school, more than high school); parent marital status (single vs. married or cohabiting); parent nativity (foreign-born vs. U.S.-born); parent current smoker (yes vs. no); presence of household smoker (any vs. none); 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); Temporary Assistance to Needy Families (TANF) (any recipient in household vs. none); and parent depressive symptoms (CES-D score >=16 vs. <16). Validation studies support a score of 16 to discriminate individuals with depressive symptomatology from those without.[170, 171] Table 4.2 presents descriptive statistics for covariates.

Among the variables assessed as potential modifiers, none was observed to be a strong or consistent modifier. Eight variables were subsequently assessed as potential confounders while eight others were excluded as possible covariates because in the psychological stress framework they were hypothesized to be on causal pathways (parent depressive symptoms, parent current smoker, household smoker), not associated with both outcome and exposure (parent asthma diagnosis, child asthma diagnosis, child sex, child age, parent age), and/or likely had bi-directional associations with other variables in the model (parent depressive symptoms, parent asthma diagnosis, child asthma diagnosis,).

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. Final models are presented in tables 3.1 and 3.2. Covariates, coded as for EMM analyses except where otherwise noted, included: parent education (did not complete high school vs. high school diploma or beyond); parent marital status; parent nativity; child race/ethnicity; home ownership; housing type;

overcrowding; and TANF. Parent race/ethnicity was not ascertained in the study; therefore, child race/ethnicity is considered an imperfect proxy for the parent. Ten, non-black, non-Hispanic, “other” subjects were categorized as white.

Table 3.1. Risk differences (excess cases per 100 at risk) and 95% confidence intervals from unadjusted and adjusted binomial regression models of the associations of housing stressors (main exposure) and binary health outcomes*

	Risk Differences (95% Confidence Intervals)		
	Parent General Health	Child General Health	Controllability
Unadjusted models			
Intercept**	18.5 (14.2, 22.8)	9.9 (6.4, 13.4)	16.9 (12.8, 21.0)
Housing Stressors***	3.1 (0.8, 5.3)	4.2 (2.1, 6.2)	2.8 (0.7, 5.0)
Adjusted models†			
Intercept**	13.4 (8.1,18.6)	5.7 (2.7, 8.7)	15.2 (10.9, 19.5)
Housing Stressors ***	1.6 (-0.8, 3.9)	2.6 (0.5, 4.6)	2.5 (04, 4.7)
Black	--	--	--
White	--	--	--
Foreign-born	-6.9 (-14.0, 0.3)	--	--
<High School	--	11.6 (3.9, 19.2)	8.3 (0.7, 15.8)
TANF	12.2 (4.4, 19.9)	4.7 (-2.8, 12.2)	--
Unmarried	9.1 (2.2, 15.9)	--	--
Renter	--	--	--
Multi-family	--	--	--
Overcrowded	8.0 (0.03, 15.9)	7.0 (0.2, 13.8)	--

*General health recoded as fair or poor vs. excellent or very good or good (referent); Controllability recoded as not at all or somewhat vs. quite or extremely controllable (referent)
**Intercept estimate is the risk in the unexposed (not risk difference)
***Housing stressors main exposure was modeled as a square root transformation of a weighted, continuous variable; estimate is for a 1-unit change
†Adjusted for up to 8 socio-demographic covariates: black/white (referent=Hispanic); foreign-born (referent=U.S.-born parent); <high school (referent=parent has high school education or beyond); TANF (Temporary Assistance to Needy Families) (referent=no beneficiary in household); unmarried (referent=parent married or cohabiting); renter (referent=homeowner); multi-family (referent=single-family housing); overcrowded (referent=home not overcrowded)

Table 3.2. Incidence rate differences (excess days of outcome per 2 weeks) and 95% confidence intervals from unadjusted and adjusted negative binomial regression models of the associations of housing stressors (main exposure) and child respiratory outcomes*

	(Incidence Rate Differences (95% Confidence Intervals)				
	Exercise Intolerance	Waking at Night	Unplanned Medical Visits	School Absences	Rescue Medication**
Unadjusted models					
Intercept***	0.00 (-0.24, 0.24)	0.14 (-0.04, 0.33)	-0.01 (-0.20, 0.18)	-0.93 (-1.26, -0.60)	0.79 (0.51, 1.07)
Housing Stressors†	0.28 (0.17, 0.38)	0.16 (0.07, 0.25)	0.16 (0.08, 0.24)	0.01 (-0.05, 0.07)	0.04 (-0.25, 0.32)
Adjusted models††					
Intercept***	0.26 (-0.01, 0.53)	0.22 (0.01, 0.43)	-0.14 (-0.39, 0.10)	-0.64 (-1.16, -0.12)	0.68 (0.19, 1.17)
Housing Stressors†	0.30 (0.17, 0.44)	0.12 (0.01, 0.23)	0.11 (0.03, 0.19)	-0.01 (-0.10, 0.08)	0.04 (-0.24, 0.32)
Black	--	--	--	--	0.51 (-0.58, 1.60)
White	--	--	--	--	0.79 (-0.84, 2.41)
Foreign-born	-0.73 (-1.08, -0.38)	-0.57 (-0.84, -0.29)	--	-0.33 (-0.58, -0.09)	--
<High School	--	--	0.16 (-0.16, 0.47)	--	0.58 (-0.74, 1.90)
TANF	--	0.30 (-0.14, 0.74)	0.18 (-0.13, 0.49)	0.13 (-0.22, 0.49)	0.25 (-0.91, 1.42)
Unmarried	--	--	--	-0.17 (-0.40, 0.06)	-0.64 (-1.41, 0.12)
Renter	--	--	--	0.24 (-0.25, 0.72)	0.29 (-1.03, 1.60)
Multi-family	--	--	0.11 (-0.14, 0.37)	-0.18 (-0.42, 0.06)	-0.30 (-1.18, 0.58)
Overcrowded	--	0.38 (-0.12, 0.89)	--	0.26 (-0.19, 0.72)	-0.16 (-1.11, 0.79)

*Respiratory outcomes reported by parents for 2-week period except for unplanned visits (6 months), modeled as counts
**Includes diagnosed asthmatics only (n=351)
***Intercept estimate is the rate in the unexposed (not rate difference)
†Housing stressors modeled as a square root transformation of weighted, continuous variable; estimate for 1-unit change
† †Adjusted for up to 8 socio-demographic covariates: black/white (referent=Hispanic); foreign-born (referent=U.S.-born parent); <high school education (referent=parent has high school or beyond); TANF (Temporary Assistance to Needy Families) (referent=no beneficiary in household); unmarried (referent=parent married or cohabiting); renter (referent=homeowner); multi-family (referent=single-family housing); overcrowded (referent=home not overcrowded)

Regression Analyses

Tables 3.1 and 3.2 present beta coefficients for the main exposure and l covariates for unadjusted and adjusted models. To make meaningful exposure level contrasts, rather than only report the coefficient for a 1-unit change in exposure, we also estimated the RD/IRD for the 75th and 25th quartiles of housing stressors score, that is, the difference associated with an increased stressors score (table 4.3).

Binary outcomes. We used binomial regression to estimate RDs for binary outcomes (SAS GENMOD specifying identity link function and binomial distribution). The more favorable outcome group is the referent, therefore, estimated RDs in table 4.3 represent the excess cases per 100 persons at risk of the unfavorable outcome associated with the 75th vs. 25th percentile of housing stressors, with all covariates at their referent levels.

Count outcomes. We used negative binomial regression to estimate IRDs for count outcomes since all outcomes had a large proportion of zero counts (53-84%). IRDs in table 4.3 represent the excess number of days the unfavorable outcome occurred per two-week period (or six-month period for unplanned visits) associated with the 75th vs. 25th percentile of housing stressors, with all covariates at their referent levels.

The large proportion of subjects with a zero value for each outcome caused overdispersion (the observed variance of the rates was greater than expected) and required careful choice of models to address the issue of extra-Poisson variation. First, we examined two mixed regression models, zero-inflated Poisson (ZIP) and zero-inflated negative binomial (ZINB). Zero-Inflated regression is a mixture of two statistical processes, one always generating zero counts and the other generating both zero and nonzero counts. It assumes that zero counts might come from two different sources; a logit model with binomial

assumption is used to determine if an individual count outcome is from the always-zero or the not-always-zero group, and a model for either Poisson or negative binomial count data is used to model outcomes in the not-always-zero group.[172] Each model yields two sets of estimates which compare: 1) those with 0 vs. >0 outcome values and 2) the change in outcome for those with non-zero values. ZINB model fit statistics, Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC), were superior to those for ZIP models, and ZINB models had larger SEs, better reflecting extra-Poisson variation. Since ZINB models yielded estimates that demonstrated no significant effect for zero inflation, we next tried negative binominal regression models which included a dispersion parameter to accommodate the unobserved heterogeneity in the count data. SAS PROC GENMOD is not able to produce CIs for negative binomial estimates; therefore, log likelihood functions were modeled in SAS NLMIXED to obtain estimates and 95% CIs. The dispersion parameter estimates confirm that negative binomial models adequately addressed overdispersion. Log likelihood statistics indicated good model fit for models with all five outcomes (relative to the zero-inflated models with the extra parameter), thereby informing our choice of negative binomial models as our final models.

Methods for Specific Aim 2: Neighborhood Stressors and Health

This analysis uses data from parents of children with diagnosed asthma (n=158) and undiagnosed asthma (n=161) obtained during a home visit (survey 2).

Main Exposures: Physical/Social Order and Collective Efficacy

Principal components analysis. We created summary main exposure variables representing neighborhoods by data reduction techniques to take advantage of rich survey data and to capture multi-faceted characteristics of neighborhoods. We identified 27 items representing potential neighborhood stressors in the survey instrument and recoded

variables so that the lowest value represented the most favorable (least likely to cause psychological distress) category. Fourteen items were taken from the Community Survey from the Project on Human Development in Chicago Neighborhoods (PHDCN),[182] though no attempt was made to replicate PHDCN's survey nor was there an expectation that PCA for this study's sample would yield scales identical to those used in PHDCN research. Thirteen additional survey items were developed by the main study's investigators and hypothesized to have potential health effects. We dropped 13 variables not correlated with at least one other item at the level of 0.510 or greater from further analysis (appendix A). We used an iterated principal components analysis of 14 remaining variables for the extraction method in the absence of *a priori* theoretical knowledge of underlying constructs or of shared variance among variables (table 5.2). Varimax (orthogonal) rotation summarized the co-variation among the variables since the goal was data reduction to a set of uncorrelated measures for subsequent use in multivariate analyses.

Extractions for 2, 3, 4, and 5 component solutions were examined; scree plots and eigenvalues informed the selection of a three-component solution. Twelve items comprised 3 components which explained 58% of the total variance. Components1, 2 and 3 accounted for 28%, 16% and 14% of the total variance, respectively, and had eigenvalues ranging from 3.9 to 2.0 (appendix B). A fourth component had an eigenvalue of 1.2, much less than the next highest value of 2.0; therefore three rather than 4 components were chosen. Interpretation of components was limited to theoretically salient variables with loadings >0.60 and no loading >0.30 on the other components to provide a conservative representation of the amount of variation in the respective variable that is accounted for by the component (table 5.2). We computed summary scores by obtaining the mean value of all variables loading on each component, all of which were on the same metric. Summary scores rather than factor loadings were used since the component variables were untested

and exploratory, with no evidence of reliability or validity. Also, the summary scores preserve the variation in the data, beneficial for their subsequent use in multivariate analyses.[173] Scores were named: 1) physical/social order; 2) collective efficacy; and 3) recent change in neighborhood (hereafter referred to as order, collective efficacy, and change). Change, with the lowest eigenvalue among the three-component solution, was not explored in regression analyses because it comprised only three items and allowing substitution of missing data would compromise its validity. Also, its interpretation would be difficult in the absence of baseline data on the neighborhood at the start of the five-year period and whether improvement was desirable or residents were satisfied with their surroundings.

Missing data were few, but we allowed a maximum of one missing item's value to be replaced by the mean of the non-missing items for that summary score in order to retain the maximum number of cases for complete case analysis. This resulted in 31 of 37 (collective efficacy) and 19 of 24 (order) cases with missing data to receive an exposure score, yielding very few cases missing final exposure scores. Internal consistency, measured by Cronbach's alpha, was above acceptable[174] for both summary scores (table 3.3).

Table 3.3. Internal consistency and descriptive statistics for summary scores resulting from principal components analysis of 14 housing stressors (n=319)

Main Exposure	Cronbach's Alpha	Items N	Mean	Variance	Missing Data N (%) Substitution	
					Before	After
Physical/ social order	0.831	4	6.25	5.590	24 (7.5)	5 (1.6)
Collective efficacy	0.783	5	11.0	7.851	37 (11.6)	6 (1.9)

Summary scores for collective efficacy and physical/social order were then categorized into three levels based on natural cut-points in the distributions and on clarity of

interpretation of results (*i.e.,* a summary score above the mean tends towards agreement with variables indicative of high collective efficacy and should not be categorized as low). Three-level variables better reflected variation in these exposures than binary variables; sample size would not support the use of quartile or quintile coding. Three-level coding allowed two exposure group contrasts: middle versus high and low versus high, with high reflecting the most favorable (least health-harming) exposure category. The majority of scores for collective efficacy and order fell into the middle category (45% and 43%, respectively) and roughly equal proportions in the best (25% and 31%) and worst (30% and 26%) categories.

Outcomes. We recoded eight parent-reported health outcomes as binary variables with the absence of symptoms or the most favorable outcome serving as the referent category. Child and parent general health were rated on a five-response scale and recoded as poor or fair vs. good or very good or excellent. The number of unplanned visits to an emergency department, physician's office or clinic for child's asthma or breathing problems during the previous six months was coded as any vs. none. Four child asthma/breathing outcomes: 1) waking at night; 2) school absences; 3) rescue medication use, and 4) exercise intolerance were reported for a two-week period and coded as any vs. none. Analyses using the rescue medication outcome included only diagnosed asthmatics since undiagnosed children might not have access to prescribed asthma medications. Controllability of child's asthma was a four-response variable recoded as not at all or somewhat vs. quite or extremely controllable.

Demographic covariates. We assessed EMM by stratification analyses for the following variables: child age (<7.9, 7.9-10, >10 years); child sex (male vs. female); child race/ethnicity (Hispanic, non-Hispanic black, non-Hispanic white); child asthma diagnosis (diagnosed vs. symptoms but no diagnosis); parent age (20-29, 30-39, >=40 years; parent

education (less than high school, high school, more than high school); parent marital status (married or cohabiting vs. not); parent nativity (foreign-born vs. U.S.-born); and parent depressive symptoms (CES-D score >=16 vs. <16).

Among the variables assessed as potential modifiers, none was observed to be a strong or consistent modifier. Five variables were subsequently assessed as potential confounders while four others were excluded as possible covariates because in the psychological stress framework they are hypothesized to be on causal pathways (parent depressive symptoms), not associated with both outcome and exposure (child asthma diagnosis, child sex, child age), and/or likely had bi-directional associations with other variables in the model (child asthma diagnosis, parent asthma diagnosis, parent depressive symptoms).

Since we identified potential confounders conceptually in DAG analyses and then quantitatively assessed a subset with a 10% change-in-estimate criterion, each model had a potentially unique set of adjustment variables. Final models are presented in tables 3.4 and 3.5. Covariates, coded as for EMM except where otherwise noted, included: parent education (less than high school vs. high school or beyond); parent marital status; parent nativity; parent age and child race/ethnicity. Parent race/ethnicity was not ascertained in the study; therefore child race/ethnicity is considered an imperfect proxy for the parent. Five non-black, non-Hispanic, “other” subjects were categorized as white.

Table 3.4. Risk differences (excess cases per 100 at risk) and 95% confidence intervals for unadjusted and adjusted binomial regression models of the associations of collective efficacy (main exposure) and 8 binary health outcomes*

	Risk Differences (95% Confidence Intervals)			
	Parent General Health	Child General Health	Unplanned Medical Visits	Controllability
UNADJUSTED MODELS				
Intercept**	7.8 (1.8, 13.8)	11.7(4.5, 18.9)	27.6 (17.6, 37.7)	16.0 (7.7, 24.3)
Middle CE***	14.9 (5.8, 24.1)	2.5 (-6.7, 11.7)	-4.9 (-17.1, 7.3)	4.6 (-6.1, 15.2)
Low CE***	22.7 (11.7, 33.8)	6.2 (-4.3, 16.7)	-1.0 (-14.5, 12.4)	4.0 (-7.6, 15.6)
ADJUSTED MODELS†				
Intercept**	5.1 (-3.6, 13.8)	7.9 (1.0, 14.8)	33.3 (22.2, 44.4)	9.7 (-1.7, 21.1)
Middle CE***	13.5 (2.3, 24.6)	2.5 (-6.7, 11.7)	-4.7 (-16.6, 7.2)	4.8 (-5.8, 15.5)
Low CE***	20.8 (7.8, 33.9)	7.5 (-2.5, 17.6)	-3.0 (-16.3, 10.3)	7.5 (-4.0, 19.0)
Black	-1.0 (-13.4, 11.4)	--	--	--
White	-5.1 (-21.3, 11.2)	--	--	--
Foreign-born	--	--	-11.8(-21.4,-2.3)	7.2 (-2.2, 16.7)
<High School	16.7 (5.6, 27.8)	12.1 (2.5, 21.8)	--	4.3 (-5.7, 14.3)
Unmarried	4.2 (-7.3, 15.6)	--	--	4.9 (-5.0, 14.7)
20-29 years	--	--	--	-8.7 (-19.1, 1.7)
40+ years	--	--	--	3.1 (-7.9, 14.2)

Table 3.4. continued

	Risk Differences (95% Confidence Intervals)			
	Waking at Night	School Absences	Rescue Medication	Exercise Intolerance
UNADJUSTED MODELS				
Intercept***	30.3 (19.9, 40.6)	12.5 (4.9, 20.1)	32.4 (17.4, 47.5)	32.5 (22.0, 42.9)
Middle CE†	12.3, (-0.9, 25.5)	1.2 (-8.4, 10.9)	-6.0 (-24.3, 12.4)	4.9 (-8.3, 18.1)
Low CE†	21.3 (6.9, 35.7)	-3.3 (-13.1, 6.5)	-8.4 (-27.6, 10.7)	3.7 (-10.6, 18.0)
ADJUSTED MODELS†				
Intercept***	21.4 (10.2, 32.6)	16.4 (1.1, 31.7)	28.9 (5.7, 52.1)	29.6 (13.1, 46.0)
Middle CE†	8.1 (-5.0, 21.1)	-0.4 (-11.3, 10.5)	-9.8 (-27.9, 8.3)	3.9 (-8.3, 18.1)
Low CE†	16.7 (2.8, 30.6)	-5.0 (-17.2, 7.2)	-15.7 (-36.1, 4.8)	1.1 (-12.9, 15.1)
Black	13.5 (0.6, 26.4)	-1.6 (-16.9, 13.7)	7.9 (-10.8, 26.6)	10.3 (-4.6, 25.2)
White	-5.5 (-21.6, 10.7)	-6.4 (-24.6, 11.9)	-4.2 (-28.6, 20.3)	6.9 (-11.5, 25.2)
Foreign-born	--	-5.0 (-19.0, 9.0)	-9.1 (-26.9, 8.7)	-4.4 (-17.8, 9.1)
<High School	15.0 (2.9, 27.1)	1.0 (-10.5, 12.5)	--	--
Unmarried	11.5 (-0.8, 23.8)	5.9 (-4.5, 16.4)	11.7 (-4.2, 27.6)	7.1 (-5.2, 19.3)
20-29 years	--	-0.7 (-11.8, 10.4)	15.4 (-3.8, 34.6)	7.6 (-6.7, 21.9)
40+ years	--	1.3 (-10.2, 12.8)	2.9 (-13.2, 19.0)	-7.8 (-20.0, 4.5)

*GH: General health recoded as fair or poor versus excellent or very good or good (referent); Controllability recoded as not at all or somewhat v. quite or extremely controllable (referent); child respiratory outcomes were for a 2-week period and coded as any versus none (referent) except for unplanned medical visits (6 months); rescue medication ncludes diagnosed asthmatics only (n=158)
**Intercept estimate is the risk in the unexposed (not risk difference)
***CE: collective efficacy was the main exposure, modeled with 2 indicator variables; referent=highest CE category
†Adjusted for up to 5 demographic covariates: black/white race (referent=Hispanic); parent age (referent=30-39 years); parent education (referent=high school education or beyond); unmarried (referent=married or cohabiting); and foreign-born (referent=parent U.S.-born)

Table 3.5. Risk differences (excess cases per 100 at risk) and 95% confidence intervals for unadjusted and adjusted binomial regression models of the associations of physical/social order (main exposure) and 8 binary outcomes*

	Risk Differences (95% Confidence Intervals)			
	Parent General Health	Child General Health	Unplanned Medical Visits	Controllability
UNADJUSTED MODELS				
Intercept**	16.7 (9.2, 24.1)	14.6 (7.5, 21.6)	22.1 (13.8, 30.5)	14.9 (7.7, 22.1)
Middle Order***	8.3 (-2.1, 18.8)	-2.8 (-11.7, 6.1)	1.4 (-10.0, 12.4)	7.9 (-2.2, 18.0)
Low Order***	5.3 (-6.4, 16.9)	6.2 (-5.1, 6.1)	6.3 (-6.6, 19.2)	4.6 (-6.6, 15.8)
ADJUSTED MODELS†				
Intercept**	5.2 (-2.7, 13.2)	14.2 (4.1, 21.6)	41.0 (25.7, 56.3)	9.8 (-5.5, 25.1)
Middle Order***	11.4 (2.1, 20.7)	-0.8 (-10.2, 8.6)	-0.8 (-12.5, 10.8)	12.0 (1.8, 22.3)
Low Order***	4.4 (-6.7, 15.5)	6.3 (-0.5, 17.5)	4.5 (-8.5, 17.5)	8.1 (-3.2, 19.4)
Black	--	-8.5 (-19.6, 2.6)	-14.6 (-30.0, 1.2)	-9.1 (-21.9, 3.6)
White	--	-12.3 (-29.2, 4.7)	-18.5 (-35.6, 1.5)	4.8(-11.4, 21.0)
Foreign-born	3.6 (-5.4, 12.7)	--	-21.6 (-36.5, -6.7)	4.2 (-8.4, 16.9)
<High School	20.0 (9.3, 30.7)	11.2 (0.6, 21.9)	--	--
Unmarried	8.1 (-1.4, 17.5)	-1.1 (-10.4, 8.1)	--	8.8 (-1.4, 19.0)
20-29 years	--	1.4(-10.0, 12.8)	--	-6.5 (-16.5, 3.4)
40+ years	--	2.0 (-9.5, 13.4)	--	4.3 (-6.5, 15.0)

Table 3.5. continued

	Risk Differences (95% Confidence Intervals)			
	Waking at Night	School Absences	Rescue Medication	Exercise Intolerance
UNADJUSTED MODELS				
Intercept**	30.5 (21.3, 39.8)	11.8 (5.3, 18.4)	20.8 (9.3, 32.3)	26.3 (17.5, 35.2)
Middle Order***	12.9 (0.4, 25.3)	0.5 (-8.3, 9.2)	5.2 (-10.1, 20.5)	11.5 (-0.6, 23.5)
Low Order***	23.1 (8.9, 37.4)	-1.3 (-10.8, 8.2)	11.5 (-8.0, 31.0)	14.4 (0.5, 28.3)
ADJUSTED MODELS†				
Intercept**	18.6 (8.7, 28.5)	13.8 (1.4, 26.3)	10.7 (-11.4, 32.7)	21.4 (12.0, 30.8)
Middle Order***	11.4 (-0.5, 23.3)	-2.7 (-13.0, 7.7)	5.0 (-11.3, 21.2)	11.6 (-0.3, 23.6)
Low Order***	22.2 (8.6, 35.8)	-1.5 (-10.9, 7.9)	13.9 (-7.3, 35.2)	15.82 (2.1, 29.5)
Black	13.7 (0.8, 26.6)	-0.5 (-14.1, 13.0)	8.7 (-10.2, 27.6)	--
White	-3.2 (-18.9, 12.5)	-5.3 (-20.5, 9.8)	2.8 (-20.6, 26.1)	--
Foreign-born	--	-5.8 (-18.5, 6.8)	-10.7 (-29.3, 7.9)	--
<High School	15.5 (3.6, 27.4)	1.7 (-7.2, 10.5)	2.4 (-14.7,19.5)	--
Unmarried	13.2 (1.0, 25.3)	6.1 (-3.3, 15.4)	11.5 (-3.5, 26.6)	12.6 (1.6, 23.6)
20-29 years	--	--	16.8 (-2.9, 36.6)	--
40+ years	--	--	5.5 (-11.0, 22.1)	--

*GH: General health recoded as fair or poor versus excellent or very good or good (referent); Controllability recoded as not at all or somewhat v. quite or extremely controllable (referent); child respiratory outcomes were for a 2-week period and coded as any versus none (referent) except for unplanned medical visits (6 months); rescue medication includes diagnosed asthmatics only (n=158)
**Intercept estimate is the risk in the unexposed (not risk difference)
***Order was the main exposure, modeled with 2 indicator variables; referent=highest order category
†Adjusted for up to 5 demographic covariates: black/white race (referent=Hispanic); parent age (referent=30-39 years); parent education (referent=high school education or beyond); unmarried (referent=married or cohabiting); and foreign-born (referent=parent U.S.-born)

Regression Analyses

Analytic models included two indicator variables for each exposure, with the most favorable category serving as the referent group (*i.e.* highest collective efficacy and highest order), allowing two exposure group contrasts: middle versus high and low versus high. Given the high prevalence of all outcomes (12-43%) and the limitations of odds ratios to estimate relative risk, we estimated absolute measures of effect, RDs. Binomial regression (SAS GENMOD specifying identity link function and binomial distribution) estimated RDs and 95% CIs. Table 5.3 presents unadjusted and adjusted RDs for both main exposures with all covariates at their referent levels.

CHAPTER 4
AIM 1 RESULTS

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

Abstract

Objective. Asthma's social patterning is not well-explained. We hypothesize that disadvantaged populations experience housing stressors (HS) which produce psychological stress and impact health through physiologic and behavioral pathways. **Methods.** We examined 8 respiratory and general health outcomes with data from 682 low-income, Chicago parents of asthmatic children. We created a weighted exposure representing HS number/difficulty and compared 75th to 25th HS quartiles in adjusted binomial and negative binomial regression models. **Results.** Higher risks/rates were associated with higher HS for 6 unfavorable outcomes. Risk difference (RD) for poor/fair general health was larger for children [RD=6.28 (95% CI: 1.22, 11.35)] than parents [RD=3.88 (95% CI: -1.87, 9.63)]. Incidence rate difference (IRD) for exercise intolerance [IRD=0.88 (95% CI: 0.41, 1.35)] was nearly one extra day/2-weeks for the higher exposure group and almost one-third extra day 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)]. **Conclusions.** Results contribute to the conceptualization of stress as a "social pollutant" and to hypothesized stress-asthma pathways among disadvantaged populations. Interventions must address individuals' reactions to stress while structural solutions to stressors are sought.

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).[2, 4, 5] Chicago's asthma prevalence and morbidity and mortality rates are among the highest in the nation, and within the city, they are typically highest in neighborhoods with the lowest SES.[6, 7, 9, 11-13] 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.[1, 6, 16, 53] 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.[1]

Psychological stress has been conceptualized as a "social pollutant that when 'breathed into the body' may disrupt biological systems through inflammatory processes."[15, 16] Evidence demonstrates that acute and chronic stressors have psychological effects that influence psychologic and physiologic functioning as well as behavior.[17] Studies link stress to asthma onset, exacerbation and phenotypes through hypothesized "dysregulated immunity" mechanisms.[15, 16, 18-26] Stress experienced by children or their parents may also have indirect effects on asthma by causing health-compromising behaviors and co-morbidities that adversely affect disease management.[27-38] 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.[18, 19, 24-26, 33-35, 37, 39, 85, 87, 93, 95, 96, 175]

The housing and health relationship has long been acknowledged, not surprising given that water, warmth, air, shelter and safety are fundamental human needs.[51] U.S. children spend as much as 80-90% of their time indoors.[52] 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.[39-41, 43] 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.[40, 43-48] Just as material exposures act on biology, social environments may "get under the skin"[49] and become "biologically embedded"[50] to influence health.[1, 16] 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.[129] We hypothesize that issues involving housing security, mobility, comfort, safety, finances, dynamic household membership, and relationships with neighbors and landlords cause stress, which in turn 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 public 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 asthma, 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 parent-reported parent and child general health and child respiratory 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 study surveyed for respiratory problems with a validated tool[149, 162] 15 public elementary schools that met the following eligibility criteria: 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. Eligible families (n=1244) participated in a longitudinal study with 3 data collection phases over 12 months in English or Spanish. The current study used data from parents of children with diagnosed asthma (n=351) and undiagnosed (possible) asthma (n=331) obtained during the baseline telephone survey; 562 children with no diagnosis and no symptoms were excluded from this analysis.

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, 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;[166] 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[167] 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 4.1); broader neighborhood conditions and events were excluded. 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. We recoded 3 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 rated on a 5-response scale and recoded as poor or fair vs. good or very good or excellent. Controllability of child's asthma was a 4-response variable 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.

Socio-demographic 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), 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 4.2 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 9 (of 13 possible). For the 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 4.3). Adjusted estimates are discussed below and graphically represented in figures 4.1 and 4.2. Table 4.3 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, 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 controllability changed little with adjustment, was of relatively large magnitude, and the CI did not contain the null value of 0 [RD=6.19 (95% CI: 0.85, 11.54)].

Count outcomes. Exercise intolerance was most strongly associated 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."[15, 20] 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.[158, 159] and may influence problem-solving and perceived control.[160] There is evidence that parent stress may produce stress responses in children and cue children to adopt suboptimal coping

strategies.[35] 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.[157] Stress may lead to health-harming coping strategies such as smoking, overeating and being sedentary.

The distinction between "traditional" environmental 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.[16, 176] While community groups, clinicians, researchers and politicians 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 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 3 of 4 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 most strongly associated with housing stressors. 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. For parent general health, the RD was of moderate magnitude though the CI contained the null value of 0. The association for child general health was stronger. 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.[28] 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 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.[8, 57] 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 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.

"Environmental" 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.[16, 120, 176, 177] 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.[178] Future research must address psychological factors over the lifecourse, the relative importance of chronic and acute stressors,[99] the possibility that the asthma phenotype is programmed before birth,[21] and reverse causality, since stress and consequent problems may be caused or aggravated by having asthma or caring for someone with asthma.[99, 179-181] 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 mediators or moderators of the effects of low-SES on respiratory health. Mediators, in fact, are not expected to explain additional variance in health outcomes. Moderators, however, would help explain differential outcomes among

places and populations that share low-SES and environmental 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.

Table 4.1. Descriptive statistics for parent-reported housing stressors in the previous 6 months and their difficulty level used to create weighted main exposure (n=682)

Housing Stressor	Parents Reporting Event N (%)	Parent-reported Difficulty* Mean (SD)**
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)

*Parents who reported that the event occurred in the previous 6 months rated its difficulty on a 4-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
**SD: standard deviation

Table 4.2. Descriptive statistics for parents and children in study population, main exposure, and health outcomes (n=682)

Sociodemographics	Number (Percent)			
Race/ethnicity*				
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**	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***	216 (31.7)			
Child health insurance				
Public	321 (47.2)			
Private	273 (40.1)			
Uninsured	86 (12.6)			
Asthma diagnosis				
Child[+]	351 (51.5)			
Parent	106 (15.6)			
Smokers in household				
Parent	180 (26.4)			
Any smoker	290 (42.5)			
	Mean	Med[++]	SD[++]	Min-Max[++]
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 [+++]	14.7	12.0	11.3	0.0-55.0
Main Exposure				
Housing stressors (continuous, untransformed)				
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[#]				
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[##]	2.2	0.0	3.8	0.0-14.0
Binary Health Outcomes[###]	Number (Percent)			

Fair/poor general health	
Parent	111 (16.3)
Child	158 (23.2)
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[##]	165 (47.4)

*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

**TANF: Temporary Assistance for Needy Families

***Overcrowding defined as unit occupied by 1.01 persons or more per room excluding bathrooms

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

[++]Med: median; SD: standard deviation; min: minimum value; max: maximum value

[++ +]CES-D: Center for Epidemiologic Studies-Depression; possible score ranges from 0-60

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

[##]Includes diagnosed asthmatics only; n=351

[###]General health was a 5-response item recoded as fair or poor versus excellent or very good or good (referent); controllability was a 4-response item recoded as not at all or somewhat vs. quite or extremely controllable (referent); no reference time periods specified; 5 other outcomes coded as continuous variables in models; any occurrence vs. none frequencies presented for descriptive purposes

Table 4.3. Crude and adjusted risk and incidence rate differences and 95% confidence intervals for the associations of increased housing stressors (75th v. 25th percentile) with parent and child general and child respiratory health (n=682)

BINARY OUTCOMES***	Crude RD (95% CI)*	Adjusted** RD (95% CI)*
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†	Crude IRD (95% CI)*	Adjusted** IRD (95% CI)*
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	0.44 (0.19, 0.69)	0.30 (0.059, 0.54)
School absences	0.03 (0.12, 0.19)	-0.03 (-0.24, 0.19)
Rescue medication use††	0.09 (-0.62, 0.79)	0.09 (-0.61, 0.80)

* RD: risk difference; IRD: incidence rate difference; CI: confidence interval

**Adjusted for up to 8 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

*** 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); General health were recoded as fair or poor versus excellent or very good or good (referent); controllability was recoded as not at all or somewhat v. quite or extremely controllable (referent); no reference time periods specified

†IRD is the excess number of days the unfavorable outcome occurred per 2 weeks associated with increased housing stressors (75th v. 25th percentile of exposure); count outcomes were coded as continuous variables for a 2-week period except for unplanned medical visits for which IRD is the excess number of days per 6 months

††Includes diagnosed asthmatics only; n=351

Figure 4.1. Associations of housing stressors and binary health outcomes (n=682)

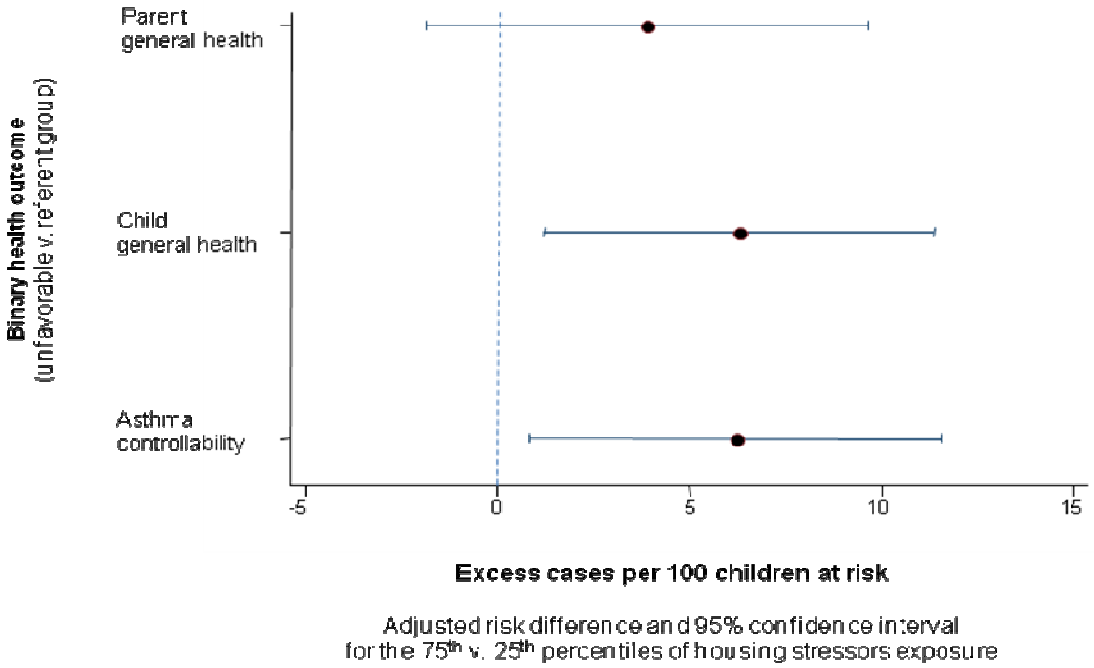
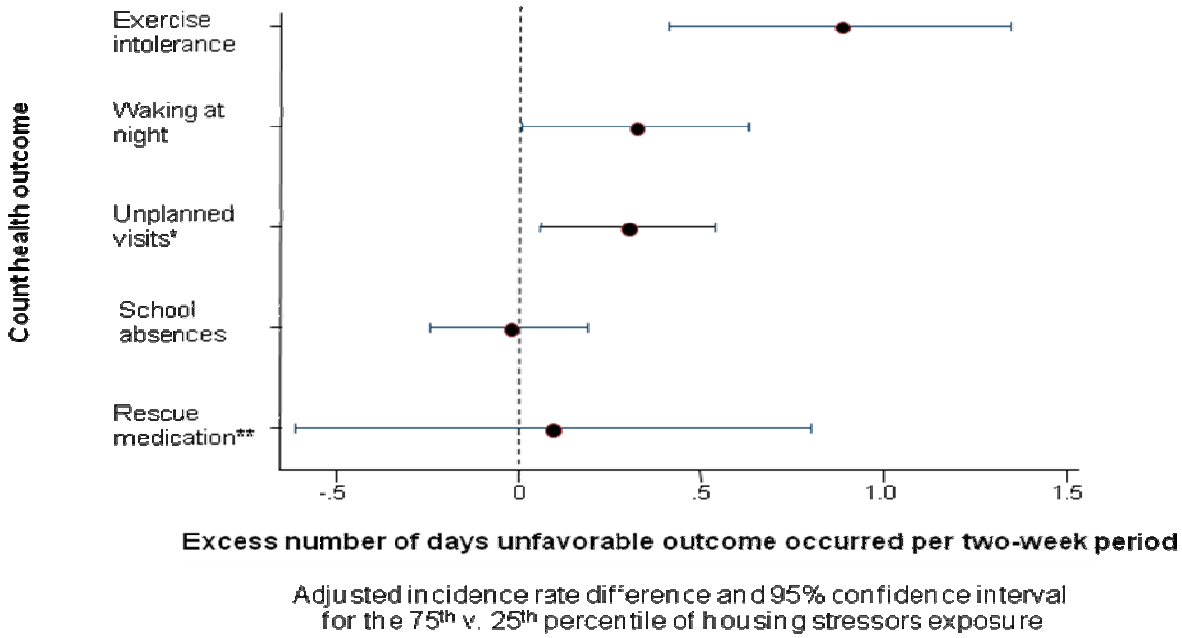


Figure 4.2. Associations of housing stressors and respiratory outcomes (n=682)



*six-month period
**n=351 diagnosed asthmatics

CHAPTER 5
AIM 2 RESULTS

Parent Perceptions of Neighborhood Collective Efficacy and Order are Associated with General Health and Child Respiratory Health among Low-income, Urban Families

Abstract

Background*.* This cross-sectional study examines parents' perceptions of their neighborhoods and general and respiratory health among low-income Chicago families. Asthma disproportionately affects non-white, urban, and low socioeconomic status (SES) populations, but Chicago's burden, and the national epidemic, are not well-explained by known risk factors. Urban dwellers experience acute and chronic stressors that produce psychological distress and are hypothesized to impact health through biological and behavioral pathways. Identifying factors that covary with lower SES and minority-group status -- e.g. stress -- is important for understanding asthma's social patterning. ***Methods.*** We used survey data from 319 parents of children 5-13 years with asthma/respiratory problems and principal components analysis to create exposure variables representing parents' perceptions of two aspects of neighborhoods: collective efficacy ("CE") and physical/social order ("order"). Adjusted binomial regression models estimated risk differences (RD) and 95% confidence intervals (CI) for eight binary outcomes. ***Results.*** Magnitude was generally as expected, i.e., RD for low versus high (most favorable) exposure groups ($RD_{low\ v.\ high}$) was larger than for the middle vs. high contrast ($RD_{mid\ v.\ high}$). "Parent general health" was strongly associated with "CE" [$RD_{low\ v.\ high}$=20.8 (95% CI: 7.8, 33.9)] and "order" [$RD_{mid\ v.\ high}$=11.4 (95% CI: 2.1, 20.7)] unlike "child general health" which had nearly null associations. Among respiratory outcomes, only "waking at night" was

strongly associated with "CE" [$RD_{low\ v.\ high}$=16.7 (95% CI: 2.8, 30.6)] and "order" [$RD_{low\ v.\ high}$=22.2 (95% CI: 8.6, 35.8)]. "Exercise intolerance" [$RD_{low\ v.\ high}$=15.8 (95% CI: 2.1, 29.5)] and "controllability" [$RD_{mid\ v.\ high}$=12.0 (95% CI: 1.8, 22.3)] were moderately associated with "order" but not with "CE," while "school absences," "rescue medication use," and "unplanned visits" had nearly null associations with both exposures. ***Conclusions.*** More negative perceptions tended to be associated with higher risk of undesirable outcomes, adding to evidence that the social environment contributes to health and supporting research on stress' health impact among disadvantaged populations. Interventions must address not only "traditional" environmental factors but individuals' reactions to stress and attempt to mitigate effects of stressors while structural solutions to health inequities are sought.

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).[2, 4, 5] Chicago's asthma mortality and hospitalization rates are among the highest in the nation.[6, 7, 11, 12] Underdiagnosis, suboptimal care, and dramatic racial/ethnic disparities among Chicagoans have been documented.[6-10, 57, 76] Within Chicago, prevalence, morbidity, and mortality are typically highest in neighborhoods with the lowest SES.[6, 9, 13] Despite a decade of extraordinary efforts to increase asthma equity in Chicago, progress has been modest.[13]

Chicago's burden, and the national epidemic, are not well-explained by known risk factors. Not all urban communities have excess asthma though they may share low-SES and environmental exposures with high-risk urban areas.[1, 6, 16, 53] 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.[1]

Recent literature has also called for understanding how social environments "get under the skin"[49] and become "biologically embedded"[50] to influence health.[1, 16] Acute and chronic stressors are believed to have psychological effects that in turn influence psychologic and physiologic functioning as well as behavior. Low-SES and other disadvantaged groups may experience increased stressors and be more strongly affected by them due to already-compromised psychological health, social supports, and coping resources.[44, 47, 48] Studies link psychological stress to asthma, including onset of disease, precedent phenotypes, and disease exacerbation, through hypothesized "dysregulated immunity" mechanisms.[15, 16, 20, 23-26, 85] Psychological stress experienced by children or their parents may also have indirect effects on asthma by causing health-compromising behaviors and co-morbidities that compromise disease management.[27-30, 85, 85] In general, individual perceptions are important because they are related to psychological distress. Neighborhoods matter because families reside in environments which may impact their physical and mental health.

While an enormous asthma literature exists, the alarming burden of asthma on inner-city populations demands more thoughtful investigation of determinants of risk. This study 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 psychological stress pathways. We examined associations between parents' perceptions of neighborhood stressors and parent-reported parent and child general health and child respiratory health under the hypothesis that less positive perceptions of one's neighborhood would be associated with increased risk of poor health.

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 and survey data collected in 2002-2004 for an observational investigation of childhood asthma disparities among low-income Chicago families with children aged 5-13 years. The study surveyed for respiratory problems with a validated tool[162, 163] in 15 public elementary schools that met the following eligibility criteria: 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. Eligible families participated in a longitudinal study with three data collection phases over 12 months in English or Spanish and comprised three groups: 351 diagnosed asthmatics; 331 undiagnosed (possible) asthmatics; and 562 non-asthmatics (no diagnosis and no respiratory problems). The current study used data from parents of children with diagnosed asthma (n=158) and undiagnosed asthma (n=161) obtained during a home visit (phase 2) since the focus of our hypothesis is exacerbation of respiratory problems, not the development of asthma.. Also, non-asthmatics did not participate after baseline and therefore are not included in the phase 2 dataset (and neighborhood perceptions items were not included in the baseline survey). Descriptive statistics demonstrate that 47% of those who participated at baseline also participated in the home visit and that this subsample is similar to the main study sample for all sociodemographic variables examined, with three exceptions. The current study sample had a higher proportion of parents who were foreign-born (44% vs. 34%), Spanish speakers (34% vs. 23%), and married/cohabiting (64% vs. 57%). The current sample had a lower proportion of children with the unfavorable outcome

for three respiratory outcomes: unplanned medical visits (27% vs. 44%); school absences (12% vs. 18%); and rescue medication use (27% vs. 47%).

Analytic strategy

Principal Components Analysis (PCA)

We created summary variables representing neighborhoods by data reduction techniques to take advantage of rich survey data and to capture multi-faceted characteristics of neighborhoods. We identified 27 items representing parent perceptions of their neighborhood in the survey instrument, 14 of which were taken from the Community Survey from the Project on Human Development in Chicago Neighborhoods (PHDCN),[182] though no attempt was made to replicate PHDCN's survey nor was there an expectation that PCA for this study's sample would yield scales identical to those used in PHDCN research. Thirteen additional survey items were developed by the main study's investigators and hypothesized to have potential health effects. We dropped 13 variables not correlated with at least one other item at the level of 0.50 from further analysis. We used an iterated PCA of 14 remaining variables for the extraction method in the absence of *a priori* theoretical knowledge of underlying constructs or of shared variance among variables. Varimax (orthogonal) rotation summarized the co-variation among the variables since the goal was data reduction to a set of uncorrelated measures for subsequent use in multivariate analyses. A three-component solution was supported by scree plot and eigenvalues. Twelve items comprised 3 components which explained 58% of the total variance (table 5.2). Components1, 2 and 3 accounted for 28%, 16% and 14% of the total variance, respectively. Interpretation of components was limited to theoretically salient variables with loadings >0.60 and no loading >0.30 on the other components. We computed summary scores by obtaining the mean value of these variables, all of which were on the same metric within each component, allowing a maximum of one missing item's value to be replaced by

the mean of the non-missing items. Summary scores were named: 1) physical/social order; 2) collective efficacy; and 3) recent change in neighborhood (hereafter referred to as "order," "collective efficacy," and "change"). "Change" was not explored in regression analyses because interpretation of associations would be difficult in the absence of baseline data on the neighborhood at the start of the 5-year period and whether change was desirable. Also, it comprised only 3 items, and allowing substitution of missing data would compromise its validity. Internal consistency, measured by Cronbach's alpha, was above acceptable[174] for both summary scores chosen as main exposures: 0.78 ("collective efficacy") and 0.83 ("order").

Variables

Neighborhood Exposures. Summary scores resulting from PCA were each categorized into 3 levels based on natural cut-points in the distributions and to assure sufficient numbers in each category. Table 5.1 presents descriptive statistics for the summary scores as well as the categorical exposure variables. "Collective efficacy" was coded as: high (1-1.9); middle (2.0-2.4); and low (2.5-3.8). "Order" was coded as: high (1.0); middle (1.1-1.9); and low (2.0-3.0). Analytic models included 2 indicator variables for each exposure, with the most favorable (hypothesized to be health-protective) category serving as referent (*i.e.* high collective efficacy and high order), allowing 2 exposure group contrasts: middle versus high and low versus high.

Health Outcomes. We recoded 8 parent-reported health outcomes as binary variables with the absence of symptoms/healthcare or medication utilization/school absences or the most favorable outcome (for general health and controllability) serving as the referent category*, i.e.,* outcomes were modeled as any versus none or unfavorable versus favorable . We used the global/general health items from the Child Health Questionnaire[165] and the Short Form-12,[164] to measure parent-reported child health and

self-reported parent health, respectively. We recoded the 5-response scale as poor or fair vs. good or very good or excellent. Respiratory outcomes were developed by the main study's investigators, including a pediatric allergist, and based on their clinical and research expertise and review of the literature. A number of measures of asthma symptoms, therapies and control for children and adults exist, though time reference periods differ.[183-185] We used the following four commonly-utilized respiratory outcomes with a two-week reporting period to minimize recall bias: 1) waking at night; 2) school absences; 3) rescue medication use, and 4) exercise intolerance, recoded as any versus none. Analyses using the rescue medication outcome included only diagnosed asthmatics since undiagnosed children might not have access to prescribed asthma medications. The number of unplanned visits to an emergency department, physician's office or clinic for child's asthma or breathing problems was obtained for a six-month period since urgent care is not typically a common occurrence and coded as any vs. none. Controllability was intended to be a subjective measure of a parent's sense of whether her child's asthma is controllable; this 4-response variable was recoded as not at all or somewhat vs. quite or extremely controllable.

Individual-level sociodemographic variables. Covariates included: parent education (less than high school versus. high school or beyond (referent)); parent marital status (unmarried versus married or cohabiting partner (referent)); parent nativity (foreign-born versus. U.S,-born (referent); parent age (20-29, 30-39 (referent), >=40 years); and child race/ethnicity (Hispanic (referent), non-Hispanic black, non-Hispanic white). Parent race/ethnicity was not ascertained in the main study; child race/ethnicity is considered a proxy. Five non-Hispanic, non-Hispanic black, "other" cases were categorized as white. The parent age range in table 5.1 highlights the fact that nine participants (2.8%) were grandmothers. Three participants (0.9%) were aunts.

Binomial Regression Analyses

Univariate analyses demonstrated high prevalence of most outcomes (12-43%); therefore, odds ratios would overestimate relative risk. We preferred an absolute measure of effect, risk difference (RD). Binomial regression (SAS GENMOD specifying identity link function and binomial distribution) estimated RDs and 95% confidence intervals (CI) as the measures of association between the summary neighborhood scores and binary health outcomes for cases with complete data. Bivariate analyses first estimated crude RDs. We assessed effect measure modification (EMM) by Mantel-Haenszel chi-squared tests of homogeneity and did not observe consistent evidence of modification for variables including child asthma diagnosis, race, age and sex and parent age, nativity, marital status, education, and depressive symptoms score.

With the same variables assessed as potential modifiers, we next identified potential confounders in directed acyclic graph (DAG) analyses;[166] therefore, we did not quantitatively assess variables on causal pathways, variables that were not associated with both outcome and exposure, or variables that had hypothesized bi-directional associations with other variables as confounders. We then assessed all potential confounders with a change-in-estimate strategy[167] 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%. Each model therefore had a potential unique set of adjustment variables. We evaluated the predictive importance of each exposure by the magnitude of the RDs and the width of the CIs.

We used SPSS 16.0 (SPSS Inc., Chicago, Illinois) for data reduction by PCA, SAS 9.1 (SAS Institute Inc., Cary, North Carolina) for estimation of risk differences by binomial regression, STATA 10.1 (StataCorp, College Station, Texas) for graphical data displays, and

the Microsoft Excel program “Episheet” (version of June 11, 2008) written by Ken J. Rothman for additional tabular analyses.

Results

Sample characteristics

The following descriptive statistics are presented in table 5.1. More of than half of children were identified as Hispanic while, about one-third were non-Hispanic black and 12% were non-Hispanic white. Only 10% of children but 44% of parents were foreign-born, and about 1/3 spoke Spanish for the survey. Approximately equal numbers of boys and girls participated, but the majority of parent respondents were female. Twenty-eight percent of parents had not earned a high school diploma; 64% were married or cohabiting, and 1/3 of families owned their homes. Most children (88%) and parents (72%) had health insurance. The mean Center for Epidemiologic Studies-Depression (CES-D) score was 12 (SD=11), range=0-54. Validation studies support a score of 16 to discriminate individuals with depressive symptomatology from those without.[170, 171] Parent-reported general health was fair or poor for 21% of parents and 15% of children. Prevalence of undesirable child respiratory outcomes was high. One quarter had at least 1 unplanned medical visit. Night disturbance had the highest prevalence (43%) and school absences had the lowest (12%) while rescue medication use and exercise intolerance were experienced by 27% and 35%, respectively. Twenty percent of parents reported their child’s asthma/breathing problems as not at all or somewhat controllable.

Risk differences

Adjustment did not change the magnitude of the RDs dramatically from the crude RDs (tables 5.3 and 5.4); adjusted RDs are discussed below and graphically represented in figures 5.1 and 5.2. Adjustment caused about half of the RDs to move closer to the null value of 0 and about half to move away. Generally, the magnitude of the RDs for each

outcome was as expected; that is, the RD for the middle versus high contrast ($RD_{mid\ v.\ high}$) was smaller than for the low vs. high contrast ($RD_{low\ v.\ high}$).

Collective efficacy

The association of general health and "collective efficacy" was strong for parents [$RD_{mid\ v.\ high}$ =13.5 (95% CI: 2.3, 24.6); $RD_{low\ v.\ high}$ =20.8 (95% CI: 7.8, 33.9)] but not for children. (table 5.3). "Waking at night" was strongly associated with "collective efficacy" yielding $RD_{low\ v.\ high}$ approximately twice as large as $RD_{mid\ v.\ high}$: [$RD_{mid\ v.\ high}$ =8.1 (95% CI: -5.0, 21.1); $RD_{low\ v.\ high}$ =16.7 (95% CI: 2.8, 30.6)] (table 5.4).

Physical/social order

. For the association of "order" with parent health, $RD_{mid\ v.\ high}$ was unexpectedly larger [11.4 (95% CI: 2.1, 20.7)] than $RD_{low\ vs.\ high}$ [4.4 (95% CI: -6.7, 15.5)], while for child health, estimates for both contrasts were nearly null (table 5.3). "Waking at night" had a strong association with "order," again yielding $RD_{low\ v.\ high}$ approximately twice as large as $RD_{mid\ v.\ high}$: [$RD_{mid\ v.\ high}$ =11.4 (95% CI: -0.5, 23.3); $RD_{low\ v.\ high}$ =22.2 (95% CI: 8.6, 35.8)] (table 5.4). Exercise intolerance [$RD_{low\ v.\ high}$ =15.8 (95% CI: 2.1, 29.5)] and controllability [$RD_{mid\ v.\ high}$ (12.0 (95% CI: 1.8, 22.3)] had moderately strong associations with "order" (table 5.4).

Discussion

Overall, results supported the study's hypothesis; exposure levels reflecting more negative neighborhood perceptions tended to be associated with higher risk of undesirable general and respiratory health outcomes. While perceptions are subjective and do not necessarily reflect actual neighborhood characteristics, they may be important proxies of the psychological burden of stressors on individuals.[186] This study adds to growing evidence that the social environment, in addition to the physical environment, contributes to asthma burden in urban areas. Specifically, this study furthers the conceptualization of

psychological stress as a "social pollutant" that may be "breathed" into the body.[15] Acute and chronic stressors experienced by low-income, urban dwellers may impact health through psychological stress pathways, and the experience of psychological distress may be influenced by individuals' perceptions. Implications of these findings are different than for "conventional" risk factors, that is, interventions must address not only "bricks and mortar" but individuals' reactions to stress and attempt to mitigate the effects of stressors while structural solutions to health inequities are sought.

Low-SES populations, such as in the current study, may have increased vulnerability to respiratory disease because of increased exposure to acute and chronic stressors which cause psychological stress and its sequelae.[28, 44, 47, 48, 177] Single parenting may add to the burden of such circumstances.[121] Exposure to violence, problematic family relationships, parenting difficulties, caregiver stress, critical attitudes of one's mother, and negative life events have been related to wheeze, asthma onset and/or adverse asthma outcomes among infants and youths.[24-26, 34, 35, 85, 93, 96, 175] Psychological stress experienced by parents of children with asthma may lead to impaired problem solving, influence reporting of symptoms, quality of life, and perceptions of asthma outcomes, and allow suboptimal disease management and healthcare utilization.[27, 29, 34, 35]

A study of neighborhood-level variation in asthma and respiratory diseases in Chicago found that collective efficacy, but not disorder (observable physical and social decay), was protective.[53] The authors hypothesized that collective efficacy may protect against respiratory diseases through: 1) social control of health-compromising behaviors; 2) access to health services; 3) management of physical hazards; and 4) promotion of psychosocial health by minimizing fear of being outside and engaging with community. The study differs from the current study by investigating neighborhood- rather than individual-

level exposures; however, parents' perceptions may indeed be correlated with contextual factors such as collective efficacy and work through similar mechanisms to affect health.

Not all associations were strong, and results differed somewhat for "collective efficacy" and "order." Associations of parents' perceptions with their own general health were stronger than with their children's health. The influence of psychological stress through physiologic mechanisms (directly affecting a parent's health) may be stronger than through behavioral mechanisms (indirectly impacting disease management for their child). It is also possible that parents reported their children's health less accurately than their own, obscuring associations.

Whereas general health outcomes were more strongly associated with "collective efficacy," child respiratory outcomes tended to be more strongly associated with "order." The more material nature of the variables comprising "order" compared to the more interpersonal variables in "collective efficacy" may be correlated with asthma triggers (i.e. cockroaches, mold) that make disease management less predictable. Such triggers may be most common outside where exercise intolerance is likely to occur or rescue medication is needed because children are more active outdoors. The hypothesized direct effect of "collective efficacy" through physiologic pathways may be weak for children, who are typically less concerned with neighborhood affairs than adults.

"Waking at night" was the only respiratory outcome strongly associated with both exposures, and for both, the contrast of the least favorable exposure group and the most favorable group produced RDs twice as big as for the middle group. "Waking at night" is a relatively objective outcome, often used in surveys of asthma control and quality of life, (though waking from factors other than breathing difficulties, such as noise, may have been reported). Misclassification may have biased RDs toward the null. Other outcomes are more subjective, involve health behaviors and attitudes, and may not accurately reflect

symptom frequency/severity. It is unlikely that all children consistently reported symptoms that occurred while away from their parents. Associations of "school absences," "rescue medication use" and "unplanned medical visits" with both neighborhood exposures were null. The current sample compared to the main study's sample had a lower proportion of children with the unfavorable outcome for all three of these outcomes. Further, these outcomes depend not only on disease activity but on resources and health behaviors. The current sample had a higher proportion of foreign-born and Spanish-speaking parents; access to care and cultural health beliefs and practices may have caused underestimates in outcomes. Undiagnosed children were excluded from analyses with "rescue medication use" as the outcome, leading to reduced power. Bias might have resulted from the self-report nature of both exposures and outcomes. Coding outcomes as binary rather than ordinal variables should have minimized the effects of reporting bias.

The study sample was low-income by definition given the recruitment strategy of the main study, thereby minimizing confounding by SES. Parent education was included as a covariate to further control for confounding by SES. The main study's respiratory survey captured 90% of the schools' enrollment (n=12,699), thereby adding to the generalizability of results. The baseline survey achieved a 64% response rate, and survey 2, which provided the data for the current analysis, had a 47% response rate. Selection bias may have influenced results.

We used exploratory factor analysis with unique data not collected in large-scale health studies to create multidimensional measures of perceptions of neighborhoods robust to problems with single variables, for example, influences of secular, geographic or seasonal trends. This strategy summarized the relationships within a collection of public policy-relevant variables among a low-income, urban, racially/ethnically heterogeneous sample of parents of young children, allowing us to address the complex circumstances of low-income,

urban families that impact onset and expression of asthma, Summary scores, rather than PCA factor loadings, were used to represent exposures since the component variables were untested and exploratory, with no evidence of reliability or validity; summary scores also preserved the variation in the data, beneficial for their subsequent use in multivariate analyses.[173]

Assessment of EMM informed the decision not to stratify analyses based on asthma diagnosis status (though undiagnosed children were excluded *a priori* from analyses of rescue medication use). RDs tended to be imprecise due to small sample size; stratification or interaction terms in models would have further compromised precision. Parents of undiagnosed children may have been less aware of symptoms, resulting in outcome misclassification. Nonetheless, interventions to address health-harming neighborhood 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.[8, 57] 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.

Asthma etiology is complex. This study, cross-sectional in design and limited to one city, did not incorporate all known risk factors or test causation. “Traditional,” “environmental” risk factors (e.g., mold and cockroaches), have been well-documented in the biomedical literature and were not the focus of this study. Biological (e.g., cytokines and cortisol) and behavioral (e.g., smoking and allergen reduction in homes) measures are hypothesized to be on causal pathways and were not included as covariates. Future research must address psychological factors over the lifecourse, the possibility that the asthma phenotype is programmed before birth, and reverse causality, since stress and consequent problems may be caused or aggravated by having asthma or caring for someone with asthma.[99] In

addition to an ecological perspective, longitudinal data and a multi-level approach are required to understand structural forces that influence the distribution of neighborhood stressors -- and as a result, psychological stress.

These findings emphasize the importance of addressing not only neighborhood-level mediators of the effects of low-SES neighborhoods but residents' *sense* of their neighborhoods. Mediators, in fact, are not expected to explain additional variance in asthma outcomes. However, all relevant risk factors must be identified if we are to understand causal mechanisms. Psychological stress may be a crucial determinant of the burden of asthma and other illnesses experienced by urban populations. Sociodemographic factors and health outcomes 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.

Table 5.1. Neighborhood factors derived from principal components analysis

Survey Item	Item Loadings**		
	Component 1 Physical/ social order	Component 2 Collective efficacy	Component 3 Change in past 5 years
I'm going to read a list of things that are problems in some neighborhoods. For each, please tell me how much of a problem it is in your neighborhood.			
How much of a problem is litter, broken glass or trash on the sidewalks and streets?	**.811**	-.024	.087
How much of a problem is graffiti on buildings and walls?	**.792**	.064	.036
How much of a problem are vacant or deserted houses or storefronts?	**.784**	.133	.102
How much of a problem is lack of trust between local businesses and residents?	**.730**	.046	.141
How often does child play in doors instead of outdoors because of the following?			
A hazardous environment, for example, traffic, broken glass, broken playground equipment, the presence of garbage or syringes?	.571	.171	-.097
Danger caused by people, for example, violence, crime, gang or drug activity?	.516	.308	-.150
Do you strongly disagee, disagree, agree or strongly agree?			
People around here are willing to help their neighbors.	.128	**.754**	.089
This is a close-knit neighborhood.	.135	**.749**	.070
Most of my neighbors vote regularly.	-.013	**.722**	.024
People in this neighborhood can be trusted.	.229	**.693**	.083
People in this neighborhood care about who is elected to local political positions.	.055	**.686**	.061
Now I'm going to ask you about how your neighborhood has changed over the past years (even if you have not lived here the entire time). Please tell me whether you think your neighborhood has gotten better, stayed about the same, or gotten worse over the past five years.			
Personal safety	.041	.121	**.857**
The way the neighborhood looks	.050	.043	**.853**
People living in your neighborhood	.018	.098	**.845**

*Iterated extraction method and varimax (orthogonal) rotation used to obtain uncorrelated components; 3-component solution accounted for 58% of total variance
**Bolded items loading > 0.60 on one component and not crossloading >0.30 on a second component were included in summary scores

Table 5.2. Crude and adjusted risk differences (RD) and 95% confidence intervals (CI) for the associations of neighborhood factors and fair/poor parent and child general health (n=319)

General Health Outcome* by Neighborhood Exposure Level**	Excess Number of Persons per 100 at Risk of Unfavorable Outcome	
	Crude RD (95% CI)	Adjusted RD (95% CI)
COLLECTIVE EFFICACY		
Self-reported parent general health		
middle vs. high	14.9 (5.8, 24.1)	13.5 (2.3, 24.6)***
low vs. high	22.7 (11.7-33.8)	20.8 (7.8, 33.9)***
Parent-reported child general health		
middle vs. high	2.5 (-6.7, 11.7)	2.5 (-6.1, 11.1)†
low vs. high	6.2 (-4.3, 16.7)	7.5 (-2.5, 17.6)†
PHYSICAL/SOCIAL ORDER		
Self-reported parent general health		
middle vs. high	8.3 (-2.1, 18.8)	11.4 (2.1, 20.7)††
low vs. high	5.3 (-6.4, 16.9)	4.4 (-6.7, 15.5) ††
Parent-reported child general health		
middle vs. high	-2.8 (-11.7, 6.1)	-0.8 (-10.2, 8.6)†††
low vs. high	6.2 (-5.1, 17.4)	6.3 (-5.0, 17.5)†††

*General health recoded as fair or poor vs. excellent or very good or good (referent); no reference time period specified

**3-level neighborhood exposures yielded 2 RDs; high (most favorable) level= referent

***Adjusted for child race/ethnicity, parent education, and parent marital status

†Adjusted for parent education

††Adjusted for parent education, parent marital status, and parent nativity

†††Adjusted for child race/ethnicity, parent education, parent marital status, and parent age

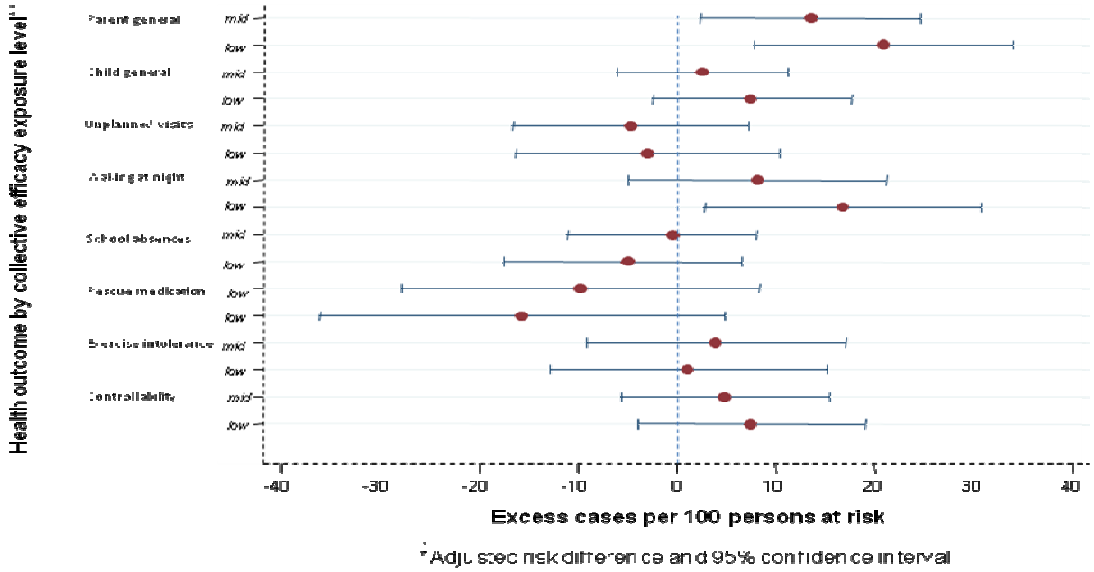
Table 5.3. Crude and adjusted risk differences (RD) and 95% confidence intervals (CI) for the associations of neighborhood factors and unfavorable parent-reported child respiratory health (n=682)

Child Respiratory Health Outcome* by Neighborhood Exposure Level**	Excess Number of Persons per 100 at Risk of Unfavorable Outcome	
	Crude RD (95% CI)	Adjusted RD (95% CI)
COLLECTIVE EFFICACY		
Waking at night		
middle vs. high	12.3 (-0.9, 25.5)	8.1 (-5.0, 21.1)***
low vs. high	21.3 (6.9, 35.7)	16.7 (2.8, 30.6)***
Exercise intolerance		
middle vs. high	4.9 (-8.3, 18.1)	3.9 (-9.2, 17.1)†
low vs. high	3.7 (-10.6, 18.0)	1.1 (-12.9, 15.1)†
School absences		
middle vs. high	1.2 (-8.4, 10.9)	-0.4 (-11.1, 8.0)††
low vs. high	-3.3 (-13.1, 6.5)	-5.0 (-17.6, 6.5)††
Rescue medication use		
middle vs. high	-6.0 (-24.3, 12.4)	-9.8 (-27.9, 8.3)†††
low vs. high	-8.4 (-27.6, 10.7)	-15.7 (-36.1, 4.8)†††
Unplanned medical visits		
middle vs. high	-4.9 (-17.1, 7.3)	-4.7 (-16.6, 7.2)#
low vs. high	-1.0 (-14.5, 12.4)	-3.0 (-16.3, 10.3)#
Controllability		
middle vs. high	4.6 (-6.1, 15.2)	4.8 (-5.7, 15.5)##
low vs. high	4.0 (-7.6, 15.6)	7.5 (-4.0, 19.0)##
PHYSICAL/SOCIAL ORDER		
Waking at night		
middle vs. high	12.9 (0.4, 25.3)	11.4 (-0.5, 23.3)###
low vs. high	23.1 (8.9, 37.4)	22.2 (8.6, 35.8) ###
Exercise intolerance		
middle vs. high	11.5 (-0.6, 23.5)	11.6 (-0.3, 23.6)@
low vs. high	14.4 (0.5, 28.3)	15.8 (2.1, 29.5)@
School absences		
middle vs. high	0.5 (-8.3, 9.2)	-2.7 (-13.0, 7.7)@@
low vs. high	-1.3 (-10.8, 8.2)	-1.5 (-10.9, 7.9)@@
Rescue medication use		
middle vs. high	5.2 (-10.1, 20.5)	5.0 (-11.3, 21.2)@@@
low vs. high	11.5 (-8.0, 31.0)	13.9 (-7.3, 35.2)@@@
Unplanned medical visits		
middle vs. high	1.4 (-9.5, 12.4)	-0.8 (-12.5, 10.8)^
low vs. high	6.3 (-6.6, 19.2)	4.5 (-8.5, 17.5)^
Controllability		
middle vs. high	7.9 (-2.2, 18.0)	12.0 (1.8, 22.3)^^
low vs. high	4.6 (-6.6, 15.8)	8.1 (-3.2, 19.4)^^

*Child respiratory outcomes were number of days in previous 2 weeks recoded as any versus none (referent) except for unplanned medical visits (6 months) and controllability (not at all/somewhat vs. quite or extremely controllable (referent); no reference time period specified); rescue medication use includes 158 diagnosed asthmatics only

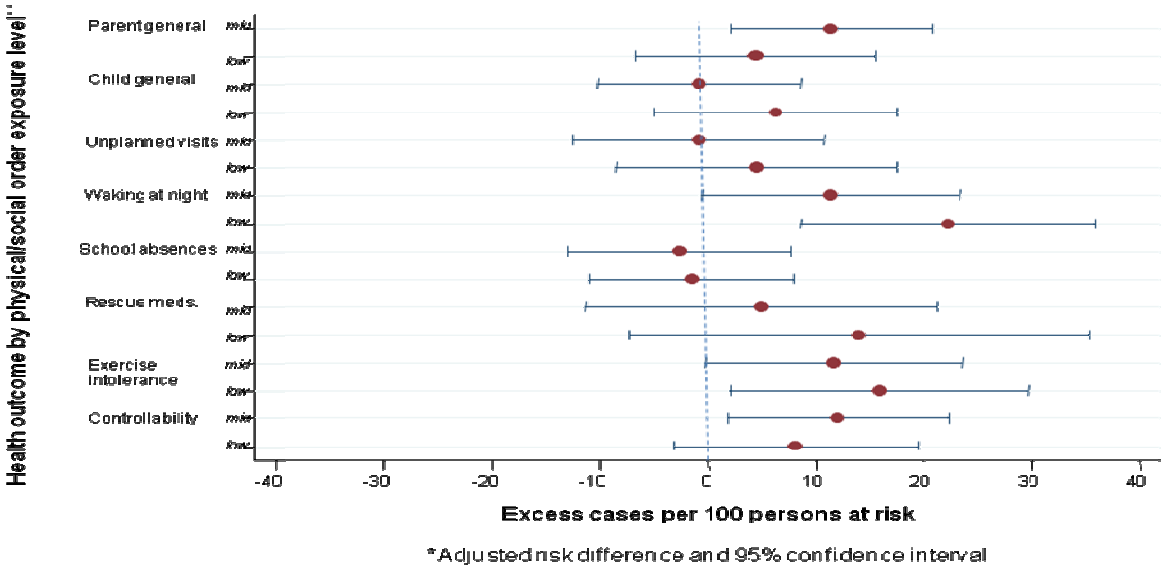
**3-level neighborhood exposures yielded 2 RDs; high (most favorable) level= referent
***Adjusted for child race/ethnicity, parent education, and parent marital status
†Adjusted for child race/ethnicity, parent marital status, parent nativity, and parent age
††Adjusted for child race/ethnicity, parent education, parent marital status, parent nativity, and parent age
†††Adjusted for child race/ethnicity, parent marital status, parent nativity, and parent age
#Adjusted for parent nativity
##Adjusted for parent education, parent marital status, parent nativity, and parent age
###Adjusted for child race/ethnicity, parent education, and parent marital status
@Adjusted for parent marital status
@@Adjusted for child race/ethnicity, parent education, parent marital status, and parent nativity
@@@Adjusted for child race/ethnicity, parent education, parent marital status, parent nativity and parent age
^Adjusted for child race/ethnicity and parent nativity
^^Adjusted for child race/ethnicity, parent marital status, parent nativity and parent age

Figure 5.1. Associations of collective efficacy and binary health outcomes (n=319)



*Adjusted for parent race, age, education, marital status, and/or nativity
**Each RD contrasts the middle (mid) or the low exposure level with the high level

Figure 5.2. Associations of physical/social order and binary health outcomes (n=319)



*Adjusted for parent race, age, education, marital status, and/or nativity
**Each RD contrasts the middle (mid, or the low exposure level with the high level

CHAPTER 6
DISCUSSION

Summary of Findings

Specific Aim 1: To investigate the association of housing stressors and child respiratory health and parent and child general health among low-income families in Chicago.

Aim 1 Findings: Results supported the study's hypothesis; exposure levels reflecting increased number and difficulty with housing stressors were associated (to varying degrees of strength) with higher risks/rates for six of the eight undesirable health outcomes examined. This study adds to growing evidence that families' home environments, including acute and chronic material and social stressors, contribute to asthma burden in urban areas. Further, the burden of stressors likely depends on individual-level attitudes and coping resources. *i.e*., psychological stress resulting from a stressor is different for individuals. Observed associations of housing stressors and general health suggest that stressors may influence a range of mental and physical health outcomes for adults and children. Specific findings include:

► Exercise intolerance was strongly associated with housing stressors. We observed moderately strong associations for waking at night, unplanned medical visits and controllability.

► Only rescue medication and school absences had no observed association with housing stressors.

► The association for child general health and housing stressors was moderately strong while that for parent general health was weak.

Specific Aim 2: To investigate the association of parents' perceptions of neighborhood stressors and child respiratory health and parent and child general health among low-income families in Chicago.

Aim 2 Findings: Overall, results supported the study's hypothesis; exposure levels reflecting more negative perceptions of one's neighborhood tended to be associated with higher risk of undesirable general and respiratory health outcomes. Not all observed associations were strong, and results differed somewhat for the two main exposures. Child respiratory outcomes tended to be more strongly associated with physical/social order while parent and child general health were more strongly associated with collective efficacy. Findings suggest that psychosocial stressors may influence a range of mental and physical health outcomes for adults and children. This study adds to growing evidence that the social environment, in addition to the physical environment, contributes to asthma burden in urban areas. Further, the impact of neighborhood stressors may be influenced by individual-level perceptions and reactions to stress. Specific findings include:

► Waking at night was the only outcome of six total child respiratory outcomes strongly associated with both main exposures.

► Exercise intolerance and controllability were associated with physical/social order but not with collective efficacy.

► Rescue medication use had associations of substantial magnitude with both main exposures, though they were imprecise due to reduced sample size

► School absences and unplanned medical visits had nearly null associations with both exposures.

► Associations of parents' neighborhood perceptions with their own general health were strong while those with their children's general health were weak.

This research advances asthma scholarship by incorporating novel exposure variables into a theoretical framework of how psychosocial stressors become biologically embedded and influence health through psychological stress pathways. Multidimensional measures of stressors reflected material, social and emotional aspects of residence for a low-income, urban, racially/ethnically heterogeneous sample of families. Three main exposures: 1) family housing stressors; 2) neighborhood collective efficacy; and 3) neighborhood order predicted respiratory and general health outcomes to varying degrees, expanding our knowledge of dimensions of residence that impact health.

Only one outcome for parents was examined – self-reported general health. Its association with housing stressors was weakest, while its association with order was moderate, and its association with collective efficacy was strong. In contrast, child general health had null associations with both neighborhood exposures and a moderately strong association with housing stressors. It is possible that children are less exposed than adults to neighborhood events and conditions and therefore less likely to experience psychological stress as a result. In this case, direct biological health effects would have a smaller role in child health than indirect effects through caretaking behaviors of parents. Children may be more susceptible to housing stressors that occur in their everyday, immediate environment and affect other household members. Parents, too, might be expected to be more severely affected by housing stressors than by broader environmental factors, but general health is a vague outcome with no explicit time reference period, and associations may have been obscured.

The tendency of child respiratory health to be associated with order but not with collective efficacy suggests that the more material nature of variables comprising order may be proxies for environmental asthma triggers. Children may be protected from the interpersonal, community-level dynamics reflected in the collective efficacy exposure, and

therefore health impacts would more likely occur via parent behaviors than via stress effects on child biology. Also, the variables comprising the neighborhood exposures had no stated time reference period. Parent perceptions may incorporate their memories and attitudes of neighborhood conditions from the past, and their children may not have recently experienced similar stressors.

Analyses with housing stressors as the main exposure suggest that psychosocial factors play an important role in child respiratory health. Again, housing stressors may exert an influence on children that is more meaningful than neighborhood social stressors, particularly for young children, and that is more likely to result in distress. Four of six child respiratory outcomes were moderately-to-strongly associated with housing stressors. (School absences and rescue medication were not associated with the main exposures in either study; limitations of these outcomes are discussed in chapters 4 and 5). Waking at night, exercise intolerance and controllability had the most consistent and strong associations across exposures.

Methodological Considerations

Asthma epidemiology has grown increasingly complex. Numerous risk factors are well-documented, but disentangling material and social factors is difficult because they are likely to co-occur as a result of distal determinants, such as poverty and segregation. Furthermore, individual-level mechanisms are influenced by community-level conditions and processes.[16, 120, 176, 177] This research, motivated by the need to look beyond race/ethnicity and SES to explain asthma disparities, sought to expand the relevant dimensions of residential stressors that are hypothesized to impact respiratory disease through stress pathways. Deeper and broader exploration of the circumstances of urban families is required before embarking on large-scale data collection efforts and attempting to quantify causality with

enormously sophisticated statistical models. This research incorporates non-traditional exposures and some covariates that are not consistently measured in asthma research. The most appropriate covariates for the psychological stress framework are included and results discussed in light of data limitations and the inherent complexity in studying psychosocial determinants and stress and asthma.

Numerous risk factors are beyond the scope of this study or not included in the dataset, *e.g.*, genetics, and air pollution. Some risk factors, such as household allergens, have a direct effect on asthma onset and exacerbation and do not require the stress pathway to influence health. Biological markers of stress, such as from neurogenic and immune-mediated processes, are included in the conceptual framework as causal pathways and are therefore not included as covariates in models. Similarly, behavioral measures, *e.g.*, allergen reduction in homes and accessing healthcare, are hypothesized to be on causal pathways. Psychological morbidity is hypothesized to be produced by the main exposures; therefore, a depressive symptoms score is not included as a covariate. Likewise, smoking, a health behavior and a coping strategy, is influenced by stress. A bi-directional relationship between smoking and health is also hypothesized since smoking among household members may be influenced by the respiratory health of household members. Parent depression also likely has a bi-directional association with stressors and with health outcomes. Demographic variables typically included in asthma research such as child age and gender as well as health insurance coverage are related to asthma outcomes, but there is no obvious association with residential stressors. Parent and child asthma diagnosis status may be associated with respiratory and general health outcomes, but since asthma onset and eventual diagnosis are not necessarily comtemporaneous with current residential stressors, these variables were not considered potential confounders.

Though a number of risk factors were deemed not to be potential confounders in these analyses, they were examined as possible modifiers of the relationship between residential stressors and health. None was found to have a consistent or strong modifying effect. EMM assessment confirmed our strategy not to stratify or to include an interaction term for child asthma diagnosis status. Parents of undiagnosed children may have been less familiar with symptoms or less likely to utilize healthcare or keep children home from school than parents of asthmatics. Nonetheless, interventions to address stressors and stress would likely be targeted to low-income families generally, especially since underdiagnosis is a well-documented problem.[8, 57] The average effect for all children with respiratory problems was desired since diagnosis is a sociological process based not only on presence of disease but on family and community resources and health attitudes and practices.

Respiratory health measures may be influenced by underlying severity and individual triggers as well as by disease management, which reflects an individual attitudes and practices and societal factors such as access to care and diagnostic biases. A single composite measure may obscure important relationships; therefore, we examined several respiratory outcomes in separate models. Interpretation of results is complicated by differing relationships across exposures and outcomes. Bias might have resulted from the self-report nature of outcomes and exposures, particularly since some outcomes are subjective, and exposures reflected perceptions and self-rated difficulty ratings by design. Personality type and other individual traits might affect the extent to which attitudes and survey responses are optimistic or pessimistic and could cause non-null results.

Using parent-reported difficulty of housing stressors and parent perceptions of neighborhood stressors allowed us to capture the potential for psychological stress in more meaningful ways than if stressors were ranked or weighted by researchers. Sample size

and the number of neighborhoods represented by this dataset are not sufficient to support a random effects model that explicitly examines between-neighborhood variance. However, for this exploration of stress pathways to health, individual-level stressors data are preferred to contextual data since subjectivity and individual reactions influence the downstream biological and behavioral changes. Summarizing multiple stressors allows us to capture the complexity of urban life. Unlike many single risk factors, the three main exposures are robust to problems with single variables, for example, influences of secular, geographic or seasonal trends. A parent's report of residential stressors provides only a proxy for those that affect her child. If they are not highly-correlated, psychological stress levels may be disparate for parent and child. However, because a parent plays a crucial role in disease management for her child, stress-influenced behaviors are likely to influence a child's health whether or not the child is exposed to stressors. Adult asthma was not examined in this study; a comparison of child and parent respiratory health could disentangle the relative contributions of biological and behavioral changes resulting from stress.

The study population is comprised of families from one city. Findings are not necessarily generalizable to all urban areas or to all low-SES neighborhoods within Chicago. However, the careful recruitment of schools and the high participation rate for the respiratory survey yielded a low-income, racially/ethnically diverse sample of families. As SES measurement and adequate adjustment in statistical models are difficult, additional SES measures were included when warranted by confounding assessment. Nonetheless, selection bias may have affected generalizability of findings; schools were a convenience sample, the baseline survey had a 64% response rate, and approximately half of the study sample was lost-to-follow-up at survey 2. Surveys were conducted in English and Spanish, and though validated instruments were used for most questions, linguistic and cultural biases may have influenced data quality.

Despite the limitations to causal inference for the stressors-asthma relationship, the specific aims and analytic strategies employed for these studies are appropriate given the state of asthma scholarship. A large-scale, randomized, controlled trial of interventions to mitigate the effects of stressors among high-stress populations at this time would waste resources and unduly burden participants. Asthma trends and inequities are not fully-explained by known risk factors. Expanding the dimensions of risk, particularly risk in urban areas, taking into account subpopulation differences, is a necessary next step. Similar to one of the aims of qualitative research -- to construct more sophisticated measures of social phenomena -- this study was designed to stimulate our "epidemiological imagination" and contribute to policies and interventions geared toward improving population health and reducing health inequities.[187] In a discussion of the complementarity of qualitative and quantative research, Muntaner and Gomez note the limitations of survey research to uncover social mechanisms and the inability of epidemiologists to generate sound hypotheses for specific populations. They highlight the ability of qualitative research to point to previously ignored or marginalized mechanisms that even rigorous and expensive quantitative research has failed to do, and the fact that sampling and data analysis cannot always overcome basic issues of response validity.[161] Though this study did not examine qualitative data, the unique dataset offered the opportunity to explore important social and psychological factors that surveillance data and the vast majority of asthma investigations do not offer.

Public Health Implications

These findings alone do not justify the allocation of funding and other resources dedicated to mitigating housing and neighborhood stressors. Additional cities must be studied as stressors undoubtedly vary by geography. Also, population

heterogeneity must be understood before designing interventions to address both stressors and stress. These findings support a psychological stress mechanism that results in poor health outcomes for children with respiratory problems. Results also show that general health is associated with the exposures of interest, lending support for the idea that other chronic diseases, injuries, disabilities and perhaps acute health events are likely to have similar influences. Increasing awareness of the role of the social environment in the health of disadvantaged populations can be seen as contributing to a research effort to demonstrate that stress matters. Indeed, health inequities may be driven by stress disparities. This research and additional evidence that stressors and stress are related to a range of health outcomes may eventually strengthen the charge for structural and individual-level solutions to reduce stressors and mitigate the impacts of stress. Ultimately, reductions in social and health inequities are the goals.

These findings emphasize the importance of addressing not only material residential stressors but social and emotional stressors that may be mediators or moderators of the effects of low-SES on respiratory health. The housing and neighborhood stressors illuminated in this study provide opportunities for interventions that may be more feasible and have more immediate impact than the more distal determinants also driving inequities. It would be inappropriate to make inferences based on one exposure without considering the interrelated factors that contribute to stress. Good interventions have multifaceted effects and address a breadth of interrelated factors. The distinction between “traditional” environmental and psychosocial determinants of disease may be crucial 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.[16, 176] 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. While community groups, clinicians, researchers and politicians seek structural solutions to inequities, interventions can address individuals' reactions to stressors.

Future Research Directions

As already noted, disentangling material and social risk factors for asthma is difficult. Research to uncover factors that co-vary with known risk factors is an important next step toward understanding asthma's social patterning. This research attempted to identify residential stressors among a population known to be disproportionately affected by asthma prevalence and morbidity -- low-income, Chicago families. We did not test whether stressors and resulting stress are mediators or moderators of health. Mediators would not be expected to explain additional variance in health outcomes. Moderators, however, would help explain differential outcomes among places and populations that share low-SES and environmental exposures. Moving beyond cross-sectional studies that demonstrate associations toward studies that allow causal inference will be required to justify interventions designed to improve population health. Longitudinal data would allow examination of psychological factors over the lifecourse and the importance of chronic and acute stressors[99] as well as their frequency and duration. The possibility that the asthma

phenotype is programmed before birth[21] makes understanding stressors for women especially important since stress mechanisms may impact asthma *in utero*. Reverse causality poses a challenge for asthma research since stress and its sequalae may be caused or aggravated by having asthma or caring for someone with asthma[99, 179-181] and highlights the need for longitudinal data. Other life transitions, such as moving residence or immigrating, are important influences on health and should be noted in asthma research since they have the potential to impact stress and are also correlated with social and material asthma risk factors. Not just quantity but quality of data – including information about difficult-to-measure but nonetheless important social determinants – is missing from most databases available to researchers. Future data collection endeavors should be thoughtfully planned by interdisciplinary teams with an emphasis on factors affecting populations with inequitable material and social exposures and inadequate resources for preventing their harmful health consequences.

Recent advances have illuminated mechanisms for the influence of psychological stress on asthma expression and development through "dysregulated immunity."[15, 16] Research is needed to understand a possible mediating role of psychological stress, for example, the ability of chronic stressors to mediate the effect of allergens or irritants on atopy and asthma through neuro-immune mechanisms.[15, 16] A multi-level approach may reveal structural forces that influence the distribution of stressors and psychological stress. Individual-level mechanisms are influenced by community-level conditions and processes.[16, 120, 176, 177] 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.[178] A study of the social environment and adolescent asthmatics suggested that family factors may affect asthma through physiologic changes, such as inflammation, while neighborhood factors influence health behaviors, such as smoking, which impact asthma.[24]

More empirical evidence of the role of social context in asthma development and expression at different stages of the lifecourse is required to design effective interventions as well as structural solutions to inequities.[99]

Conclusions

Childhood asthma highlights the alarming burden of ill health among disadvantaged populations in the U.S., in this case, low-income city dwellers. The role of psychological stress in producing biological and behavioral reactions that impact respiratory health transiently and long-term is increasingly being clarified. Researchers posit stress as a potential determinant of health disparities. Asthma's social patterning may very well be explained by stress, which may be increased in certain populations which have inadequate resources to mitigate its effects. Stressors stemming from residence may be particularly important given that homes and communities may protect from or expose residents to physical and social toxins.

This research may broaden our view of individual, family and societal influences on asthma and generate hypotheses about other non-traditional determinants, including those that are currently challenging to study but crucial to impacting population 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 lends support to the idea that stressors are moderators of the SES-health relationship.

If stressors were only mediators, they wouldn't 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.

Asthma interventions must address not only "bricks and mortar" but causes and consequences of stress. 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 health and social policies and the reduction of health disparities.

APPENDIX A
THIRTEEN NEIGHBORHOOD STRESSORS EXCLUDED FROM PRINCIPAL COMPONENTS ANALYSIS

Potential neighborhood stressors evaluated by correlation matrix of 27 stressors and excluded from principal components analysis due to low correlation (R<0.510) with other variables

SURVEY ITEM	R*
How noisy is your neighborhood due to traffic, trains and airplanes? VERY, SOMEWHAT, NOT VERY, OR NOT AT ALL?	0.247
How often does your child play indoors instead of outdoors because of bullying or teasing from other kids? ALWAYS, OFTEN, SOMETIMES, RARELY OR NEVER?	0.435
All things considered what do you think this neighborhood will be like a few years from now? A BETTER PLACE TO LIVE, STAY THE SAME, OR BE WORSE?	0.470
Do you STRONGLY DISAGREE, DISAGREE, AGREE OR STRONGLY AGREE with the following statements?	
If people in this neighborhood worked together to prevent such a closing, positive changes would occur	0.384
People in this neighborhood would work together to prevent the closing of an important community institution (ex. a school, fire department, train station, etc.)	0.456
People in this neighborhood are likely to call and complain if the street lights are out	0.465
People in this neighborhood do not share the same values	0.505
People in this neighborhood generally don't get along with each other	0.509
How would you rate your neighborhood in the following areas? EXCELLENT, GOOD, FAIR, OR POOR?	
Mail Delivery	0.370
Ease of Transportation	0.405
Snow Removal	0.414
Garbage Collection**	0.518
Police**	0.518

*R: Correlation coefficient; reported is the highest R of a stressor with any of 26 others
**2 items with R>=0.510 (cutoff criterion for inclusion in principal components analysis) were excluded because they were correlated only with each other at an R value just above the cutoff

APPENDIX B
VARIANCE EXPLAINED BY 3-COMPONENT SOLUTION FROM PRINCIPAL COMPONENTS ANALYSIS OF FOURTEEN NEIGHBORHOOD STRESSORS

Total variance explained by a 3-component solution resulting from principal components analysis of 14 potential housing stressors variables

COMPONENT	EIGENVALUES		
	TOTAL VARIANCE	PERCENT OF VARIANCE	CUMULATIVE VARIANCE
1*	**3.928**	**28.058**	**28.058**
2*	**2.257**	**16.121**	**44.179**
3*	**1.983**	**14.161**	**58.340**
4	1.202	8.586	66.926
5	0.964	6.885	73.811
6	0.556	3.975	77.786
7	0.511	3.646	81.432
8	0.453	3.234	84.666
9	0.432	3.084	87.750
10	0.412	2.946	90.696
11	0.374	2.674	93.370
12	0.358	2.557	95.927
13	0.315	2.253	98.180
14	0.255	1.820	100.000

*Components 1, 2 and 3 were further evaluated as main exposures and labeled, respectively, physical/social disorder, collective efficacy, and change in neighborhood

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