THE SOCIAL DETERMINANTS OF HEALTH DISPARITIES: THE ROLE OF SOCIAL AND TEMPORAL CONTEXTS

by

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A dissertation submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
(Public Policy and Sociology)
in the University of Michigan
2008

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ACKNOWLEDGEMENTS

I would not have been able to finish this dissertation without the guidance, feedback, and support of my dissertation committee. I am deeply indebted to Jeffrey Morenoff and Sheldon Danziger, my Co-Chairs. Despite his incredibly busy schedule, Jeffrey Morenoff dedicated countless hours toward advising me in nearly all aspects of the dissertation process. He helped me think through the theoretical framework of my research; fielded myriad methodological, analytical, and logistical questions pertaining to my dissertation; and responded with exhaustive comments on my drafts. I am also extremely appreciative of Sheldon Danziger, whose accessibility, "turn-around time" in reading papers, and commitment to mentorship were unparalleled. Professor Danziger constantly challenged and encouraged me to produce the best quality research possible.

As my advisor for the past five years, David Williams has graciously taken me under his wing and has been a constant source of wisdom and guidance. I am especially grateful for the material and intellectual resources he provided in the past two years, which were instrumental to finishing the dissertation in Boston. Despite his own professional transitions, Professor Williams was always able to find time to brainstorm dissertation ideas, offer professional advice, or recount his own experiences in graduate school. I continue to be inspired by his humility as a scholar and his capacity to balance the critical eye with the generous spirit.

Rosalind Wright generously shared with me her data, her time, and her research staff. As a co-author for Chapter 3, she contributed to the conceptual framing of the paper, to the data analysis, and to the writing. Her medical expertise has proven incredibly useful in furthering my understanding of the biosocial processes underlying the stress-asthma relationship. She is a model for an interdisciplinary researcher.

Finally, Jim House provided insightful feedback on my work, especially in Chapter 4. He and Professor Morenoff waded through countless tables and data outputs, week after week, in an effort to help me make sense of my results. I am especially thankful for Professor House for providing me with a venue at the CCAHS weekly meetings to discuss my dissertation.

I would also like to thank my colleagues and friends: Mike Bader, who helped me acquire the SSO data and willingly trouble-shooted STATA difficulties; Jennifer Ailshire, who offered valuable methodological advice and read through paper drafts; and James Mickens, who, despite his non-sociology status, provided brilliant insights and a healthy dose of perspective (and humor) throughout the process. I am especially grateful to Jessica Wyse for her friendship, support, and daily check-ins. Her organizational help brought me immeasurable peace of mind and was a key reason I was able to complete the dissertation.

My parents, grandparents, and siblings offered precious emotional and physical sustenance throughout the past year. Their encouragement and pride, their endless patience, phone calls, and outpouring of chicken soup and homemade dinners gave me the stamina to finish. My parents, Elliot and Adrianne Sternthal, in particular, have been my most ardent supporters. My father's medical expertise has helped clarify the more

technical scientific aspects of my work; his painstaking editing and thoughtful comments sharpened my ideas and improved my writing. My mother's unyielding enthusiasm for my research has sustained me when my own enthusiasm has waned. She has operated as an intellectual sounding board and a thought-provoking critic, willing at any moment to discuss a nascent idea.

Lastly, Zeke Reich has devoted countless hours, sacrificed sleep, and traversed continents and time zones "virtually" to be an unconditional support for me over the last few months. I am appreciative of his clarity in thinking, his capacity to integrate new fields of knowledge with ease, and his invaluable writing advice. It is rare to find someone whose intellectual acumen rivals an equally extraordinary capacity to listen. Zeke, in embodying both traits, has been a true source of calm, and for that, I am most grateful.

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ABSTRACT

The goal of this dissertation is to examine contextual determinants of racial disparities in health across the life course. I progress from "downstream" to "upstream" processes by focusing in one chapter on the prenatal context, in another on health behaviors and family context, and in the third, on the neighborhood context.

Chapter 2 examines the relationship between lifetime exposure to abuse among pregnant women in the Boston area and elevated cord blood IgE. Results demonstrate that greater exposure to violence throughout the mother's life course is associated with increased risk of offspring elevated IgE at birth, after adjusting for maternal and family-level confounders. Abuse occurring more proximate to pregnancy is not correlated with elevated cord blood IgE, suggesting that the cumulative exposure to violence (i.e., chronic abuse) may have the most salient fetal effects. The results indicate that the detrimental effects of violence may a) accumulate over the life course and b) transmit across generations through the fetal environment.

Chapter 3 explores the intergenerational transmission of disadvantage by examining the relationship between teen childbearing and offspring health among a nationally representative sample of children ages 5-19. Logistic regressions reveal no increased risk of low birthweight, chronic illness, obesity or asthma among offspring of teens versus non-teens and a slight decrease in obesity among offspring of teens,

suggesting that the timing of one's pregnancy may matter less than other contextual factors in influence offspring health.

Chapter 4 uses multilevel methods to investigate the extent to which one's residential environment is linked to currently active asthma. No association is found between neighborhood sociodemographic factors and asthma. Random-slope models demonstrate significant effects of affluence and immigrant concentration for non-blacks; however, the unexpected direction of the coefficients and the small sample size call into question the reliability and validity of these findings.

Emerging from these three studies is a complex picture of how contextual factors may affect health disparities. The findings confirm the value of incorporating social contexts in studying health disparities, while underscoring the pitfalls in overlooking the diversity in age, ethnicity, life stage, and health outcomes within such research.

CHAPTER 1

INTRODUCTION

Racial disparities persist across multiple measures of health status (Fiscella and Williams 2004). According to national mortality statistics, African Americans have an overall age-adjusted mortality rate that is 1.5 higher than whites (Akinbami 2006). Moreover, they have significantly higher mortality rates from cardiovascular and cerebrovascular disease, most cancers, diabetes, HIV, sudden infant death syndrome, and homicide than do whites (Eberhardt, Ingram, and Makuc 2001). The sources of these differences are not well understood. Though scientific research has historically attributed black-white differences to biological causes, the overwhelming evidence indicates that the major determinants of poor health among blacks are social and behavioral (Williams, Lavizzo-Mourey, and Warren 1994).

The goal of this dissertation is to examine racial disparities in health across the life course. Through the course of three papers, I progress from "downstream" to "upstream" processes linking race to health disparities. Unlike conventional dissertations, the three papers are not intricately related, each examining a different dataset, sample population, and health outcome. Nevertheless, all three studies address the central issue of health disparities at specific life course stages.

In Chapter 2, "Associations among maternal exposure to prenatal and lifetime interpersonal violence and cord blood IgE," I use a Boston-based prospective data set

(the ACCESS study) to examine one physiological pathway through which violence—a proposed mediator of the race-health relationship—may "get under the skin" to heighten the risk of asthma. Specifically, I analyze the association between fetal IgE production, an immunological biomarker predictive of subsequent childhood asthma onset, and a mother's experience of family violence, both throughout her life course and during pregnancy. The study's theoretical framework draws heavily from the concept of "perinatal programming," which posits that adverse exposures *in utero* may program the function of organs, tissues, or body systems, setting the stage for the development of chronic illness late in life (Dole, Savitz, Hertz-Picciotto, Siega-Riz, McMahon, and Buekens 2003; Welberg and Seckl 2001). It also utilizes a cumulative effects perspective (Ben-Shlomo and Kuh 2002) to assess whether the *accumulation* of violence exposure over the mother's life course, rather than abuse during any *specific* time period, is relevant for IgE.

Chapter 3 presents the second paper, "Is teen childbearing good for offspring health? A test of two hypotheses." Similar to the prior study, this paper draws on a cumulative effects perspective (Ben-Shlomo and Kuh 2002) to examine teenage childbearing, a health behavior disproportionately clustered in segregated neighborhoods and an often hypothesized cause of health disparities. Central to this paper is Geronimus' weathering framework (Geronimus 1992), which sees early fertility timing as a culturally adaptive response to maternal "weathering" (accelerated aging). More broadly, Chapter 3 underscores the value of incorporating the broader social, cultural, and physical contexts, as well as the mother's health trajectory, in properly understanding health behaviors.

Chapter 4, "Does place matter? Neighborhood context and asthma," uses the Chicago Community Adult Health Survey (CCAHS) to assess the extent to which neighborhood factors, as opposed to individual factors, are associated with currently active asthma in an adult population. The multilevel study examines the link between neighborhood sociodemographic conditions (concentrated disadvantage, affluence, age composition, immigrant composition) and adult asthma in both a full sample and a sub-sample of previously diagnosed asthmatics and includes cross-level interactions to test for differential neighborhood effects by race.

Taken together, these papers underscore the role of spatial and temporal contexts in initiating, perpetuating, and perhaps exacerbating health disparities. Each paper on its own addresses a specific life course stage (in utero, childhood, adulthood). Combined, they aim to present a) a clearer understanding of the relationships between and among fundamental, intermediate, and proximate factors, and the various mechanisms through which these factors may lead to racial disparities in health, and b) the relative importance of sensitive/critical periods and cumulative exposures at different stages of the life course.

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CHAPTER 2

ASSOCIATIONS AMONG MATERNAL EXPOSURE TO PRENATAL AND LIFETIME INTERPERSONAL VIOLENCE AND CORD BLOOD IGE

Background

Evidence linking psychosocial stress to the expression of asthma continues to grow (Chida, Hamer, and Steptoe 2008; Wright, Cohen, and Cohen 2005). Interest in this subject is motivated by an enhanced understanding of the natural history of allergic asthma, the neurobiology of stress and asthma pathophysiology (Wright 2005), as well as an increasing effort to determine why asthma remains a leading cause of health disparities unexplained by physical environmental risk factors (Gold and Wright 2005).

Because asthma is a disease of dysregulated immunity and the most important periods of immune development with regard to expression of asthma are likely during gestation and early childhood, a logical means to examine the role of stress on asthma development is to investigate the effect of prenatal environmental exposure on *in utero* immune response. Notably, studies on prenatal exposure to stress and its relationship to the immune response are only now emerging (Pincus-Knackstedt, Joachim, Blois, Douglas, Orsal, Klapp, Wahn, Hamelmann, and Arck 2006; Wright, Cohen, Carey, Weiss, and Gold 2002; Wright, Finn, Contreras, Cohen, Wright, Staudenmayer, Wand, Perkins, Weiss, and Gold 2004b). Animal research examining the effects of early life adversity on stress neurobiology provide initial evidence that *in utero* psychological

stress[1] may prove especially critical for asthma (Wright 2007). However, no human studies to date have directly examined the influence of prenatal stress on asthma risk in children or the pathways linking the two. Also unknown is the nature and scope of the stressor(s) that would likely have measurable impact.

This study examines interpersonal violence experienced across mothers' life course as a key stressor that may play a role in asthma etiology in their children. We focus on interpersonal violence because, as a high magnitude stressor that disproportionately affects members of racial/ethnic minorities and those of lower SES (Cunradi, Ames, and Moore 2008; Cunradi, Caetano, and Schafer 2002; Field and Caetano 2005), such exposures may in part explain the excess burden of asthma in these populations. Furthermore, exposure to violence (and other traumas) has been implicated in key physiological disruptions likely underlying asthma etiology (Altemus, Cloitre, and Dhabhar 2003; Altemus, Dhabhar, and Yang 2006; Heim and Nemeroff 2002). The biobehavioral disruption linked to violence exposure can also be transmitted across generations (Yehuda, Engel, Brand, Seckl, Marcus, and Berkowitz 2005). As such, it is biologically plausible that traumatic stress during early development may be instrumental in subsequent asthma susceptibility (Wright and Enlow 2008).

While the existing literature on the risk factors contributing to immune dysregulation at birth focuses on exposure during pregnancy, few studies have explored the effects of cumulative social risk—that is, maternal exposure prior to pregnancy. Growing epidemiological evidence demonstrates that exposure to trauma can generate disrupted physiological stress responses even several years following the experienced events (Anda, Felitti, Bremner, Walker, Whitfield, Perry, Dube, and Giles 2006; Bremner

[1] As opposed to physical stressors (e.g., HTN in mothers).

and Vermetten 2001; Bryant, Harvey, Guthrie, and Moulds 2000; Rick and Douglas 2007). Remote and cumulative exposures to trauma (violence) in a mother may be relevant to her child's neuroimmune development in this paradigm.

Studies that incorporate these strands of overlapping research and strategies for studying maternal stress during pregnancy are needed to continue to elucidate the mechanisms underlying the links between stress and atopic asthma development. In this study, we utilize a life course perspective to investigate the relation of maternal current and lifetime trauma history with cord blood total immunoglobulin E expression (IgE), controlling for a number of important confounders.

Cord blood IgE was selected because, as a marker of the prenatal immune response, it has been associated with increased risk of aeroallergen sensitization and the later development of allergic asthma in children, particularly among those with a family history of atopy (Halken 2003; Odelram, Bjorksten, Leander, and Kjellman 1995; Tariq, Arshad, Matthews, and Hakim 1999). Research examining a possible association between maternal trauma history and early indicators of childhood asthma risk may begin to illuminate key biological mechanisms through which violence may "get under the skin" as well as provide further evidence for the intergenerational transmission of trauma effects.

Specifically, the study addresses two research questions: first, are cord blood IgE levels significantly higher among children born to women experiencing abuse during pregnancy, compared to children of their non-abused counterparts? Second, does the cumulative exposure to violence throughout one's life course (i.e. total exposure during

early childhood, teenage years, adulthood, during pregnancy) influence cord blood IgE levels?

Literature Review

The theoretical and empirical basis for our paper relies heavily on biomedical research on asthma pathogenesis, the neurobiology of the stress response, perinatal programming, life course epidemiology, and the transgenerational transmission of stress effects. We briefly review the overlapping evidence from these historically disparate areas of scholarship in order to ultimately frame our hypothesized associations between violence and biomarkers of early asthma risk.

Asthma Biology and its Relationship to Atopy

Asthma is a disease typified by airway obstruction, airway inflammation, and heightened airway sensitivity to a range of stimuli (Burrows, Martinez, Cline, and Lebowitz 1995; Robinson, Hamid, Bentley, Ying, Kay, and Durham 1993; Wright 2005). Conceptualized as a disease of dysregulated immunity, most asthma involves allergy-mediated airway inflammation and biological hypersensitivity to environmental stimuli. Immune responses that lead to the development of antigen-specific IgE are essential to the development of atopic asthma (Peden 2000). The most important periods of immune development with regard to expression of atopic asthma are likely during gestation and early childhood (Peden 2000; Reed 2006). Consequently, there is increasing interest in identifying aspects of the fetal environment that may alter neuro-immune expression and trigger atopic disorders (Barker 1990; Wright 2007; Wright and Enlow 2008).

Stress and Asthma

Beyond allergen exposure (Hoffjan, Nicolae, Ostrovnaya, Roberg, Evans, Mirel, Steiner, Walker, Shult, and Gangnon 2005) and gene-environment interactions (Cohen, Kessler, and Gordon 1995), psychosocial stress may play a role in asthma development and morbidity (Chida, Hamer, and Steptoe 2008; Wright 2005). Chronic stress has been linked to asthma exacerbations in cross-sectional (Oh, Kim, Yoo, Kim, and Kim 2004) and prospective studies (Sandberg, Jarvenpaa, Penttinen, Paton, and McCann 2004). Other evidence suggests a role for stress in the onset of asthma (Wright, Cohen, and Cohen 2005; Wright et al. 2002). Perceived stress has been linked to asthma symptoms, bronchoconstriction (Lehrer, Isenberg, and Hochron 1993) and reduced pulmonary flow rates (Isenberg, Lehrer, and Hochron 1992).

The link between stress and atopic asthma is thought to be mediated through neuroimmunregulation. Stress dysregulates the body's neuroimmune systems. The neuroimmune systems may, in turn, modulate the body's hypersensitivity responses (e.g., airway inflammation, airway sensitivity). According to the life stress model (Wright and Fisher 2003; Wright, Rodriguez, and Cohen 1998), when individuals perceive themselves as being under stress, they experience a range of negative affective states (e.g., depression and anxiety), which in turn exert effects on biological processes or behavioral patterns that influence disease risk (Cohen, Janicki-Deverts, and Miller 2007). Accompanying these emotional changes are a cascade of neuroendocrine and immunological reactions, including the dysregulation of the hypothalamic-pituitary-adrenocortical (HPA) axis and the sympathetic and adrenomedullary (SAM) system

(McEwen 2002; Wright 2005). Though system dysregulation is useful for short-term immune, metabolic, and neural defense responses to stress, continuous or repeated activation of the stress response can result in long-term bodily damage (McEwen 2002). For example, chronic stress may precipitate a state of hyporesponsiveness of the HPA axis, depressing the secretion of anti-inflammatory hormones (Buske-Kirschbaum, Fischbach, Rauh, Hanker, and Hellhammer 2004). Such neuroendocrine and immune modulations may ultimately heighten the risk of asthma.

Perinatal Programming and Stress

An extensive body of research suggests that the *in utero* environment may influence fetal development, a concept known as "perinatal programming" (Welberg and Seckl 2001). Numerous studies have found associations between prenatal factors and lifelong risk of developing coronary heart disease (Hales, Barker, Clark, Cox, Fall, Osmond, and Winter 1991; Rich-Edwards, Colditz, Stampfer, Willett, Gillman, Hennekens, Speizer, and Manson 1999; Roseboom, van der Meulen, Osmond, Barker, Ravelli, Schroeder-Tanka, van Montfrans, Michels, and Bleker 2000), diabetes mellitus (Barker 1992; Levitt, Lambert, Woods, Hales, Andrew, and Seckl 2000), and hypertension (Barker 1990). Barker and colleagues have argued that adverse fetal conditions during late gestation may disturb the programming of blood pressure regulation, cholesterol metabolism, and glycemic control. Because one of the most important periods of immune development with regard to expression of atopy and asthma are likely during gestation (Reed 2006; Wright 2007), it is also possible that the asthma phenotype could start to be programmed before birth. A number of lines of evidence

suggest other environmental factors influence prenatal programming of the asthma phenotype. Studies report associations between asthma risk and maternal antibiotic use, probiotic use, and maternal infections during gestation (Calvani, Alessandri, Sopo, Panetta, Tripodi, Torre, Pingitore, Frediani, and Volterrani 2004; Coe and Lubach 2003; Hughes, Jones, Wright, and Dobbs 1999; McKeever, Lewis, Smith, and Hubbard 2002; Xu, Pekkanen, Jarvelin, Olsen, and Hartikainen 1999). Other studies have found that maternal exposure to inhaled allergens prime fetal T cells toward an atopic phenotype (Wright, Rodriguez, and Cohen 1998).

The growing list of potential programming agents includes psychological stress. Indeed, experiencing high levels of *in utero* stress may prove especially critical for asthma development (Wright 2007). As studies of mechanisms underlying perinatal stress and asthma risk begin to emerge, proof of concept is provided by animal studies examining the effects of early life adversity on stress neurobiology and development and more recent human data that parallel the animal research (Wright 2007). Animal studies have found associations between perinatal stress and preterm and low birth weight (LBW) (Egliston, McMahon, and Austin 2007; Van den Bergh, Van Calster, Smits, Van Huffel, and Lagae 2007), altered immune function (Hessl, Dawson, Frey, Panagiotides, Self, Yamada, and Osterling 1998), and heightened stress reactivity (Calvani et al. 2004; Kurukulaaratchy, Waterhouse, Matthews, and Arshad 2005; Kurzius-Spencer, Halonen, Lohman, Martinez, and Wright 2005; Xu et al. 1999).

It is hypothesized that the physiological disruptions generated by stress (i.e. dysregulation of the HPA axis and the SAM system) may influence programming of key physiological systems in offspring during pregnancy and early childhood, setting the

stage for the inflammatory processes and altered reactivity to stimuli that are characteristic of the atopic phenotype (Wright 2007; Wright and Enlow 2008). Maternal HPA activation may stimulate specific placental hormones (Frim, Emanuel, Robinson, Smas, Adler, and Majzoub 1988). Stress-induced hormones such as cortisol may influence T helper cell phenotype differentiation in the fetal immune system, elevating IgE production and leading to an increased risk of atopic asthma (von Hertzen 2002). Emerging empirical evidence supports these claims. A 2004 study of caregiver stress and early childhood immune response found that higher stress in the 6 to 18 months after birth was associated with a higher IgE level and an atopic immune profile (Wright et al. 2004b) in toddlers (Wright et al. 2004b).

Research in monkeys has shown that prenatal maternal stress influences the infant's response to aeroallergens (Coe and Lubach 2003). Additionally, maternal nervousness during pregnancy has been shown to be predictive of elevated cord blood IgE (Lin, Wen, Lee, and Guo 2004). Animal studies have shown that stress increases intestinal permeability (Santos, Benjamin, Yang, Prior, and Perdue 2000; Söderholm, Yang, Ceponis, Vohra, Riddell, Sherman, and Perdue 2002) and induces intestinal sensitization to luminal antigens (Yang, Jury, Soderholm, Sherman, McKay, and Perdue 2006). This is particularly relevant given compelling evidence that the gut is involved in immune maturation (von Hertzen 2002) and that fetal exposure to antigens is, at least in part, through access to gut-associated lymphoid tissue (Holloway, Warner, Vance, Diaper, Warner, and Jones 2000). In our cohort, we have previously documented a significant association between prenatal dust mite exposure and cord blood IgE.

Conceptual Framework Linking Violence to Asthma

If, as the above overview suggests, stress is a risk factor for asthma development, it follows that violence, a particularly high magnitude stressor taxing vulnerable individuals, may be a particularly robust potentiator of the cascade of physiological responses that may increase vulnerability to atopy (Altemus, Cloitre, and Dhabhar 2003; Altemus, Dhabhar, and Yang 2006; Heim and Nemeroff 2002; Wright and Enlow 2008). The conceptualization of interpersonal violence in particular as a stressor is based in trauma theory and an extensive literature on family violence (Baum 1990; Baum, Cohen, and Hall 1993; Claussen and Crittenden 1991; De Bellis, Baum, Birmaher, Keshavan, Eccard, Boring, Jenkins, and Ryan 1999; Egeland, Sroufe, and Erickson 1983; Putnam and Trickett 1997; Wright 2006). All forms of interpersonal abuse may have the distressing characteristics of being unpredictable, uncontrollable, and potentially threatening (Cicchetti, Rogosch, Lynch, and Holt 1993; Lynch and Cicchetti 1998). Moreover, even sporadic or acute episodes of abuse can generate long-lasting stress responses for the victims (e.g., in the form of intrusive thoughts and ruminations) (Baum 1990; Baum, Cohen, and Hall 1993). Thus, victims of family violence may be subject to chronic trauma and stress.

Because violence is a major life stressor, it may trigger physiological reactions characteristic of a "chronic stress response" (e.g., dysregulation of the HPA axis and the SAM system) (Baum 1990; Massey 2004; Murali and Chen 2005; Wilson, Kliewer, Teasley, Plybon, and Sica 2002). Specifically, stress-induced disruption of the maternal HPA axis may prime fetal sensitization to allergies and increase atopic risk through

transplacental passage (Wright 2007). Consequently, maternal experiences of violence may influence the infant's immunologic and neuroendocrine developmental processes, leading to the inflammatory processes and altered reactivity to stimuli characteristic of chronic asthma (Wright 2007).

Violence and the Life Course Framework

The life course approach to chronic disease proposes that the combination, accumulation, and/or interaction of biological and social exposures experienced during different stages of life may impact current and future events, environments and health conditions, and thus influence adult health (Ben-Shlomo and Kuh 2002). A cumulative life course model hypothesizes that psychosocial experiences and environments during early and later life accumulate to influence adult health status. This model suggests that if factors operating at different life stages are combined, larger differences in disease risk will be observed (e.g., using a summary variable indicating number of adverse events/environments over the life course to predict adult disease status) (Ben-Shlomo and Kuh 2002; Kuh and Ben-Shlomo 1997; Power and Hertzman 1997).

A cumulative life course model may further elucidate the processes linking maternal violence exposure history to asthma risk in the next generation. Animal and human studies (Anda et al. 2006; Bremner and Vermetten 2001; Bryant, Harvey, Guthrie, and Moulds 2000; Gunnar and Donzella 2002; Heim and Nemeroff 2002; Moorman, Rudd, Johnson, King, Minor, Bailey, Scalia, and Akinbami 2007) demonstrate that exposure to traumatic stress during early development may permanently alter physiological responses for the victims, with adverse health consequences extending into

adulthood (McEwen 2002). Furthermore, exposure to violence not only increases the likelihood of subsequent victimization (Bowen, Heron, Waylen, and Wolke 2005; Saltzman, Johnson, Gilbert, and Goodwin 2003), but may also prime one's body, enhancing vulnerability to proximal stressful experiences (Murburg 1997). Individuals with a history of assault, for instance, are more vulnerable to HPA dysregulation and exhibit blunted cortisol levels in the face of current trauma, compared to those with no preexisting trauma history (Bremner and Vermetten 2001; Bryant, Harvey, Guthrie, and Moulds 2000; Resnick, Yehuda, Foy, and Pitman 1995; Saltzman, Holden, and Holahan 2005). When these disruptions occur or persist during pregnancy, the infant's developing neuroendocrine axis and immune system may also be impacted. As such, a cumulative life course framework—which accounts for both past and current maternal exposure to trauma—may be necessary to understand how maternal experiences of violence influence an infant's immunologic and neuroendocrine developmental processes relevant for asthma.

Hypotheses

Based on these overlapping strands of research, we propose two hypotheses. The first draws on perinatal programming and fetal origins concepts to understand how violence may influence fetal outcomes.

H1: Maternal exposure to intimate partner violence (IPV) during pregnancy (*proximal violence)* will predict higher cord blood IgE levels, even when controlling for other sources of stress and relevant confounders.

The second hypothesis incorporates a cumulative life course framework, which presumes that *accumulation* of violence exposure over the mother's life course, rather than abuse during any *specific* time period, will be most relevant for predicting offspring IgE. More specifically:

H2: Maternal cumulative lifetime exposure to IPV will predict elevated offspring IgE, such that the more time periods in which a mother has experienced abuse in her life (childhood, teenage years, adulthood, pregnancy), the greater the risk of elevated cord blood IgE.

Data

Data for this study come from the Asthma Coalition on Community, Environment, and Social Stress (ACCESS), a prospective pregnancy cohort of women recruited from prenatal clinics throughout Boston in order to assess the role of early life exposure to both physical environmental determinants and psychosocial factors on the rising childhood asthma burden in Boston urban communities. Mother-infant pairs were recruited during prenatal visits from August 2003 to January 2007. Pregnant women receiving prenatal care at Brigham & Women's Hospital, Boston Medical Center, three urban community health centers, and women attending Women, Infants and Children (WIC) programs associated with the health centers in the Boston metropolitan area and one WIC program serving a large suburban population 20 miles south of Boston were eligible for enrollment. The project was designed to take advantage of the structure of the WIC programs, given particular difficulties with recruitment and retention of study participants in this largely lower-income, ethnically diverse inner-city population who moved frequently and/or did not have telephones. Women enrolled in the WIC programs

made monthly visits to pick up vouchers provided for food and childcare and were followed from 3 months to 5 years postnatally. Women who did not speak either English or Spanish and who were less than 18 years of age, or were not in the 2nd or 3rd trimester of pregnancy were excluded. Trained research assistants approached all women receiving prenatal care on selected clinic days that changed weekly depending on patient flow. At the time of this analysis, Project ACCESS had approached 2261 mothers, of whom 1,437 (64%) met eligibility criteria and completed a screening questionnaire. After this screening, 1,156 (80.4% of those eligible) agreed to participate in the longitudinal Project ACCESS cohort. Written informed consent was obtained in the subject's primary language (English or Spanish) and the study was approved by the human studies committees at the Brigham and Women's Hospital and Boston Medical Center. Of the subjects enrolled in the study, 643 were randomly selected to have their child's cord blood IgE collected. Data imputation was conducted to account for all missing predictors**.**

The low baseline participant response rates, the subsequent attrition, and missing data are limitations of this sample and warrant further discussion. Sociodemographic and health-related information from the screener permitted analyses on potential sample differences between those who filled out the screener and those in the longitudinal study. Results (presented in Table 2.A) of mean scores and t-tests revealed no significant differences between the two groups.

Though the subsample of 643 respondents with available cord blood were chosen at random, and should therefore be expected to exhibit no significant difference from the larger longitudinal study, similar analyses were nevertheless conducted on the two groups

to test for potential differences. We also compared a restricted sample of those with no missing data on interpersonal violence (n=858) (the key predictor variable) versus the larger longitudinal sample. For both comparisons, the more detailed nature of the longitudinal study allowed us to assess disparities in all predictor variables. Table 2.B identified few statistically significant differences between the two subsamples and the longitudinal study, which, though minor, should be noted. These differences included a greater concentration of Hispanics, a lower concentration of whites, and more homes with detectable dust mites, in the IgE subsample versus the longitudinal sample. For the violence-completed subsample (those with no missing data on violence exposure), a greater percentage reported their race as "other/missing," were born outside of the United States, and had detectable dust mites in their homes, compared to the longitudinal study.[2]

In spite of the sample attrition, this data set offers several advantages. The study contains a range of information about psychosocial factors and environmental factors associated with asthma, including socioeconomic status, interpersonal violence (sexual, physical, psychological), community violence, other negative life events (unemployment, housing stress, and relationship difficulties), maternal and household smoking characteristics, and samples of in-home dust and cockroach allergen levels. Such extensive measures, in addition to the collected information about the mothers' medical histories, allow us to control for a range of confounding factors with greater accuracy than typically available. Moreover, because of the project's affiliation with the WIC programs, we have better access to hard-to-reach and typically underrepresented

[2] More pronounced differences emerged in terms of the overall percentage of missing information, evident in Table 2.C. In reference to the longitudinal study, the IgE subsample had more data missing for the following independent variables: maternal education, abuse during childhood, abuse during teenage years, drinking during pregnancy, smoking during pregnancy, and dust mites. The violence-completed subsample had more data missing for the baby's sex and number of stressful life events.

populations particularly vulnerable to asthma. The data also offer biomarkers of early-life asthma vulnerability, namely, IgE levels, for a sub-cohort of women and their children. Finally, the study's longitudinal design enables us to establish temporal priority between maternal reports of violence and subsequent cord blood IgE levels.

Measures

Dependent Variable

Total Serum Cord Blood IgE Levels. Serum samples from infant cord blood were analyzed for total IgE antibodies, reported in IU/mL. Cord blood was analyzed using CAP fluorescent enzyme immunoassay (Pharmacia, Uppsala, Sweden). A modified protocol was used in determining cord blood total IgE, reducing the lower limit of detection (LLOD) from 2.0 IU/ml to 0.2 IU/ml as previously described (Platts-Mills, Erwin, Allison, Blumenthal, and Barr 2003). Cord blood concentrations were log transformed to address issues of normality.

Various cut-off points have been used to define elevated IgE levels in cord blood that may more likely indicate increased asthma risk (Hansen, Halken, Host, Moller, and Osterballe 1993; Liu, Wang, Chuang, Ou, Hsu, and Yang 2003; Scirica, Gold, Ryan, Abulkerim, Celedón, Platts-Mills, Naccara, Weiss, and Litonjua 2007; Tariq, Arshad, Matthews, and Hakim 1999). However, because there is no established level for total cord blood IgE that consistently predicts asthma risk in epidemiologic studies, IgE levels were divided into quartiles, with serum levels at or above the upper 25% (1.07 IU/mL) considered to be "high." Sensitivity analyses using higher and lower cut-off values were also conducted to assess the robustness of the findings.

Key Predictors – Violence Exposure Measures

Proximal Abuse: Proximal abuse, defined dichotomously, was assessed using the Revised Conflict-Tactics Scale (R-CTS) (Straus and Douglas 2004), the most widely used reliable and validated instrument to identify intimate partner violence (IPV). Respondents were asked whether, during their index pregnancy, anyone had pushed, grabbed, or shoved them; kicked, bit, or punched them; hit them with something that hurt their body; choked or burned them; forced them to have sexual activities; or physically attacked them in some other way. An affirmative response to *any* of the delineated types of abuse met the criteria for proximal abuse.

Abuse in Prior Time Periods: In addition to abuse during pregnancy, respondents were also asked to recount their experiences with abuse during three prior periods: childhood, adolescence, and adulthood before the pregnancy. For each of these designated periods, the respondent answered identical questions, using the R-CTS instrument. Three dummy variables were constructed. Childhood abuse was coded as 1 if the respondent experienced any of the above abuse types (e.g., being pushed, grabbed, or shoved; kicked, bit, or punched, etc.) before age 12. Adolescent abuse similarly captured any abuse occurring between ages 12 and 17; and adult abuse captured abuse between ages 18 and the index pregnancy.

Lifetime History of Abuse: We constructed a lifetime history of abuse based on the total number of time periods (1 through 4) in which a study participant reported experiencing abuse, as defined above: childhood, adolescence, adulthood before the

index pregnancy, and during the index pregnancy. Because of the small cell sizes, the categories were then collapsed to create a categorical measure (0=no abuse, 1=1 to 2 time periods, 2= 3 or more periods).

Control Variables

Sociodemographic Factors. We adjusted for a number of variables that have been associated with cord blood IgE and/or violence exposure. Minorities and individuals of low SES experience higher rates of family violence than their higher SES counterparts (Cunradi, Caetano, and Schafer 2002; Field and Caetano 2005; Fox, Benson, DeMaris, and Wyk 2002) and are more likely to have elevated IgE (Scirica et al. 2007) or engage in asthma-inducing health behaviors (e.g., smoking) (Shohaimi, Luben, Wareham, Day, Bingham, Welch, Oakes, and Khaw 2003; Tseng, Yeatts, Millikan, and Newman 2001). Moreover, they are more likely to experience other stressors (i.e., community violence, poverty, food insecurity) (Williams and Jackson 2005) that may also heighten IgE levels (Pike, Smith, Hauger, Nicassio, Patterson, McClintick, Costlow, and Irwin 1997; Wright, Mitchell, Visness, Cohen, Stout, Evans, and Gold 2004a). Immigration status has been shown to be an important protective factor in the development of asthma and allergy, with US-born Mexican American children exhibiting higher asthma rates than their Mexican-born Mexican American counterparts (Eldeirawi, McConnell, Freels, and Persky 2005; Gold and Acevedo-Garcia 2005). As such, we controlled for maternal race (categorized as white/other, black, or Hispanic), maternal education (categorized into less than high school, high school degree, and some college or more), nativity status (categorized into US-born, moved to US before age 18, and moved to US at 18 or older)

and financial strain, a three-item subjective assessment of one's finance, found to be independently predictive of a range of physical and mental health outcomes in previous research (Conger, Conger, Elder, Lorenz, Simons, and Whitbeck 1992; Conger, Lorenz, Elder, Simons, and Ge 1993). Financial strain was constructed from responses to the following questions: a) How difficult is it (1=not difficult at all, 5=extremely difficult) for you to live on your total household income right now? b) In the next two months, how likely is it (1=not at all likely, 5=extremely likely) that you and your family will experience actual hardships, such as inadequate housing, food, or medical attention? c) In the next two months, how likely is it that you and your family will have to reduce standard of living to the bare necessities in life? Responses were divided into three categories: high financial strain (if the respondent scored a 4 or above on any single item — corresponding to "very difficult" or "extremely difficult"/ "very likely" or extremely likely"), moderate financial strain (a score of 2 or 3 for all items —corresponding to "somewhat difficult" or "difficult"/ "somewhat likely" or "likely"), and no financial strain (a value of 1 on all item questions—corresponding to "not at all difficult"/ "not at all likely").

Since higher total CB IgE levels have been reported among male children (Kulig, Tacke, Forster, Edenharter, Bergmann, Lau, Wahn, Zepp, and Wahn 1999; Scirica et al. 2007), children born to younger women (Bergmann, Schulz, Gunther, Dudenhausen, Bergmann, Bauer, Dorsch, Schmidt, Luck, Lau, Grass, and Wahn 1995; Karmaus, Arshad, Sadeghnejad, and Twiselton 2004; Scirica et al. 2007), and first-born children (Sunyer, Ant, Harris, Torrent, Vall, Cullinan, and Newman-Taylor 2001), we adjusted for child's sex (coded as 1 if male), maternal age (in years), and maternal parity

(categorized into no children, 1 child, 2 children, and 3 or more). Finally, we controlled for gestational age at time of survey (in weeks), because women have been shown to become less physiologically responsive to stress as their pregnancy advances, suggesting that the effects of fetal exposure to violence on CB IgE early in pregnancy may be more pronounced than those experienced later in pregnancy (Glynn, Wadhwa, Dunkel-Schetter, Chicz-Demet, and Sandman 2001; Liu et al. 2003).

Maternal Factors. We adjusted for smoking during pregnancy (coded as 1 if smoked during pregnancy) and maternal history of atopic disease (defined as ever being diagnosed with asthma, hay fever, or eczema) because of their associations with elevated CB IgE (Bergmann et al. 1995; Hanrahan, Tager, Segal, Tosteson, and Castile 1992; Liu et al. 2003; Noakes, Holt, and Prescott 2003; Scirica et al. 2007) and stressful or violent experiences (Beckham, Roodman, Shipley, Hetzberg, and Cunha 1995; Ganz 2000; Lucas 1999; Wright, Cohen, and Cohen 2005). We also controlled for drinking during pregnancy, because of its higher prevalence among victimized women and its association with adverse pregnancy outcomes (i.e. low birth weight, preterm births) (Cokkinides, Coker, Sanderson, Addy, and Bethea 1999). The drinking patterns were divided into four categories: 0 drinks/week, 1 drink/week, 2 to 6 drinks/week, 7 or more drinks/week.[3]

Other life stressors that may confound the relationship between violence and IgE were measured using the Crisis in Family Systems-Revised (CRISYS-R). Validated in both English and Spanish, the CRISYS is a 63-item instrument designed to capture the

[3] In addition to their alcohol consumption during pregnancy, survey participants were also asked to assess their drinking habits in the period prior to knowledge of their current pregnancy. Responses to these questions were incorporated in the measure of drinking during pregnancy. Two factors motivated this decision. First, alcohol exposure is most harmful for the fetus in the earliest stages of gestation, when the pregnancy may still be undetected. Failing to measure drinking patterns in the period prior to knowledge of pregnancy may overlook key health behaviors affecting fetal health. Second, there is widespread stigma attached to alcohol consumption while pregnant. Respondents may be less likely to underreport their alcohol consumption prior to knowledge of pregnancy than to admit to willfully drinking during pregnancy.

stressful life events of vulnerable and low-income communities (Berry, Quinn, Portillo, and Shalowitz 2006; Shalowitz, Berry, Rasinski, and Dannhausen-Brun 1998). The CRISYS-R has been shown elsewhere to be predictive of maternal mental health and their children's asthma morbidity (Shalowitz, Mijanovich, Berry, Clark-Kauffman, Quinn, and Perez 2006). Participants indicated whether they experienced a list of events that span several domains (safety, finances, community career, death, relationships, medical issues, home issues, authority, drug use, and child delinquency) in the last six months. The total number of events designated as "negative" by the respondent (with the exception of those relating to home safety, which were measured separately) was summed and divided into four categories (no stressful life events, 1 to 2, 3 to 4, 5 or more stressful life events).

Community Violence. Similar to IPV, exposure to community violence may cause physiological disruptions relevant to asthma onset (Wright 2006). Moreover, high levels of neighborhood violence can influence and increase the risk and co-occurrence of IPV (Little and Kantor 2002). To control for this potential confounder, we extracted the following eight questions on community violence from the CRISYS-R instrument (discussed above): 1) Did anything happen in your neighborhood to make you feel unsafe? 2) Were you a victim of a crime while you were outside or away from your home? 3) Did you hear violence outside your home (e.g. gunfire)? 4) Did you see violence? 5) Did your children see violence? 6) Was your child a victim of a crime? 7) Was anyone else in your household a victim of a crime? 8) Did you see drug dealing in your building or neighborhood? The total "yes" responses were collapsed into four categories (no violence, 1 violent event, 2 to 3 violent events, 4 or more violent events).

Environmental Factors. Environmental allergens tied to elevated IgE, such as cockroach and dust mite, are more prevalent in low-income populations who may be more likely to experience violence (Wright and Subramanian 2007). Several studies have demonstrated that even low levels of mite and cockroach allergens are significant risk factors for sensitization (Heinrich, Bolte, Holscher, Douwes, Lehmann, Fahlbusch, Bischof, Weiss, Borte, and Wichmann 2002; Huss, Adkinson, Eggleston, Dawson, Van Natta, and Hamilton 2001). Settled dust was collected during pregnancy from the mother's bed and bedroom floor using a standardized protocol (Chew 1999). Vacuumed dust and cockroach samples were collected at home site visits by trained research assistants. Briefly, all layers of bedding and 2 m^2 of the adjacent floor were vacuumed for 5 minutes each. Dust mite allergen was measured by monoclonal antibody enzyme-linked immunosorbent assay (ELISA) (Indoor Biotechnologies, Charlottesville, VA). Household dust mite allergen levels was categorized as 'low' for concentrations < LLOD (0.02 μg/g) and 'high' ≥ LLOD. We therefore measured prenatal exposure to home allergens through two dummy variables: dust mite allergen (defined as a detectable level [20ng/G] or greater of either Der p or Der f allergens found in the home) and cockroach allergen (defined as a detectable level [0.4 U/g] or greater of Bla g 2).

Childhood SES. Living in poverty is a risk factor both for experiencing IPV over the life course (Fox, Benson, DeMaris, and Wyk 2002) and for increased risk of asthma in these mothers (Gold and Wright 2005). Maternal atopy/asthma, in turn, is a risk factor for elevated IgE in children at birth, as noted above. Mothers were asked to report whether their parents owned their home over three periods during their childhood (age birth to 5 years, 6 to 10 years, and 11 to 15). It is increasingly recognized that housing

status is a robust marker of economic circumstances,[4] one that correlates with other conventional indicators of SES (e.g., income, assets), but housing status can be retrospectively reported with a much higher degree of accuracy compared to other traditional indicators of SES (Cohen, Doyle, Turner, Alper, and Skoner 2004). Moreover it has been shown to be a significant predictor of later life outcomes (adolescence and adulthood) including physiological disruption (Cohen et al. 2004; Miller and Chen 2007; van de Mheen, Stronks, and Mackenbach 1998). A binary variable was created to indicate parental home ownership (yes/no) over the participant's childhood (ages 0-15 years) as an indicator of childhood SES.

Analytic Strategy

The analysis proceeded in two stages. The aim of the first stage was to examine the association between proximal abuse and cord blood IgE levels. An initial binary logistic regression model estimated the unadjusted relationship between proximal abuse exposure and elevated IgE. Standard sociodemographic covariates and other confounders were added in a stepwise fashion to assess a) whether violence exposure was a proxy for demographic factors tied to asthma onset (i.e., poverty, race) and b) whether any association between violence exposure and IgE level was in part driven by other correlated factors.

[4] The use of parental home ownership as a measure of childhood SES may prove potentially problematic for non-US born participants since the cost/value of buying a home may vary between countries. In Mexico, for instance, even quite poor residents may own their own homes, in which case, home ownership would be a poor proxy for SES. We addressed this concern by adjusting for immigration status in our analyses.

The goal of the second stage was to identify whether the *accumulation* of violence exposure over the mother's life course, rather than abuse during any *specific* time period, would be predictive of IgE. To accomplish this, we first tested whether abuse during each of the designated time periods (adult, teen, childhood) independently predicted elevated IgE. Four sets of regression models identical to those in stage 1 were estimated, with the first set focusing on adult abuse, the second, teenage abuse, the third, childhood abuse, and the fourth, incorporating all four measures of abuse (proximal, adult, teen, and childhood abuse).

Next, we examined the relationship between cumulative abuse exposure and cord blood IgE level. The analysis proceeded in a similar fashion, with the baseline model regressing IgE levels on chronic abuse history, and subsequent models incorporating the relevant covariates. All results were presented as log odd coefficients.

In addition to these analyses, a series of sensitivity analyses was conducted to test the robustness of the findings. An interaction term between gestational age and proximal abuse was added to the first-stage models to assess whether the effect of abuse was stronger for women interviewed at later stages of their pregnancy, who had more "opportunities" for abuse during their pregnancy, compared to those interviewed early on. Additionally, all analyses were rerun using alternative IgE cut-points of the top 20% and top 33%.

Results

Table 2.1 describes sample characteristics. As shown, 25 % (n=161) of the women had offspring with cord blood IgE levels at or above 1.07. The majority of the

sample did not experience proximal abuse; only five percent of the subjects reported abuse during pregnancy, a prevalence rate consistent with other hospital or clinic-based samples of pregnant women (Martin, Mackie, Kupper, Buescher, and Moracco 2001). Reflective of the general U.S. population, 20% reported abuse during adulthood (Field and Caetano 2005), 28% reported abuse during childhood; and 25%, during their teenage years. In terms of lifetime prevalence, some 45% recounted one or more experiences of abuse throughout their life. The average subject was 26 years old, married, approximately 29 weeks pregnant at the time of interview, Hispanic, and relatively uneducated (64% with a high school degree or less). About 44% were born in the United States, and another 16% immigrated as a child. Over half reported that their parents owned a home when they were growing up (56%). In terms of current maternal risk factors, approximately a third of the sample reported a lifetime diagnosis of maternal atopy, 14% smoked during pregnancy, 16% drank moderately or heavily, and approximately 20% reported living in a violent neighborhood. Respondents experienced, on average, 2.1 stressful life events. Finally, almost 80% had detectable dust mite levels and 20% had detectable cockroach levels in their mothers' bedroom.

[Table 2.1 about here]

Proximal Abuse and IgE

Table 2.2 presents log odds of elevated cord blood IgE by mother's proximal abuse history (i.e., during pregnancy). Contrary to our first hypothesis, proximal abuse was not significantly related to elevate IgE levels in the unadjusted analysis. Adjusting for standard sociodemographic controls and maternal risk factors had no influence on

proximal violence effects. Also surprising, only three of the covariates significantly predicted IgE, a finding inconsistent with the literature on *in utero* exposures and IgE. High financial strain yielded independent, positive effects on IgE, corresponding to an over twofold increase (log coefficient of 0.78), whereas parental home ownership produced a 60% reduction in elevated IgE risk (log coefficient of 0.86). Maternal atopy was associated with cord blood IgE at borderline significant ($p<0.09$). While not significant, the effects for gender and maternal age were in the expected direction.

[Table 2.2 about here]

Tables 2.3 and 2.4 present a series of sensitivity analyses to test the robustness of the results. Table 2.3 utilizes alternative cut-points for IgE (top tertile and top 20%), and Table 2.4 adds an interaction term between gestational age and proximal abuse. No substantive differences emerged in either table. Evident from Table 2.3, columns 1 and 6, the alternative IgE cut-points yielded no change in the primary findings; proximal abuse continued to remain uncorrelated with IgE.

[Table 2.3 about here]

Likewise, neither the main effects for gestational age and proximal abuse, nor the interaction term, were significant in Table 2.4, suggesting that the relationship between IgE and proximal abuse did not vary based on duration of pregnancy.

[Table 2.4 about here]

Lifetime Abuse History: Independent or Cumulative Effects

In Table 2.5, columns 1-6, we regressed elevated cord blood IgE on abuse during adulthood, teenage years, and childhood, respectively, to test whether abuse during any of the indexed periods independently predicted IgE (i.e., were there critical periods of development when exposure may be more salient). A final model (columns 7 and 8) incorporated all three abuse measures, plus proximal abuse, as independent covariates. Similar to abuse during pregnancy, adulthood (columns 1 and 2) and childhood abuse (columns 3 and 4) were uncorrelated with IgE. In contrast, abuse during teenage years (columns 5 and 6) increased the likelihood of elevated IgE by about 75% (a log coefficient of 0.57). However, the effect for teenage abuse attenuated to non-significance with the inclusion of all abuse measures (columns 7 and 8).[5] As with proximal abuse, high levels of financial strain and home ownership were both significantly correlated with IgE in the expected direction in all models, though the effect size for financial strain was only borderline significant.

[Table 2.5 about here]

Table 2.6 shows results from analyses on cord blood IgE and lifetime abuse history. Strikingly different findings emerged. The bivariate analyses revealed a substantial increase in risk of elevated IgE with each abuse rank order. Experiencing abuse in one or two time periods increased the likelihood of elevated IgE by 75% (a log

[5]Given that prior abuse at any age is a risk factor for future abuse, there is the possibility that the absence of an effect for teenage abuse, evident in models 7 and 8, is due to collinearity with one or more of the other abuse measures. However, additional analyses revealed moderate to low correlations between teenage abuse and abuse during pregnancy, childhood, and adulthood (correlations of 0.17, 0.43, and 0.28, respectively). As such, it seems unlikely that the effects are driven by a statistical artifact.

coefficient of 0.56); experiencing three or more periods of abuse increased the risk by an additional 44% (a log coefficient of 0.93). Adjusting for standard sociodemographic, maternal, and environmental factors further increased the effect size (model 5). Consistent with the prior findings, parental home ownership predicted elevated IgE in the expected direction. Financial strain, however, yielded no significant effect.

[Table 2.6]

Figure 2.1 further illustrates the graded relationship between cumulative maternal history of interpersonal violence and elevated cord blood IgE. Based on estimates from the fully adjusted model, the figure shows an increase in the predicted probability of elevated IgE with each abuse category ($P<0.000$).

[Figure 2.1 about here]

Once again, using alternative cut-points for IgE produced no meaningful change in our findings. Childhood and adult abuse continued to be non-significant, whereas teen abuse was significant in the intermediate, but not fully adjusted model (Table 2.3). Likewise, lifetime history abuse remained significantly correlated with elevated IgE in a graded fashion (Table 2.7), though the magnitude of the effect varied slightly with differing cut-points.

[Table 2.7 about here]

Discussion

This is the first study to examine the relationship between maternal experiences of interpersonal violence over her life course and fetal cord blood IgE. Our results show a

graded association between lifetime exposure to violence and cord blood IgE, a biological marker for future potentiated atopic asthma risk. Greater exposure to violence throughout one's life course was independently associated with increased risk of offspring elevated IgE, after simultaneously adjusting for maternal sociodemographics (including current and childhood SES), and behavioral, psychosocial, and environmental exposures, suggesting that violence was not simply a marker for these other factors.

The association between maternal lifetime exposure to violence and offspring IgE indicates that the detrimental effects of interpersonal violence may not only accumulate over the mother's life course, but also transmit across generations through the fetal environment. These findings add to an emerging literature linking traumatic stressors (e.g., violence) to asthma expression (Clougherty, Levy, Kubzansky, Ryan, Suglia, Canner, and Wright 2007; Wright 2006; Wright, Hanrahan, Tager, and Speizer 1997; Wright et al. 2004a; Wright and Steinbach 2001). Notably, our results provide some of the first empirical evidence that maternal experiences of violence may alter fetal immune developmental processes relevant to asthma etiology (Barker 1997; Barker, Gluckman, Godfrey, Harding, Owens, and Robinson 1993; Ben-Shlomo and Kuh 2002; Welberg and Seckl 2001).

Interestingly, we found no association between proximal abuse (during pregnancy) and elevated IgE in the unadjusted and adjusted models. One possible explanation is that acute abuse and chronic abuse have different biological consequences for IgE. This distinction is further bolstered by the notable lack of association between IgE and abuse at each given time period. The results suggest that the *cumulative* exposure to violence over all ages, rather than any specific abuse experience, may be the

most salient factor in fetal effects, a notion consistent with our conceptualization of violence as a pervasive but extreme stressor. As with other life stressors, exposure to violence may disrupt physiological reactions characteristic of a stress response (dysregulation of the HPA axis and the SAM system). While short-term dysregulation (i.e., limited to a single occasion or age range) may be adaptive, the continuous or repeated activation of the stress response (i.e. over multiple age ranges) can result in long-term bodily damage. Though some studies reveal that sporadic or acute episodes of abuse can generate long-lasting responses for the victim (Baum, Cohen, and Hall 1993), other research indicates that the frequency of violent experiences over the life course is the most robust predictor of adverse biological markers (Chen, Fisher, Bacharier, and Strunk 2003; Murali and Chen 2005). Such an interpretation would suggest that victimization need not occur *during* the index pregnancy in order to adversely affect the fetus. Likewise, for women with no prior history of violence exposure, physical/sexual abuse during pregnancy may in fact have minimal consequences for cord blood IgE levels.

Another possibility is that the results for proximal abuse are due to a lack of statistical power. Recall that only 21 subjects reported abuse during pregnancy, a sample size too small to detect statistical significance. Further research using a greater sample size will prove useful in clarifying whether or not these results reflect substantive differences or statistical relics.

Independent of their abuse history, current SES, smoking behavior, life stressors, or community violence, subjects raised in homes owned by their parents were less likely to have offspring with elevated IgE, compared to those raised in rented homes. This is in

agreement with other evidence linking unfavorable SES circumstances early in life (as measured by home ownership) with a heightened vulnerability to respiratory and cardiovascular diseases in adulthood (Chen, Fisher, Bacharier, and Strunk 2003; Chen, Hanson, Paterson, Griffin, Walker, and Miller 2006; Cohen et al. 2004; Miller and Chen 2007), even when adjusting for health behaviors, life stress, or adult SES. Our findings are unique in suggesting that the physiological effects of childhood SES, at least in terms of elevated IgE, may have transgenerational implications. While the mechanisms underlying this process are not well understood, extant research indicates that adverse early life SES may program biological systems, resulting in pro-inflammatory epigenetic processes that prime the body to later life respiratory infections or inflammatory diseases (Chen et al. 2006; Miller and Chen 2007). Additional research will be useful to further elucidate the socio-psycho-biological pathways linking maternal childhood SES and offspring-IgE.

In lieu of the persistent home ownership-IgE effect, the overall non-significance of the remaining covariates (i.e. gestational age, maternal atopy, smoking during pregnancy) is particularly striking. Such findings are inconsistent with past studies linking elevated cord blood IgE to factors such as maternal smoking during pregnancy (Noakes, Holt, and Prescott 2003), dust and cockroach exposure (Heinrich et al. 2002), and maternal parity(Sunyer et al. 2001). One possible explanation for the null effect is the large proportion of missing data for many variables (i.e. drinking during pregnancy, stressful life events), which may diminish the statistical power necessary to identify significance. The nonsignificance may also be due, at least in some cases, to the definition of elevated IgE; maternal atopy, for instance, became significant when a top

tertile cut-off replaced the top 20% cut-off for "high" IgE. Given these potential methodological challenges—and the incongruity of the null results with the current literature—the findings should be interpreted cautiously.

The study has several limitations. First, because the sample does not represent a random selection of mothers, the results may be subject to selection bias. All participants were recruited from either prenatal care units at hospitals or WIC sites. These mothers may differ systematically from those who do not receive prenatal care, are not participants in WIC, or chose not to participate in the study. Additionally, the sample faced significant attrition and missing data on abuse measures. While sensitivity analyses (noted above) revealed no significant attrition or selection biases, we cannot rule out systematic differences due to the missing data.

Second, measures of violence victimization are self-reported and retrospective. Though several studies indicate the accuracy of retrospectively obtained abuse histories, the potential for recall bias remains (Brewin, Andrews, and Gotlib 1993; Maughan and Rutter 1997; Paivio 2001). Similarly, the use of survey questions, especially in such a sensitive area, raises the possibility of social desirability response bias.

Third, measuring violence victimization is challenging. Any one type of abuse can range vastly depending on the age of initial onset, the frequency, and chronicity. Though the use of widely accepted, validated scales of violence hopefully minimizes measurement error, we recognize that any survey-based measure of violence necessarily sacrifices the personally-tailored and fine-grained assessment of a clinical diagnosis.

Future research could build on these findings by examining prenatal exposure to family violence within the context of community-level physical and social stressors.

Recent evidence suggests a synergistic effect between traffic-related air pollution and urban exposure to violence on urban asthma etiology (Clougherty et al. 2007). Though we asked about individual-level exposure to community violence, we did not assess it within a multilevel context. Additional studies should investigate whether *in utero* exposure to family violence operates multiplicatively with neighborhood violence such that victimized pregnant women living in socially toxic areas have offspring with the highest asthma rates.

Further research should also consider the role of proximal and lifetime psychological abuse for asthma pathogenesis. Psychological maltreatment in the form of humiliation, isolation, and disempowerment within a relationship has been linked to post-traumatic stress disorder (PTSD) (Basile, Arias, Desai, and Thompson 2004) and other adverse physical outcomes (Coker, McKeown, and Alerts 2000), and may be just as damaging as other types of violence (Claussen and Crittenden 1991; Coker, McKeown, and Alerts 2000; Egeland, Sroufe, and Erickson 1983). Given that the co-occurrence of abuse types may be more detrimental to health than any single form, future research should test for potential independent, additive, and multiplicative influences of multiple abuse types.

Conclusion

While past research has identified associations between violence and asthma morbidity (Wright 2006; Wright, Hanrahan, Tager, and Speizer 1997; Wright et al. 2004a; Wright and Steinbach 2001), minimal evidence exists on the relationship between violence and asthma etiology. Moreover, no prior studies have examined the influence of

prenatal stress and asthma risk in children or the intermediate mechanisms linking the two. Our work therefore contributes to the existing literature by providing some of the first empirical evidence that chronic experiences of abuse may have transgenerational implications for asthma susceptibility. We demonstrated that the detrimental effects of violence may a) accumulate over the life course and b) transmit across generations through the fetal environment. Such research may not only inform our understanding of the role of "critical windows" in asthma development, but provide some insight as to why asthma remains a leading cause of health disparities unexplained by physical environmental risk factors.

Table 2.1 Descriptive Statistics of ACCESS Sample

	Mean	SD
Elevated Cord Blood	0.25	0.43
Abuse at Life Stage		
During Pregnancy	0.05	0.21
During Adulthood	0.20	0.40
During Teen Years	0.25	0.43
During Childhood	0.28	0.45
Abuse Over Life Course		
No Abuse	0.56	0.50
1-2 Time Periods	0.35	0.48
3+ Time Periods	0.09	0.28
Maternal Age (Years)	26.45	5.46
Gestational Age	29.68	7.56
# Natural Births		
1	0.40	0.49
2	0.22	0.42
3 or more	0.15	0.36
Baby's Sex		
Male	0.51	0.50
Maternal Race		
White	0.04	0.19
Non-Hispanic Black	0.26	0.44
Hispanic	0.53	0.50
Other	0.17	0.38
Maternal Education		
High School Degree	0.32	0.47
Some College +	0.31	0.46
Financial Strain	1.96	0.74
No Financial Strain	0.21	0.40
Moderate Financial Strain	0.57	0.50
High Financial Strain	0.23	0.42
Marital Status		
Single	0.34	0.47
Divorced/Separated	0.04	0.20
Married	62.20	0.48
Nativity Status		
Born in US	0.44	0.50
Moved as Child	0.16	0.37
Moved as Adult	0.40	0.49

*Significantly different at $p<.05$

Table 2.1 Descriptive Statistics of ACCESS Sample (continued)

	Mean	SD
Drinking During Pregnancy		
No Drinks	0.67	0.47
1 Drink/Week	0.17	0.38
2-6 Drinks Week	0.13	0.34
7 or More Drinks/Week	0.03	0.16
Smoking During Pregnancy (yes)	0.14	0.34
CRISYS Life Events	2.12	2.35
Community Violence		
Infrequent Violence	0.14	0.35
Some Violence	0.18	0.39
Heavy Violence	0.03	0.18
Parental Home Ownership	0.56	0.50
Detectable Dust Mite	0.81	0.39
Detectable Cockroach Allergen	0.21	0.41
Maternal Atopy	0.36	0.48

*Significantly different at $p<.05$

Table 2.2 Log Odds of Elevated Cord Blood IgE by Proximal Abuse History[a] (n=643)

Predictors	1	2	3	4	5
Interpersonal Abuse					
During Pregnancy	0.41	0.14	0.31	0.31	0.56
	(0.48)	(0.51)	(0.55)	(0.55)	(0.56)
Gestational Age		-0.02	-0.01	-0.02	-0.02
		(0.02)	(0.02)	(0.02)	(0.02)
Maternal Age		-0.05*	-0.04	-0.04	-0.03
		(0.02)	(0.03)	(0.03)	(0.03)
Male Child		0.08	0.08	0.08	0.09
		(0.19)	(0.20)	(0.20)	(0.20)
# of Natural Children					
1		-0.18	-0.24	-0.26	-0.26
		(0.32)	(0.34)	(0.35)	(0.35)
2 or More		-0.02	-0.14	-0.17	-0.13
		(0.38)	(0.41)	(0.42)	(0.42)
Maternal Race (Black)					
White		-0.03	0.05	-0.06	-0.00
		(0.50)	(0.54)	(0.55)	(0.55)
Hispanic		-0.29	-0.21	-0.35	-0.30
		(0.49)	(0.56)	(0.57)	(0.58)
Other		-0.06	-0.03	-0.10	-0.07
		(0.58)	(0.62)	(0.63)	(0.63)
Maternal Education (<H.S)					
H.S. Degree		0.19	0.11	0.08	0.10
		(0.25)	(0.26)	(0.26)	(0.27)
Some College +		0.44	0.38	0.29	0.35
		(0.28)	(0.29)	(0.29)	(0.30)
Financial Strain (No strain)					
Some financial strain		-0.02	0.04	-0.06	-0.05
		(0.30)	(0.31)	(0.32)	(0.32)
Major financial strain		0.69*	0.83*	0.82*	0.78*
		(0.33)	(0.37)	(0.38)	(0.38)
Marital Status (Married)					
Single			0.10	0.14	0.14
			(0.25)	(0.25)	(0.25)
Divorced			0.27	0.23	0.30
			(0.54)	(0.54)	(0.55)
Nativity Status (Born in US)					
Moved as Child			-0.20	-0.14	-0.03
			(0.34)	(0.34)	(0.35)
Moved as Adult			-0.09	0.05	0.22
			(0.31)	(0.32)	(0.33)
Maternal Atopy			0.45	0.41	0.42
			(0.24)	(0.25)	(0.25)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.

** p<0.01, * p<0.05

Table 2.2 Log Odds of Elevated Cord Blood IgE by Proximal Abuse History[a] (n=643) (continued)

Predictors	1	2	3	4	5
Drinking During Pregnancy					
1 Drink/Week			0.45	0.49	0.51
			(0.37)	(0.37)	(0.37)
2-6 Drinks/Week			-0.09	-0.07	-0.14
			(0.47)	(0.48)	(0.48)
7 or More Drinks/Week			-0.31	-0.38	-0.45
			(1.14)	(1.14)	(1.15)
Smoking Dur. Preg.			-0.18	-0.13	-0.08
			(0.34)	(0.35)	(0.35)
Life Stressors (No Stressors)					
1-2 Stressors			-0.27	-0.08	0.01
			(0.29)	(0.30)	(0.31)
3-4 Stressors			-0.55	-0.49	-0.48
			(0.36)	(0.37)	(0.37)
5 or more Stressors			-0.53	-0.44	-0.42
			(0.39)	(0.40)	(0.40)
Community Violence					
Infrequent Violence			0.16	0.16	0.06
			(0.32)	(0.32)	(0.33)
Some Violence			-0.47	-0.50	-0.61
			(0.40)	(0.40)	(0.41)
Heavy Violence			0.41	0.63	0.59
			(0.64)	(0.65)	(0.66)
Housing Risk Factors					
Dust Allergens				0.20	0.25
				(0.32)	(0.32)
Cockroach allergen				-0.59	-0.60
				(0.33)	(0.33)
Parents Owned Home					-0.86**
					(0.25)
Constant	-1.10**	0.40	0.05	0.18	0.15
	(0.11)	(0.85)	(0.96)	(0.98)	(1.00)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.
** p<0.01, * p<0.05

Table 2.3 Relationship Between Abuse and Elevated IgE, with Alternative Cut-offs for IgE Levels (n=643)

	IgE Cord Blood: Cut-off at Top 33%					IgE Cord Blood: Cut-Off at Top 20%				
	1	2	3	4	5	6	7	8	9	10
Predictors	Pregnancy	Adulthood	Teen Years	Childhood	All Periods	Pregnancy	Adulthood	Teen Years	Childhood	All Periods
Interpersonal Abuse										
During Pregnancy	0.49				0.31	0.27				-0.10
	(0.53)				(0.55)	(0.62)				(0.64)
During Adulthood		0.29			0.07		0.64			0.50
		(0.29)			(0.32)		(0.33)			(0.35)
During Teen Years			0.58*		0.47			0.71*		0.59
			(0.27)		(0.29)			(0.30)		(0.33)
During Childhood				0.40	0.20				0.45	0.14
				(0.26)	(0.28)				(0.30)	(0.32)
Gestational Age	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01	-0.01
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Maternal Age	-0.04	-0.04	-0.03	-0.04	-0.03	-0.02	-0.03	-0.02	-0.02	-0.02
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Male Child	0.30	0.30	0.29	0.28	0.28	0.04	0.02	0.01	0.01	-0.01
	(0.19)	(0.19)	(0.19)	(0.19)	(0.19)	(0.22)	(0.22)	(0.22)	(0.22)	(0.22)
# of Natural Children										
1	-0.20	-0.21	-0.26	-0.23	-0.28	-0.12	-0.14	-0.21	-0.16	-0.24
	(0.33)	(0.33)	(0.33)	(0.33)	(0.33)	(0.38)	(0.39)	(0.39)	(0.39)	(0.39)
2 or More	0.17	0.17	0.13	0.17	0.12	0.06	0.00	-0.00	0.05	-0.04
	(0.36)	(0.36)	(0.36)	(0.36)	(0.36)	(0.45)	(0.46)	(0.46)	(0.45)	(0.46)
Maternal Race										
White	-0.14	-0.08	-0.04	-0.10	-0.06	-0.05	0.02	0.08	-0.02	0.12
	(0.52)	(0.52)	(0.52)	(0.52)	(0.52)	(0.58)	(0.58)	(0.59)	(0.58)	(0.59)
Hispanic	-0.38	-0.37	-0.29	-0.35	-0.30	-0.55	-0.52	-0.41	-0.52	-0.42
	(0.54)	(0.53)	(0.54)	(0.54)	(0.54)	(0.61)	(0.61)	(0.61)	(0.61)	(0.62)

** p<0.01, * p<0.05

Table 2.3 Relationship Between Abuse and Elevated IgE, with Alternative Cut-offs for IgE Levels (n=643) (continued)

	IgE Cord Blood: Cut-Off at Top 33%					IgE Cord Blood: Cut-Off at Top 20%				
	1	2	3	4	5	6	7	8	9	10
Predictors	Pregnancy	Adulthood	Teen	Childhood	All Periods	Pregnancy	Adulthood	Teen	Childhood	All Periods
Other	-0.14	-0.10	-0.05	-0.05	-0.07	-0.11	-0.10	0.00	-0.03	0.02
	(0.59)	(0.59)	(0.59)	(0.59)	(0.60)	(0.66)	(0.66)	(0.67)	(0.66)	(0.68)
Maternal Education										
H.S. Degree	-0.12	-0.12	-0.14	-0.12	-0.13	0.16	0.13	0.16	0.16	0.12
	(0.25)	(0.25)	(0.25)	(0.25)	(0.25)	(0.29)	(0.29)	(0.29)	(0.29)	(0.30)
Some College +	0.23	0.22	0.24	0.25	0.24	0.23	0.21	0.26	0.25	0.22
	(0.27)	(0.27)	(0.27)	(0.27)	(0.28)	(0.32)	(0.32)	(0.32)	(0.32)	(0.33)
Financial Strain										
Some strain	0.23	0.19	0.18	0.18	0.18	-0.13	-0.17	-0.18	-0.18	-0.20
	(0.29)	(0.29)	(0.29)	(0.30)	(0.30)	(0.34)	(0.34)	(0.34)	(0.34)	(0.35)
Major strain	0.81*	0.76*	0.72*	0.78*	0.71	0.56	0.45	0.44	0.51	0.35
	(0.36)	(0.36)	(0.36)	(0.36)	(0.36)	(0.40)	(0.41)	(0.41)	(0.41)	(0.42)
Marital Status										
Single	0.13	0.12	0.15	0.13	0.15	0.22	0.25	0.23	0.22	0.27
	(0.23)	(0.23)	(0.23)	(0.23)	(0.23)	(0.27)	(0.27)	(0.27)	(0.27)	(0.28)
Divorced	0.07	0.08	0.07	0.08	0.06	0.37	0.37	0.33	0.38	0.32
	(0.51)	(0.51)	(0.52)	(0.51)	(0.52)	(0.58)	(0.58)	(0.59)	(0.58)	(0.59)
Nativity Status										
Moved as Child	-0.16	-0.15	-0.15	-0.14	-0.12	0.05	0.12	0.06	0.09	0.12
	(0.32)	(0.32)	(0.32)	(0.32)	(0.32)	(0.37)	(0.38)	(0.38)	(0.38)	(0.38)
Moved as Adult	0.04	0.06	0.08	0.08	0.08	0.22	0.29	0.26	0.26	0.31
	(0.30)	(0.30)	(0.30)	(0.30)	(0.30)	(0.35)	(0.36)	(0.36)	(0.36)	(0.36)
Maternal Atopy	0.64**	0.63**	0.67**	0.62**	0.65**	0.47	0.49	0.51	0.45	0.52
	(0.23)	(0.23)	(0.23)	(0.23)	(0.23)	(0.26)	(0.27)	(0.27)	(0.27)	(0.27)

** $p<0.01$, * $p<0.05$

Table 2.3 Relationship Between Abuse and Elevated IgE, with Alternative Cut-offs for IgE Levels (n=643) (continued)

	IgE Cord Blood: Cut-off at Top 33%					IgE Cord Blood: Cut-Off at Top 20%				
	1	2	3	4	5	6	7	8	9	10
Predictors	Pregnancy	Adulthood	Teen	Childhood	All Periods	Pregnancy	Adulthood	Teen	Childhood	All Periods
Drinking During Pregnancy										
Infrequent	0.11	0.13	0.15	0.15	0.14	0.63	0.64	0.69	0.69	0.70
	(0.31)	(0.31)	(0.31)	(0.31)	(0.31)	(0.40)	(0.40)	(0.40)	(0.40)	(0.41)
Moderate	0.54	0.53	0.58	0.58	0.59	0.26	0.26	0.21	0.20	0.19
	(0.36)	(0.36)	(0.36)	(0.36)	(0.36)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)
Heavy Drinker	-0.59	-0.60	-0.61	-0.64	-0.66	-0.09	-0.09	-0.10	-0.13	-0.10
	(0.46)	(0.46)	(0.46)	(0.46)	(0.46)	(1.17)	(1.17)	(1.18)	(1.17)	(1.19)
Smoked While Pregnant	-1.11	-1.11	-1.12	-1.12	-1.12	-0.14	-0.15	-0.13	-0.11	-0.16
	(1.14)	(1.13)	(1.14)	(1.14)	(1.15)	(0.38)	(0.38)	(0.38)	(0.37)	(0.38)
Life Stressors										
1-2 Stressors	-0.10	-0.13	-0.14	-0.13	-0.13	-0.07	-0.12	-0.15	-0.10	-0.20
	(0.28)	(0.28)	(0.28)	(0.28)	(0.28)	(0.33)	(0.33)	(0.33)	(0.33)	(0.33)
3-4 Stressors	-0.44	-0.47	-0.51	-0.52	-0.53	-0.68	-0.72	-0.78	-0.77	-0.83*
	(0.34)	(0.34)	(0.34)	(0.34)	(0.35)	(0.40)	(0.41)	(0.41)	(0.41)	(0.42)
5 or more Stressors	-0.32	-0.32	-0.40	-0.36	-0.43	-0.43	-0.51	-0.56	-0.47	-0.61
	(0.36)	(0.37)	(0.37)	(0.37)	(0.37)	(0.43)	(0.43)	(0.44)	(0.43)	(0.44)
Community Violence										
Infrequent Violence	-0.10	-0.12	-0.17	-0.15	-0.20	-0.18	-0.22	-0.25	-0.23	-0.31
	(0.31)	(0.31)	(0.31)	(0.31)	(0.31)	(0.36)	(0.36)	(0.36)	(0.36)	(0.37)
Some Violence	-0.30	-0.31	-0.38	-0.33	-0.43	-0.59	-0.68	-0.70	-0.65	-0.78
	(0.37)	(0.37)	(0.37)	(0.37)	(0.38)	(0.44)	(0.45)	(0.45)	(0.44)	(0.46)
Heavy Violence	0.41	0.38	0.28	0.36	0.27	0.88	0.77	0.73	0.81	0.67
	(0.63)	(0.63)	(0.63)	(0.64)	(0.64)	(0.67)	(0.67)	(0.68)	(0.68)	(0.69)

** p<0.01, * p<0.05

Table 2.3 Relationship Between Abuse and Elevated IgE, with Alternative Cut-offs for IgE Levels (n=643) (continued)

	IgE Cord Blood: Cut-Off at Top 33%					IgE Cord Blood: Cut-Off at Top 20%				
	1	2	3	4	5	6	7	8	9	10
Predictors	Pregnancy	Adulthood	Teen	Childhood	All Periods	Pregnancy	Adulthood	Teen	Childhood	All Periods
Housing Risk Factors										
Dust Allergen	0.38	0.39	0.32	0.37	0.34	0.33	0.33	0.26	0.33	0.27
	(0.30)	(0.30)	(0.30)	(0.30)	(0.30)	(0.35)	(0.35)	(0.35)	(0.35)	(0.36)
Cockroach Allergen	0.12	0.10	0.17	0.17	0.19	-0.55	-0.60	-0.49	-0.47	-0.54
	(0.28)	(0.28)	(0.28)	(0.28)	(0.28)	(0.36)	(0.36)	(0.36)	(0.36)	(0.37)
Parents Owned Home	-0.64**	-0.62**	-0.63**	-0.59*	-0.64**	-0.78**	-0.79**	-0.78**	-0.74**	-0.80**
	(0.23)	(0.23)	(0.23)	(0.23)	(0.24)	(0.27)	(0.27)	(0.27)	(0.27)	(0.28)
Constant	-0.09	-0.04	-0.34	-0.19	-0.36	-0.72	-0.61	-1.03	-0.87	-0.88
	(0.91)	(0.91)	(0.93)	(0.92)	(0.93)	(1.07)	(1.08)	(1.09)	(1.08)	(1.11)

** p<0.01, * p<0.05

Table 2.4 Interaction of Proximal Abuse and Gestational Age (n=643)

	1	2
Predictors	No Interaction	With Interaction
Interpersonal Abuse	0.51	0.32
	(0.56)	(2.20)
Gestational Age	-0.02	-0.02
	(0.02)	(0.02)
Abuse*Gestational Age		0.01
		(0.07)
Maternal Age	-0.02	-0.02
	(0.02)	(0.02)
# Natural Children		
Male Child	0.11	0.11
	(0.20)	(0.20)
Maternal Race (Black)		
White	0.05	0.05
	(0.55)	(0.55)
Hispanic	-0.26	-0.26
	(0.58)	(0.58)
Other	-0.02	-0.02
	(0.63)	(0.63)
Maternal Education (<H.S)		
H.S. Degree	0.06	0.06
	(0.27)	(0.27)
Some College +	0.27	0.27
	(0.29)	(0.29)
Marital Status (Married)		
Single	0.14	0.14
	(0.25)	(0.25)
Divorced	0.25	0.24
	(0.54)	(0.54)
Nativity Status (Born in US)		
Moved as Child	-0.05	-0.05
	(0.35)	(0.35)
Moved as Adult	0.16	0.16
	(0.32)	(0.32)
Drinking During Pregnancy		
1 Drink/Week	0.52	0.53
	(0.37)	(0.37)
2-6 Drinks/Week	-0.13	-0.13
	(0.48)	(0.48)
7 or More Drinks/Week	-0.53	-0.53
	(1.15)	(1.15)

** $p<0.01$, * $p<0.05$

Table 2.4 Interaction of Proximal Abuse and Gestational Age (n=643) (continued)

	1	2
Predictors	No Interaction	With Interaction
Smoked While Pregnant	-0.10	-0.10
	(0.34)	(0.34)
Life Stressors (No Stressors)		
1-2 Stressors	-0.01	-0.01
	(0.30)	(0.30)
3-4 Stressors	-0.46	-0.46
	(0.37)	(0.37)
5 or more Stressors	-0.32	-0.32
	(0.39)	(0.39)
Financial Strain (No strain)		
Some Strain	-0.10	-0.10
	(0.32)	(0.32)
Major Strain	0.70	0.70
	(0.38)	(0.38)
Community Violence		
Infrequent Violence	0.09	0.09
	(0.32)	(0.32)
Some Violence	-0.54	-0.53
	(0.41)	(0.41)
Heavy Violence	0.67	0.67
	(0.66)	(0.66)
Parents Owned Home	-0.88**	-0.88**
	(0.25)	(0.25)
Dust Allergen	0.30	0.30
	(0.32)	(0.32)
Cockroach Allergen	-0.53	-0.53
	(0.33)	(0.33)
Maternal Atopy	0.44	0.44
	(0.24)	(0.24)
Constant	-0.20	-0.19
	(0.95)	(0.95)

** $p<0.01$, * $p<0.05$

Table 2.5 Log Odds of Elevated IgE by Abuse at Select Periods[a] (n=643)

	Adulthood		Teen Years		Childhood		All 4 Periods	
Predictors	1	2	3	4	5	6	7	8
Interpersonal Abuse								
During Pregnancy							0.07	0.28
							(0.50)	(0.58)
During Adulthood	0.48	0.61					0.30	0.46
	(0.26)	(0.31)					(0.29)	(0.33)
During Teen Years			0.57*	0.57*			0.46	0.46
			(0.24)	(0.28)			(0.27)	(0.31)
Childhood					0.34	0.29	0.06	0.01
					(0.23)	(0.28)	(0.27)	(0.31)
Gestational Age		-0.02		-0.02		-0.02		-0.02
		(0.02)		(0.02)		(0.02)		(0.02)
Maternal Age		-0.04		-0.03		-0.04		-0.03
		(0.03)		(0.03)		(0.03)		(0.03)
Male Child		0.07		0.07		0.07		0.06
		(0.20)		(0.20)		(0.20)		(0.21)
# of Natural Children								
1		-0.29		-0.33		-0.29		-0.35
		(0.35)		(0.36)		(0.35)		(0.36)
2 or More		-0.17		-0.17		-0.12		-0.21
		(0.42)		(0.42)		(0.42)		(0.42)
Maternal Race								
White		0.08		0.10		0.03		0.11
		(0.56)		(0.56)		(0.55)		(0.56)
Hispanic		-0.27		-0.19		-0.27		-0.21
		(0.58)		(0.58)		(0.57)		(0.58)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.

** p<0.01, * p<0.05

Table 2.5 Log Odds of Elevated IgE by Abuse at Select Periods[a] (n=643) (continued)

	Adulthood		Teen Years		Childhood		All 4 Periods	
Predictors	1	2	3	4	5	6	7	8
Other		-0.05		0.04		0.01		-0.02
		(0.63)		(0.63)		(0.63)		(0.64)
Maternal Education								
H.S. Degree		0.08		0.09		0.10		0.08
		(0.27)		(0.27)		(0.27)		(0.27)
Some College +		0.34		0.37		0.37		0.35
		(0.30)		(0.30)		(0.30)		(0.30)
Financial Strain								
Some Strain		-0.11		-0.10		-0.10		-0.11
		(0.32)		(0.32)		(0.32)		(0.33)
Major Strain		0.68		0.70		0.75		0.63
		(0.39)		(0.39)		(0.38)		(0.39)
Marital Status								
Single		0.14		0.14		0.13		0.17
		(0.25)		(0.25)		(0.25)		(0.25)
Divorced		0.32		0.29		0.32		0.27
		(0.54)		(0.55)		(0.54)		(0.55)
Nativity Status								
Moved as Child		0.01		-0.03		-0.02		0.02
		(0.35)		(0.35)		(0.35)		(0.35)
Moved as Adult		0.27		0.26		0.25		0.28
		(0.33)		(0.33)		(0.33)		(0.33)
Maternal Atopy		0.42		0.45		0.40		0.44
		(0.25)		(0.25)		(0.25)		(0.25)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.

** p<0.01, * p<0.05

Table 2.5 Log Odds of Elevated IgE by Abuse at Select Periods[a] (n=643) (continued)

	Adulthood		Teen Years		Childhood		All 4 Periods	
Predictors	1	2	3	4	5	6	7	8
Drinking During Pregnancy								
1 Drink/Week		0.50		0.54		0.54		0.54
		(0.38)		(0.38)		(0.37)		(0.38)
2-6 Drinks/Week		-0.14		-0.15		-0.16		-0.19
		(0.49)		(0.49)		(0.49)		(0.49)
7 or More Drinks/Week		-0.46		-0.49		-0.48		-0.48
		(1.15)		(1.16)		(1.15)		(1.16)
Smoked While Pregnant		-0.07		-0.05		-0.04		-0.08
		(0.35)		(0.35)		(0.35)		(0.35)
Life Stressors								
1-2 Stressors		-0.05		-0.05		-0.02		-0.07
		(0.31)		(0.31)		(0.31)		(0.31)
3-4 Stressors		-0.52		-0.56		-0.54		-0.57
		(0.38)		(0.38)		(0.38)		(0.38)
5 or more Stressors		-0.46		-0.51		-0.43		-0.55
		(0.40)		(0.41)		(0.40)		(0.41)
Community Violence								
Infrequent Violence		0.02		0.01		0.04		-0.03
		(0.33)		(0.33)		(0.33)		(0.33)
Some Violence		-0.68		-0.67		-0.62		-0.76
		(0.41)		(0.41)		(0.41)		(0.42)
Heavy Violence		0.49		0.45		0.54		0.40
		(0.67)		(0.68)		(0.67)		(0.68)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.
** $p<0.01$, * $p<0.05$

Table 2.5 Log Odds of Elevated IgE by Abuse at Select Periods[a] (n=643) (continued)

	Adulthood		Teen Years		Childhood		All 4 Periods	
Predictors	1	2	3	4	5	6	7	8
Housing Risk Factors								
Dust Allergens		0.25		0.19		0.24		0.20
		(0.33)		(0.33)		(0.32)		(0.33)
Cockroach allergen		-0.64		-0.55		-0.54		-0.60
		(0.34)		(0.33)		(0.33)		(0.34)
Parents Owned Home		-0.85**		-0.84**		-0.81**		-0.88**
		(0.25)		(0.25)		(0.25)		(0.26)
Constant	-1.19**	0.26	-1.24**	-0.10	-1.19**	0.05	-1.29**	0.04
	(0.13)	(1.00)	(0.13)	(1.01)	(0.13)	(1.00)	(0.15)	(1.02)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.

** p<0.01, * p<0.05

Table 2.6 Log Odds of Elevated IgE by Lifetime Abuse History[a] (n=643)

Predictors	1	2	3	4	5
Lifetime Exposure to Abuse					
1-2 Time Periods	0.56*	0.49*	0.68*	0.64*	0.63*
	(0.23)	(0.24)	(0.27)	(0.27)	(0.28)
3 or More Time Periods	0.93*	0.65	1.02*	1.01*	1.14*
	(0.37)	(0.40)	(0.45)	(0.45)	(0.46)
Gestational Age		-0.02	-0.01	-0.02	-0.02
		(0.02)	(0.02)	(0.02)	(0.02)
Maternal Age		-0.04	-0.04	-0.04	-0.03
		(0.02)	(0.03)	(0.03)	(0.03)
Male Child		0.08	0.05	0.06	0.06
		(0.19)	(0.20)	(0.20)	(0.21)
# of Natural Children					
1		-0.19	-0.31	-0.34	-0.37
		(0.32)	(0.35)	(0.35)	(0.36)
2 or More		0.14	0.05	0.04	0.04
		(0.35)	(0.38)	(0.38)	(0.39)
Maternal Race					
White		0.07	0.16	0.05	0.14
		(0.51)	(0.55)	(0.55)	(0.56)
Hispanic		-0.18	-0.12	-0.25	-0.19
		(0.50)	(0.57)	(0.57)	(0.58)
Other		0.02	0.05	-0.01	0.04
		(0.59)	(0.62)	(0.63)	(0.64)
Maternal Education					
H.S. Degree		0.15	0.06	0.05	0.06
		(0.25)	(0.26)	(0.26)	(0.27)
Some College +		0.40	0.35	0.27	0.33
		(0.27)	(0.29)	(0.29)	(0.30)
Financial Strain					
Some Strain		-0.15	-0.12	-0.22	-0.19
		(0.30)	(0.32)	(0.32)	(0.33)
Major Strain		0.51	0.64	0.61	0.57
		(0.34)	(0.38)	(0.38)	(0.39)
Marital Status					
Single			0.16	0.19	0.19
			(0.25)	(0.25)	(0.25)
Divorced			0.12	0.07	0.18
			(0.54)	(0.55)	(0.55)
Nativity Status					
Moved as Child			-0.13	-0.08	0.01
			(0.34)	(0.34)	(0.35)
Moved as Adult			-0.05	0.06	0.24
			(0.31)	(0.32)	(0.33)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.

** $p<0.01$, * $p<0.05$

Table 2.6 Log Odds of Elevated IgE by Lifetime Abuse History[a] (n=643) (continued)

Predictors	1	2	3	4	5
Maternal Atopy			0.40	0.36	0.37
			(0.25)	(0.25)	(0.25)
Drinking During Pregnancy					
1 Drink/Week			0.56	0.59	0.61
			(0.38)	(0.38)	(0.38)
2-6 Drinks/Week			-0.23	-0.20	-0.28
			(0.48)	(0.48)	(0.49)
7 or More Drinks/Week			-0.28	-0.39	-0.50
			(1.14)	(1.15)	(1.17)
Smoked While Pregnant			-0.15	-0.09	-0.03
			(0.34)	(0.35)	(0.35)
Life Stressors					
1-2 Stressors			-0.34	-0.17	-0.09
			(0.29)	(0.30)	(0.31)
3-4 Stressors			-0.72	-0.66	-0.65
			(0.37)	(0.37)	(0.38)
5 or more Stressors			-0.61	-0.50	-0.48
			(0.39)	(0.40)	(0.40)
Community Violence					
Infrequent Violence			0.04	0.04	-0.07
			(0.32)	(0.33)	(0.33)
Some Violence			-0.62	-0.64	-0.77
			(0.41)	(0.41)	(0.42)
Heavy Violence			0.27	0.47	0.40
			(0.66)	(0.67)	(0.68)
Dust Allergen				0.21	0.26
				(0.32)	(0.33)
Cockroach Allergen				-0.47	-0.46
				(0.33)	(0.33)
Parents Owned Home					-0.87**
					(0.25)
Constant	-1.40**	0.02	-0.34	-0.26	-0.32
	(0.16)	(0.87)	(0.97)	(0.99)	(1.01)

[a] Elevated cord blood is defined as top quartile (score of 1.07) of sample.
** p<0.01, * p<0.05

Table 2.7 Relationship Between Lifetime History of Abuse and Elevated IgE, with Alternative Cut-offs for IgE (n=643)

	Cut-off of 33%		Cut-off of 20%	
Predictors	Unadjusted	Adjusted	Unadjusted	Adjusted
Lifetime Exposure to Abuse				
1-2 Time Periods	0.46*	0.52*	0.81**	0.92**
	(0.21)	(0.25)	(0.25)	(0.30)
3 or More Time Periods	0.66	0.86*	0.98*	1.24*
	(0.35)	(0.43)	(0.39)	(0.50)
Gestational Age		-0.01		-0.01
		(0.02)		(0.02)
Maternal Age		-0.04		-0.02
		(0.02)		(0.03)
Male Child		0.32		0.02
		(0.19)		(0.22)
# of Natural Children				
1		-0.27		-0.27
		(0.33)		(0.39)
2 or More		0.13		-0.06
		(0.36)		(0.46)
Maternal Race				
White		-0.04		0.05
		(0.52)		(0.59)
Hispanic		-0.29		-0.45
		(0.54)		(0.62)
Other		-0.09		-0.04
		(0.59)		(0.67)
Maternal Education				
H.S. Degree		-0.17		0.14
		(0.24)		(0.29)
Some College +		0.18		0.23
		(0.27)		(0.33)
Financial Strain (No strain)				
Some financial strain		0.14		-0.25
		(0.30)		(0.35)
Major financial strain		0.69		0.40
		(0.36)		(0.42)
Marital Status				
Single		0.15		0.27
		(0.23)		(0.28)
Divorced		0.05		0.29
		(0.51)		(0.60)
Nativity Status				
Moved as Child		-0.15		0.15
		(0.32)		(0.38)

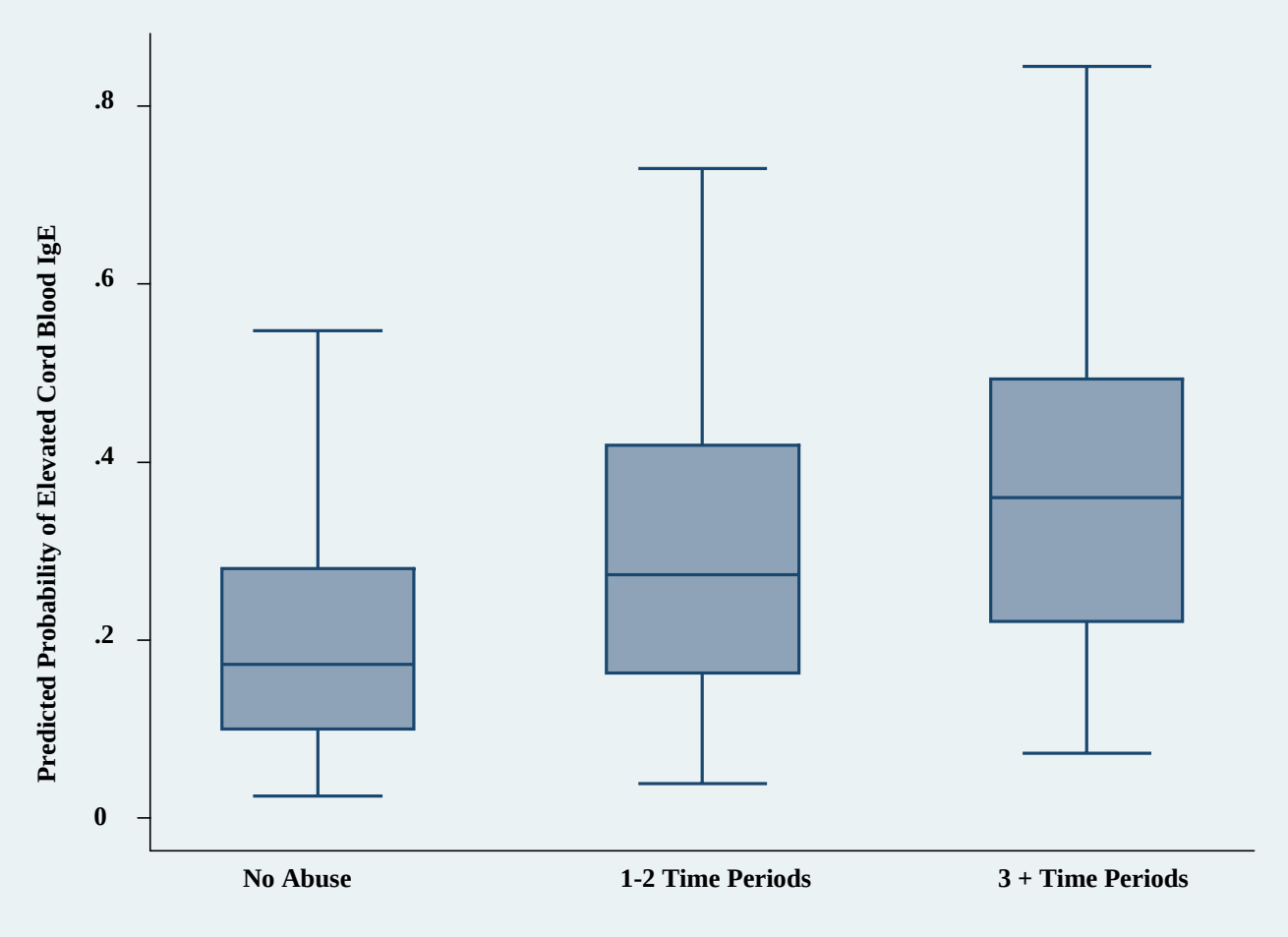
** p<0.01, * p<0.05

Table 2.7 Relationship Between Lifetime History of Abuse and Elevated IgE, with Alternative Cut-offs for IgE (n=643) (continued)

	Cut-off of 33%		Cut-off of 20%	
Predictors	Unadjusted	Adjusted	Unadjusted	Adjusted
Moved as Adult		0.06		0.31
		(0.29)		(0.36)
Maternal Atopy		0.65**		0.44
		(0.23)		(0.27)
Drinking During Pregnancy				
Infrequent		0.63		0.77
		(0.36)		(0.41)
Moderate		-0.63		0.09
		(0.45)		(0.50)
Heavy Drinker		-1.17		-0.11
		(1.13)		(1.18)
Smoked While Pregnant		0.10		-0.09
		(0.31)		(0.38)
Life Stressors				
1-2 Stressors		-0.18		-0.19
		(0.28)		(0.34)
3-4 Stressors		-0.53		-0.90*
		(0.34)		(0.42)
5 or more Stressors		-0.41		-0.62
		(0.37)		(0.44)
Community Violence				
Infrequent Violence		-0.22		-0.38
		(0.31)		(0.37)
Some Violence		-0.39		-0.83
		(0.37)		(0.45)
Heavy Violence		0.26		0.62
		(0.64)		(0.70)
Dust Allergen		0.39		0.28
		(0.30)		(0.36)
Cockroach Allergen		0.19		-0.44
		(0.28)		(0.36)
Parents Owned Home		-0.61**		-0.78**
		(0.23)		(0.27)
Constant	-0.92**	-0.39	-1.79**	-1.12
	(0.14)	(0.89)	(0.18)	(1.10)

** p<0.01, * p<0.05

Figure 2.1 Predicted Probability of Elevated Cord Blood IgE by Maternal Lifetime Exposure (n=643)



Note: All probabilities are based on adjusted, log transformed cord blood IgE scores. Trend significant at p<.01. High IgE= 1.07 IU/mL & above.

Table 2.A Comparison of Mean Scores for Screener vs. Longitudinal Samples

	Screener (n=1437)	SD	Longitudinal (n=1156)	SD	Sig. Diff.
	A		B		
Maternal Age (Years)	26.35	5.73	26.48	5.69	
# Natural Births					
0	0.26	0.44	0.26	0.44	
1	0.38	0.48	0.38	0.49	
2	0.22	0.42	0.22	0.41	
3 or More	0.14	0.35	0.14	0.35	
Maternal Race					
White	0.10	0.30	0.09	0.28	
Non-Hispanic Black	0.31	0.46	0.29	0.46	
Hispanic	0.45	0.50	0.47	0.50	
Other	0.15	0.35	0.15	0.36	
Maternal Education					
Less than 12	0.34	0.47	0.35	0.48	
High School Degree	0.33	0.47	0.32	0.47	
Some College +	0.34	0.47	0.33	0.47	
Marital Status					
Married	0.60	0.49	0.61	0.49	
Single	0.34	0.47	0.33	0.47	
Divorced/Separated	0.06	0.24	0.06	0.24	
Nativity Status					
Born in US	0.46	0.50	0.45	0.50	
Moved as Child	0.19	0.39	0.18	0.39	
Moved as Adult	0.35	0.48	0.37	0.48	
Drinking During Pregnancy					
None	0.62	0.49	0.63	0.48	
Infrequent	0.17	0.38	0.17	0.38	
Moderate	0.17	0.37	0.16	0.37	
Heavy Drinker	0.04	0.19	0.04	0.19	
Smoking					
No Smoking	0.82	0.38	0.83	0.38	
During Smoking	0.18	0.38	0.17	0.38	
Missing	0.09	0.29	0.09	0.29	
Perceived Stress Scale	1.35	0.80	1.35	0.79	

*Significantly different at p<.05

Table 2.B Comparison of Mean Scores for Longitudinal vs. CB IgE Sample and Violence Sample

	Longitudinal (n=1156)	SD	CB IgE (n=643)	SD		Violence Responses (n=858)	SD	
	A		B			C		
Maternal Age (Years)	26.49	5.69	26.45	5.46		26.65	6.01	
Gestational Age	29.84	7.88	29.68	7.56		29.83	7.87	
# Natural Births								
0	0.26	0.44	0.22	0.41		0.27	0.44	
1	0.38	0.49	0.40	0.49		0.37	0.48	
2	0.22	0.41	0.22	0.42		0.21	0.41	
3 or more	0.14	0.35	0.15	0.36		0.15	0.35	
Baby's Sex								
Male	0.52	0.50	0.51	0.50		0.51	0.50	
Maternal Race								
White	0.08	0.30	0.04	0.19	*	0.10	0.30	
Non-Hispanic Black	0.29	0.46	0.26	0.44		0.30	0.46	
Hispanic	0.47	0.50	0.53	0.50	*	0.50	0.50	
Other	0.15	0.36	0.17	0.38		0.10	0.30	+
Maternal Education								
Less than 12	0.35	0.48	0.37	0.48		0.35	0.48	
High School Degree	0.32	0.47	0.32	0.47		0.31	0.46	
Some College +	0.33	0.47	0.31	0.46		0.34	0.47	
Marital Status								
Married	0.61	0.49	0.62	0.49		0.63	0.48	
Single	0.33	0.47	0.34	0.47		0.31	0.46	
Divorced/Separated	0.06	0.24	0.04	0.20		0.06	0.24	
Nativity Status								
Born in US	0.45	0.50	0.44	0.50		0.39	0.49	+
Moved as Child	0.18	0.39	0.16	0.37		0.20	0.40	
Moved as Adult	0.37	0.48	0.40	0.49		0.41	0.49	
Abuse at Life Stage								
During Pregnancy	0.05	0.21	0.05	0.21				
During Adulthood	0.20	0.40	0.20	0.40				
During Teen Years	0.28	0.45	0.25	0.43				
During Childhood	0.31	0.46	0.28	0.45				
Abuse Over Life								
No Abuse	0.53	0.50	0.56	0.50				
1-2 Time Periods	0.38	0.49	0.36	0.48				
3+ Time Periods	0.09	0.28	0.09	0.28				
Drinking Dur. Preg								
None	0.63	0.48	0.67	0.47		0.63	0.48	
Infrequent	0.17	0.38	0.17	0.38		0.17	0.38	
Moderate	0.16	0.37	0.13	0.34		0.16	0.37	

*Significantly different at p<.05 between A & B
+Significantly different at p<.05 between A & C

Table 2.B Comparison of Mean Scores for Longitudinal vs. CB IgE Sample and Violence Sample (continued)

	Longitudinal (n=1156)	SD	CB IgE (n=643)	SD		Violence Responses (n=858)	SD	
	A		B			C		
Heavy Drinker	0.04	0.19	0.03	0.16		0.04	0.19	
Smoked While Preg.	0.17	0.36	0.14	0.34		0.16	0.37	
Life Stressors	2.12	2.43	2.12	2.35		2.10	2.67	
Financial Strain	1.94	0.75	1.96	0.74		1.93	0.81	
Community Violence								
No Violence	0.67	0.47	0.64	0.48		0.67	0.47	
Infrequent Violence	0.14	0.35	0.14	0.35		0.13	0.34	
Some Violence	0.16	0.37	0.18	0.39		0.16	0.37	
Heavy Violence	0.03	0.17	0.03	0.18		0.03	0.17	
Parental Home Ownership	0.57	0.50	0.56	0.50		0.58	0.49	
Dust Allergen	0.76	0.43	0.81	0.39	*	0.80	0.40	+
Cockroach Allergen	0.17	0.38	0.21	0.41		0.18	0.38	
Maternal Atopy	0.35	0.48	0.36	0.48		0.35	0.48	

*Significantly different at $p<.05$ between A & B
+Significantly different at $p<.05$ between A & C

Table 2.C Percent of Sample with Missing Data for All Predictors: Comparison Across Samples

	Screener (n=1437)	SD	Longitudinal Study (n=1156)	SD	CB IgE (n=643)	SD	Sig. Diff.	Violence Responses (n=858)	SD	Sig. Diff.
	A		A		B		A-B	C		A-C
Maternal Age	0.06	0.01	0.07	0.01	0.10	0.01		0.02	0.00	*
Gestational Age			0.30	0.01	0.32	0.02		0.06	0.01	
# Natural Births	0.32	0.46	0.33	0.47	0.36	0.48		0.30	0.46	
Baby's Sex			0.08	0.27	0.02	0.13	*	0.06	0.23	*
Maternal Education	0.08	0.28	0.10	0.30	0.14	0.35	*	0.05	0.21	*
Marital Status	0.11	0.31	0.12	0.32	0.14	0.35		0.07	0.25	*
Nativity Status	0.02	0.13	0.01	0.12	0.02	0.12		0.02	0.12	
Abuse at Life Stage										
During Pregnancy			0.27	0.44	0.30	0.46				
During Adulthood			0.30	0.46	0.33	0.47				
During Teen Years			0.27	0.44	0.31	0.46	*			
During Childhood			0.26	0.44	0.30	0.46	*			
Abuse Over Life Course			0.26	0.45	0.30	0.46	*			
Drinking During Pregnancy	0.49	0.50	0.50	0.50	0.54	0.50	*	0.47	0.50	
Smoking During Pregnancy	0.09	0.29	0.09	0.01	0.12	0.01	*	0.02	0.14	*
Life Stressors			0.30	0.01	0.30	0.02		0.35	0.02	*
Perceived Stress Scale	0.12	0.33	0.14	0.34						
Financial Strain			0.29	0.45	0.30	0.46		0.16	0.36	*
Community Violence			0.33	0.47	0.33	0.47		0.20	0.40	*
Parental Home Ownership			0.28	0.45	0.29	0.46		0.15	0.36	
Dust/Cockroach allergen			0.16	0.01	0.29	0.45	*	0.04	0.20	*
Maternal Atopy			0.27	0.01	0.28	0.02		0.10	0.01	*

*Significantly different at p<.05

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CHAPTER 3

IS TEEN CHILDBEARING GOOD FOR OFFSPRING HEALTH? A TEST OF TWO HYPOTHESES

Do children of teen mothers have worse health than their comparable peers, controlling for background factors? While current political and cultural discourse consistently positions teen motherhood as a health problem (Luker 1997), the scientific evidence is disputable.

Opponents of teen childbearing typically cite two reasons. First, a teen mother's physiologic immaturity heightens the risk of preterm or low birthweight (LBW) babies (Hediger, Scholl, Schall, and Krueger 1997). Second, a teen mother may lack the economic resources or psychological maturity critical to raise healthy children (Maynard 1997; Scholl, Hediger, and Belsky 1994).

Others propose a culturally adaptive model of fertility timing. Since disadvantaged mothers are susceptible to "weathering"—early health deterioration from an accumulation of insults to health— they may enjoy their prime health in their teens. For such women, teen childbearing may actually improve offspring health by synchronizing their births with maternal peak health (Geronimus 2004).

A closer examination of the literature suggests that the negative effects of teen pregnancy are overstated. Most studies inadequately control for confounding maternal background factors, such as socioeconomic status (SES). Teen mothers differ from the

general population in ways that could lead to poor offspring health outcomes for women of any age. Analyses employing techniques to account for unobserved background factors reveal attenuated effects, either in terms of the mother's subsequent economic trajectory or the child's health (Geronimus and Korenman 1993; Rich-Edwards 2002).

Yet, the evidence in favor of teen childbearing is equally scarce. A lack of prospective data has resulted in a paucity of health scholarship on older children of teens. Moreover, most longitudinal studies assess cognitive or behavioral functioning rather than physical health. This research gap is significant because health disparities not apparent during infancy may emerge over time. The effects of biological and social exposures during gestation, early childhood, and adolescence may accumulate and only express themselves later in life. This paper contributes to the literature by considering a range of theoretically and empirically relevant health outcomes to assess child health, including LBW, chronic conditions, asthma, and obesity. To my knowledge, this is the most comprehensive examination of health outcomes to date for offspring of teens. Given the multidimensional nature of health, casting a wider net allows a more fine-grained assessment of childhood health than permitted by single measures. Furthermore, access to longitudinal data allows me to examine children beyond infancy, ranging in ages 5 to 19. These age groups, of clear policy import, warrant further research.

Theoretical Background

Developmental/Social Causation Framework

The literature opposing teen pregnancy typically draws from two conceptual models. The first, the developmental perspective, posits that teen motherhood is harmful

because of the mother's physiologic immaturity (Scholl et al. 1994). The nutritional needs of a growing fetus must compete with the high nutrient demands of adolescence and pregnancy (Borja and Adair 2003). Furthermore, adolescents' unstable hormonal patterns could increase the risk of preterm or LBW births (Hedger et al. 1997), leading to higher rates of neonatal mortality, infant morbidity, and chronic disability (Barker 2004).

Biomedical evidence indicates, however, that adolescent female bodies are no less biologically capable of baring healthy children, except among the youngest teens (e.g. 13 and below) (Kline, Stein, and Susser 1989; Phipps and Sowers 2002). Most studies supportive of the developmental perspective predominately focus on the youngest teens, at the extreme end of the distribution (Hediger et al. 1997). As such, these studies are unrepresentative of the broader teen population.

The second argument against teen pregnancy draws from social causation theory (Elstad and Krokstad 2003). This framework moves away from the biological and highlights the social risk factors associated with teen pregnancy. Children of teens, for instance may be more vulnerable to abuse or neglect because of young parents' poor parenting skills. Alternatively, early childbearing may, by disrupting a young woman's schooling, propel her on a downward economic trajectory, increasing her children's exposure to pathogenic occupational, environmental, and social conditions associated with poverty (Ensminger, Juon, and Fothergill 2002). Impoverished mothers may engage in more risky health behavior since low socioeconomic status is associated with reduced prenatal care, poor diet, infrequent medical visits, and cigarette use (Wolfe 1997).

The assertion that teenage pregnancy causes poor educational and economic outcomes is subject to much dispute. Past evidence is limited by selection bias; findings

from studies that use various methods to account for unobserved background factors reveal little to no effect. Geronimus and Korenman (1993) studied sister pairs and demonstrated that teenage motherhood bore no causal effect on high school graduation rates or family income. Similarly, Hotz et al. (2005) compared mothers who were 18 and younger with teenagers who miscarried, under the assumption that miscarriages are random. By their late twenties, the differences in education level and welfare-related income were insignificant; moreover, teen mothers had steadier employment and earned higher incomes than their counterparts. Though employing miscarriages as an instrumental variable has been criticized because of potential underreporting or non-randomness, Hotz et al. (2005) address these concerns through sensitivity analyses. Even with the more conservative assumptions, teen mothers fare better economically. Finally, studies using multiple variables to control for poverty find that teen motherhood is unrelated to offspring abuse, or inferior cognitive development and achievement test scores (Levine, Emery, and Pollack 2007; Massat 1995).

Culturally Adaptive Framework

In response to the empirical weaknesses identified above, other researchers proposed a culturally adaptive model of fertility timing. This framework positions fertility-timing norms and behaviors as "critical mechanisms through which the basic cultural imperatives toward economic and reproductive success are pursued" (Geronimus 2004). For affluent families, delayed childbearing assures that young adults achieve economic self-sufficiency before incurring the additional financial constraints of parenthood. Among the urban poor, in contrast, *early* fertility-timing may be a culturally

adaptive means of maximizing offspring wellbeing. For highly disadvantaged African American women, an accumulation of health insults beginning in infancy leads to early health deterioration and excess mortality, a phenomenon known as "weathering." Thus, disadvantaged African American women may enjoy their peak health at substantially younger ages (Rauh, Andrews, and Garfinkel 2001).

Early childbearing may also lengthen the time that extended kin, their own health precarious, provide vital material and emotional support for the child. Thus, "children may fare best if their birth and preschool years coincide with. . . access to social and practical support provided by relatively healthy kin" (Geronimus and Thompson 2004, p. 159).

Some empirical evidence links early childbearing to improved infant health (Buescher and Mittal 2006; Shaw, Lawlor, and Najman 2006). For example, a matched comparison group study between teen mothers and their siblings revealed that, among singleton first births to Michigan residents aged 15-34 in 1989, African American infants whose mothers were 25 were twice as likely to be LBW as those with 16-year old mothers (Geronimus 1996). Among whites, maternal age was unrelated to LBW or very LBW.

Nevertheless, the research beyond infancy is scarce. While international studies find no deleterious effect, they yield little evidence that early fertility is protective. A prospective study of 14-year old children of 5,260 Australian women found that offspring of mothers aged 18 and younger versus 19 and older were no more, but also no less, likely to report poor health; suffer from asthma, frequent accidents or fractures; or have 2 or more admissions to hospital since birth (Shaw et al. 2006).

American studies, nearly all assessing cognitive and academic development, provide mixed support. Some find that teen motherhood is beneficial. Geronimus, Korenman, and Hillemeier (1994) examined standardized cognitive development and achievements tests among preschool and elementary school children of a national sample of sisters, using fixed effects models to account for unobserved heterogeneity. The offspring of teen mothers ages 18 and younger fared equally well or better to children of women 19 and older. Moore et al. (1991) likewise reported higher math and reading scores among black children of 18-year-old mothers versus those in their 20s. Other studies, using similar methods, find either no or slightly worse effects (Grogger and Bronars 1993; Levine et al. 2007; Turley 2003).

Aim of Study

Given that evidence on the relationship between teen childbearing and offspring health is inconclusive, this study hopes to further the debate by examining a range of health outcomes among a nationally representative, longitudinal cohort of children ranging in ages. Utilizing the aforementioned theoretical frameworks, I test the following hypotheses:

H1. Children of teen mothers will have worse health outcomes than their comparable peers.

H1a. Developmental framework: The negative effects will persist even when controlling for maternal, child, and family characteristics.

H1b. Social causation framework: The negative effects of teen motherhood will be mediated by current socioeconomic/health behavior characteristics such that including these factors will attenuate or eliminate the teen mother-child health association.

H2. Culturally adaptive framework: Children of teen mothers will experience better health outcomes than their comparable peers.

Methods

Data

Data for this study come from the Panel Study of Income Dynamics (PSID) and its Child Development Supplement (CDS). The PSID is a longitudinal study of U.S individuals and their families. Since 1968, the PSID has collected data on individuals' marriage and fertility history, income, employment, and family composition. In 1997, the PSID supplemented its core data collection with additional information on 0-12 year-old children and their parents. The goal of the supplement was to improve understanding of the socio-demographic, economic, and psychological aspects of childhood from a longitudinal perspective. The CDS-I completed interviews with 2,394 families (88%), providing information on 3,563 children. In 2002-2003, 2,019 CDS-I families still active in the PSID sample as of 2001 were re-interviewed. The CDS-II totaled 2,907 children and adolescents aged 5-19 (91%).

The sample in this analysis is limited to 982 black and 1199 white, non-adopted, non-institutionalized children born in the U.S. between 1970 and 1997 with available data on their and their mother's birth date (75%). Of the 726 omitted, 355 (49%) were

excluded because of their race; 203 (28%) were adopted or lacked information about their biological mother; 111 (15%) were born abroad, had mothers who lived abroad during pregnancy, or were missing birth information (i.e. birthdates); 16 (2%) were born to either extremely young (ages 14 or below) or old mothers (41 and older) and were dropped because of the infrequency and selectivity of these births, and a final 41 children (6%) did not reside with their mother during the survey.

These data offer several advantages. When linked to the PSID—an intergenerational study that tracks family members in biennial interviews—the CDS offers more family data than any nationally-representative longitudinal survey of children in America (Mainier 2006). Such detail proves useful in identifying confounding background characteristics, such as childhood economic status. Moreover, the CDS module possesses a rich source of information on a range of acute and chronic child health outcomes, notably missing from many datasets. Finally, the sample's age variation, coupled with the oversampling of blacks, allows for a broader analysis of child health comparisons between and within races.

One unfortunate data limitation is the small number of teen/non-teen sibling pairs. Though matched-sisters analysis is an effective way to reduce across-family heterogeneity, an insufficient sample size prevents this approach.[6] Since standard controls may overestimate the negative effects of teen pregnancy, results in favor of the developmental/social causation perspective could be upwardly biased and must be interpreted cautiously. On the other hand, the use of conventional regression models rather than fixed effect analyses is less problematic for testing the culturally adaptive

[6] Due to the scarcity of teen/non-teen sister pairs and insufficient variation in health outcomes between first cousins, the total number of qualifying sibling pairs range between 12 and 20.

perspective, since this creates *more conservative* criteria. Were I, in spite of the upward bias associated with standard controls, to find positive associations between teen pregnancy and offspring well-being, this would offer convincing evidence for the culturally adaptive perspective.

Health Outcomes

Recent research highlights the complexity in measuring child health outcomes and the limitations in any single measure (Case, Lubotsky, and Paxson 2002). I selected the following health measures, based on their association with teen childbearing and their standard use in epidemiological research. All health outcomes are reported by the primary care giver, and, except for LBW, originates in the CDS-II.

Low Birthweight (LBW). LBW is a leading cause of morbidity and mortality among infants born to highly disadvantaged mothers (MacDorman and Atkinson 1999). Closely linked to chronic disabilities, LBW is also traditionally attributed to early childbearing (Borja and Adair 2003). Derived from the 1997 CDS-I, LBW is a dichotomous measure coded as 1 if the primary caregiver reported the child as less than 5.5 pounds at birth.

Chronic Illness. Following past health studies (Stein, Siegel, and Bauman 2006; Wolfe and Perozek 1997), chronic illness is defined as having any of the following physician-diagnosed conditions: seeing or hearing difficulties, autism, hypertension, high cholesterol, congenital heart disease, seizures, orthopedic impairment, sickle cell anemia, birth defect, asthma, digestive problems, mental retardation, diabetes, or developmental delays.

Given the overrepresentation of obesity and asthma among disadvantaged children (Luder, Melnik, and DiMaio 1998; Martinez, Wright, Holberg, Morgan, and Taussig 1992), these outcomes are examined separately.

Asthma is a dichotomous variable coded as 1 if the child ever received an asthma diagnosis; or if, during the past year, the child went to a doctor's office, health care clinic or emergency room for asthma or wheezing; experienced an asthma attack or wheezed while exercising or running; or missed school because of asthma.

Obesity[7]. Obesity is calculated from the child's body mass index (BMI), a score derived from CDS-II interviewer-measured weight and height of the child.[8] Respondents at or above the 95th percentile for the sex-specific BMI-for-age are considered obese (Hedley et al. 2004).

Predictors

Maternal Age at Birth. Maternal age at birth is derived by first subtracting the child's date of birth from the mother's, and then rounding the digit to the lowest whole number (e.g., 15.1 to 15.11 would be 15). While most research on adolescent pregnancy operationalizes teen motherhood as a dichotomous variable, binary measures may obscure health differences between younger and older teens. Thus, maternal age is divided into the following six categories: 15 to 17, 18 to 19, 20 to 24 (the reference), 25 to 29, 30 to 34, and 35 to 40.

[7] Unlike all other health outcomes, analyses on obesity are restricted to 1946 children because of missing weight and height information. Additional analyses demonstrated that the key results for all other health outcomes are robust to this more restricted sample.

[8] The 2000 Centers for Disease Control and Prevention Growth Charts for the United States provide BMI-for-age percentiles. Because of their infrequency, underweight subjects (having a BMI-for-age less than the 5th percentile were placed in the normal weight.

Note that this operationalization differs from Levine and colleagues (2007) in that they focus on maternal age at *first* birth rather than age at a *given* child's birth. Their definition avoids underestimating negative consequences of teen childbearing that could operate through the mother's economic position. For example, were teen childbearing to force the mother into poverty, then the social, physical, and environmental health risks associated with such a trajectory could extend to all children (Turley 2003). Subsequent children, born to mothers who were teens only at their first birth, may also suffer.

However, this definition could be problematic if the initial disadvantage of early childbearing dissipates and teen mothers eventually catch up to their older counterparts (Furstenberg, Brooks-Gunn, and Chase Lansdale 1989), or if the primary causes of poor pregnancy outcomes among teen mothers is biological rather than social. In both cases, including second or third-born children of mothers who were teens only at their first births in the "teen category" may potentially *understate* adverse teen motherhood effects. Thus, I define as teen mothers only those women who were teens at the time of the given child's birth. Subsequent sensitivity analyses rerun estimates on first-born children only. In such cases, maternal age at first birth is indistinguishable from maternal age at given birth, circumventing the thorny definitional issues.

Child's Race. Race is a dichotomous variable reported by the primary caregiver, with non-Hispanic blacks coded as 1 (non-Hispanic whites as reference).

Potential Confounders

Maternal Socioeconomic Status. Given that poverty, which selects women into early childbearing, may also select their children into worse health, I control for maternal

SES prior to pregnancy. Grandmother's educational attainment, a proxy for the mother's childhood socioeconomic position, is measured in four categories: less than high school, high school, some college or vocational training, and college and above (reference group). Pre-pregnancy poverty level, which captures the mother's economic status prior to childbearing, is the average of the mother's income-to-needs ratio for the three years *prior* to pregnancy.[9] Finally, government assistance is a dummy variable to identify acute financial strain during pregnancy. Mothers receiving any form of means-tested government assistance while pregnant (food stamps, WIC, AFDC, Medicaid) are coded as 1.

Additional controls include the child's sex, birth order (1=first born, 2=second born, 3=third or more),[10] age in years, current number of adults in the family unit, and maternal marital status at child's birth (1=married), all measures which are correlated with fertility timing and child health outcomes (Holt, Danoff, Mueller, and Swanson 1997). Given the evidence relating kinship networks to child wellbeing (Deleire and Kalil 2002), I also include measures of family social support/structure. Multi-generational residence consists of two dummy variables (representing CDS-I and II), coded as 1 if any of the child's grandparents reside with the child, or if the child's grandmother is the reported head of household or child's primary caregiver during the respective CDS wave. Family social support is a two-item index from the CDS-II indicating maternal satisfaction with instrumental and emotional support. The scores

[9] The poverty level is constructed by creating income-to-needs ratios for each year. The family's yearly income is divided by the US census annual needs standard figure, and the three ratios are averaged.

[10] Eighteen cases with missing data on the child's birth order are included in the reference category (first-born). Preliminary analyses assured that treating "missing" as a separate category did not change point estimates.

range from (1) completely dissatisfied to (7) completely satisfied. Finally, because family history is a risk factor for asthma, all asthma-related analyses adjust for family asthma (coded as 1 if the head of household and/or the spouse suffers from asthma).[11]

Potential Mediators

Current SES. To capture health effects arising from the financial impact of early childbearing, I include two measures of current SES. Household income is the average of 1998 and 2002 household income, categorized into dummy variables: (1) less than $5000 (2) from $5000 to $14,999 (3) $15,000 to $39,999, (4) $40,000 and greater (reference group), and (5) missing.[12] Economic strain is a count variable of 13 experiences in the past year arising economic difficulties. Examples include cashing in life insurance, filing for bankruptcy, and having wages garnished.

Healthcare/Behaviors. Inadequate access to healthcare and risky behaviors may also mediate the teen childbearing-health relationship. Because children with teen mothers may receive less preventative medical care (Weinick, Zuvekas, and Cohen 2000) and are breastfed less (Radius and Jaffe 1988), I include dichotomous measures for insurance status (1=insured), immunization status, and breastfed as infant.[13]

[11] Because the CDS does not collect information about parental asthma, these data are extracted from the PSID family file for the years 1999 and 2001. Unfortunately, the files only provide information about the head of household and his spouse, who are in most cases the child's parents. For the purpose of these analyses, I exclude cases in which neither the head nor the spouse is biologically related to the child.

[12] The PSID collected no data on family income in 2001.

[13] The primary caregiver was asked in both CDS waves whether the child was breastfed as an infant. To minimize potential recall bias, I use responses from CDS-I, except when data are missing.

Analytic Strategy

Given the differences between blacks and whites in terms of fertility timing and health status, I stratify all analyses by race[14] in three-model estimation. The first specification consists of maternal age-at-birth dummies, the child's age and gender.[15] A second model adds potentially confounding maternal and family background factors: namely, grandmother's educational attainment, maternal marital status at childbirth, and maternal financial status prior and during to pregnancy. The final model incorporates the final, potentially mediating/moderating economic, psychosocial, and behavioral characteristics.

Because of their dichotomous outcomes, logistic regressions estimate all models. Sample weights correct for the differential probability of selection and attrition across waves. All statistical estimates are computed using STATA software package.

Results

Descriptive Statistics

Table 3.1 presents weighted summary statistics, stratified by race and maternal age. African Americans make up almost half the sample, with 982 respondents. About 15% are born to teen mothers, the majority to 18 or 19 year-old mothers. The average

[14] Full-sample analyses (not shown) demonstrate significant race-age interactions, supporting the decision to stratify by race.

[15] Unlike other health outcomes, LBW models include only pre-childbirth covariates. Child age is omitted since all newborns are, by definition, the same age. However, this measure may be an effective proxy for cohort or period effects. The differential availability of medical technology or prenatal care 5 versus 15 years ago, for instance, may impact the risk of LBW. Additional models including child age yielded no substantive differences, and the term was omitted from final analyses. Initial model specifications also included an interaction term between child's age and maternal teen status to assess whether the health effects of teen childbearing varied by offspring cohort. Once again, the measure was subsequently dropped due to non-significance.

black child is a 13-year old male born out-of-wedlock (column 1) and is a second or third birth. He is likely insured and immunized, though tends to not be breastfed. The average black mother was 25 at childbirth, comes from a fairly disadvantaged family, but experiences a relatively high level of social support. Over 75% received some form of means-tested financial aid while pregnant; most currently earn $40,000 or less and report at least one economic strain.

In contrast to blacks, only 6% of the 1199 white children are born to teen mothers, with an average childbearing age of 27.7 (column 4). Seventy-three percent of the teen mothers are 18 or older at birth. Unlike her black counterpart, the average white child is 12, female, born to married parents, lives in a nuclear family with two adults, and is breastfed. The average white child's mother comes from a significantly more privileged economic background than her black counterpart, with a pre-birth income 350% of the poverty level. Only 22% of white mothers received government assistance while pregnant and nearly 80% currently earn over $40,000.

Consistent with the current epidemiological literature, black health lags behind whites. Black children are almost three times more likely than white to be LBW, report significantly higher levels of obesity, and slightly elevated asthma rates (31 vs. 26%, significant at $p<.07$).

Columns 2 through 5 present statistics further stratified by maternal teen status. With a few exceptions (i.e., economic disadvantage, intergeneration household), black offspring of teens versus non-teens are equivalent on most socio-demographic and behavioral characteristics. More pronounced differences emerge for whites, with

offspring of teens trailing non-teens on nearly all socio-demographic, economic, and behavioral factors.

In light of these differences, it is notable that, regardless of race, offspring of teens and non-teens report similar prevalence rates. While black children of teens have slightly better health outcomes, and white offspring of teens, slightly worse, than their non-teen counterparts, the differences are not statistically significant.

[Table 3.1 about here]

Regression analyses

Tables 3.2 through 3.4 extend the descriptive results to a regression framework. Table 3.2 presents race-stratified odds ratios for LBW and asthma. Consistent with the descriptive statistics, offspring of teens versus non-teens have statistically equivalent lifetime prevalence rates. The risk of LBW for blacks increase *only* after age 30, with 30-34 year-old mothers almost 3 times more likely and 35-40 year-old mothers over 8 times more likely to have a LBW baby, compared to 20-24 year-old black women (columns 1-3). The relationship between maternal age and asthma remains uncorrelated (columns 7-9). Among older white women (columns 4-6), the odds of LBW declines by about 70% for 30-34 year-old women, compared to the 20-24 category, though the results are borderline significant. For asthma, the inclusion of socioeconomic status outcomes and family asthma (column 12) reveal an increased risk for children of 25-29 year-old mothers and 35-40 year-old mothers, respectively, compared to women in their early twenties, though the effects are, once again, significant at the $p<.10$ level.

Of the significant covariates in the regression model, none mediate the maternal age-health relationship. For both blacks and whites, birth order is negatively correlated with LBW and asthma, and family history positively predicts asthma. Current SES correlates with asthma in the expected direction for whites, but not blacks, with those earning under $15,000 and/or experiencing economic strain at a higher risk. For blacks, economic strain, unexpectedly, negatively correlates with asthma while current income is unrelated. In all cases, the effects are independent of maternal age.

[Table 3.2 about here]

Table 3.3 presents race-stratified outcomes for chronic illness and obesity. Irrespective of race, maternal age is uncorrelated with chronic illness. Obesity exhibits a somewhat more complex pattern. Black offspring of the oldest, and, at a statistically marginal level, the youngest, women exhibit a lower risk of obesity than their 20-24 year-old counterparts. White children of 15-17 year-old and 25-29 year-old women enjoy a similar advantage, though the former are significant only at the $p<.10$ level. The lower likelihood of obesity for white children of 30-34 year-olds, evident in column 10, attenuates to non-significance in the full model.

As with LBW and chronic illness, no covariate mediates the maternal age-child health relationship. However, background and current SES measures predict offspring health. Family income is inversely related to chronic illness for blacks and whites; likewise, economic disadvantage is positively correlated with black obesity and white chronic illness. The risk of obesity is negatively correlated with being female and maternal economic background for whites, and grandmother's educational attainment and

immunization status for blacks. Finally, chronic illness is positively correlated with being married and birth order for blacks, and negatively correlated with being female or intergenerational co-residence for whites.

[Table 3.3 about here]

Sensitivity Analyses

Table 3.4 presents results from first-born only analyses. No statistical difference appears for offspring of teens versus non-teens of either race.

[Table 3.4 about here]

Additional models with an alternative cut-off for "adolescent childbearing" at age 18 instead of 19 are also estimated. Point estimates from these results (not shown) are consistent with those presented here, suggesting that the 19-year-old mothers do not mask potential adverse health outcomes associated with 18-year-old mothers. I also run multinomial logit models using a three-category measure of weight (underweight, normal, and obese) because the current obesity measure placed underweight children in the "normal" category. Given that poor nutritional status may also manifest as underweight, this definition may introduce a downward bias by underestimating the differences between the two groups. The alternative specification (not shown) reveals no demonstrable increase in obesity or underweight risk for black or white offspring of teens versus non-teens, nor do separate logistic regression models (also not shown) excluding underweight children from the sample change the results. It therefore seems unlikely that

collapsing underweight into the reference category had any substantive impact on the findings.

Discussion

Does teenage pregnancy result in negative health outcomes for the offspring? Though mainstream social and political discourse suggests that the answer is "yes," the evidence to date is disputable. Constrained by methodological limitations and a lack of rich data sets, prior research has examined only a limited number of early childhood outcomes, ignoring health consequences among older children.

This paper revisits the question, equipped with several advantages over the literature to date: longitudinal data from a nationally representative sample of infant, adolescent, and teenage offspring, a range of health outcomes, and extensive background controls. Specifically, I investigate two contrasting hypotheses: "teenage childbirth causes poor offspring health" and "teenage childbirth improves offspring health."

Teenage Pregnancy Does No Harm

I find a strong lack of support for the first hypothesis. Prevalence rates of LBW, obesity, asthma, and chronic illness are no different for children of teen mothers and the children of mothers in their early twenties. These results hold for both black and white children. Furthermore, the null effects are evident in the baseline model, countering the social causation theory that the negative effects of teen parenthood are mediated through economic and behavioral factors.

There results are particularly robust since my models are biased towards *overestimating* the adverse impact of teen childbearing. Although standard controls adjust for past maternal SES, it is difficult to disentangle the negative effects of the mother's background and prior SES from that of the timing of the actual pregnancy (Geronimus and Korenman 1993). Since these conservative models find no negative impact for teenage childbirth and sensitivity analyses produce no substantive changes in outcome, we can be confident in the strength of the results.

Teenage Pregnancy May Do Good

I find weak support for the second hypothesis that teen pregnancy improves offspring health. For three of the four outcomes, offspring of teens exhibits no advantage over those of women in their twenties. Although the remaining outcome (obesity) has a lower prevalence rate among children of young teens of both races versus those of women in their twenties, these results should be viewed cautiously. The coefficients are only marginally significant (at $p<.10$ level) and represent a small sample size. Moreover, the overall relationship between maternal age and obesity is perplexing. Among blacks, an even greater reduction in obesity is found for offspring in the 35-40 categories; a similar reduction occurs for white women as they delay childbearing from the early to late twenties.

As mentioned previously, current regression models may overestimate the adverse effects of teen childbearing, obscuring substantive health advantages associated with early pregnancy. Future research exploiting natural experiments or employing more sophisticated models may discover undetected benefits. However, the current study

yields insufficient evidence to support the culturally adaptive model of childbearing. These results are consistent with Moore et al. (1997), who found no association between teen pregnancy and birthweight, and Shaw et al. (2006), who studied the offspring of Australian teens and found no negative impact upon their self-reported health or hospitalization rates.

Limitations

In addition to the lack of fixed effect estimates, other limitations of the study should be mentioned. Since highly disadvantaged children—those most likely to have teen mothers—underutilize medical services (Weinick et al. 2000), disease rates may be biased downwards by undetected cases, resulting in underestimation of the negative impact of teenage childbirth. However, Medicaid and state insurance programs such as S-CHIP have successfully expanded access to health care and diminished economic disparities in health care utilization. Furthermore, all models control for insurance status, minimizing the likelihood of underdiagnosis.

Additionally, my analyses only examined children who participated in the CDS-I and CDS-II. The sickest children may have dropped out of the sample between CDS-I and CDS-II. However, inspection of the data shows that only eight children from CDS-I left due to death or illness. Given the CDS-II's high response rate, sample attrition is an unlikely source of systematic bias. It is also improbable that the sickest children died before the CDS-I, excluding them from initial eligibility; additional investigation showed this to be a rare event with negligible impact on the estimations.

Policy and Research Implications

The two predominant models of teenage pregnancy are in opposition: one posits that early pregnancy harms the offspring, whereas the other suggests that teenage pregnancy is a culturally adaptive practice. This paper challenges both views and finds that teenage pregnancy has little (if any) effect on child health.

Given the singular focus on teen childbearing within our cultural discourse and its designation as a social/health problem, it is striking that nearly all age-related variation in health occurred between the non-teen groups. These findings generate several questions of sociological interest: what are the ideological, social, and political forces driving the current pathologization of teenage motherhood? What is the role of the academy in constructing the dominant views of teen childbearing as either adaptive or deviant, and how does scientific knowledge differ from the "lived experience" of the teen mothers? What are the psychosocial costs of stigmatizing teen childbirth?

From a policy perspective, this study suggests that efforts to improve children's health through anti-teen pregnancy initiatives could be ineffective at best and harmful at worst, since such efforts could stigmatize teen mothers while distracting attention from empirically supported policy interventions. These policies may also overlook equally vulnerable populations such as poor children of non-teens. Rather than expending scarce resources toward preventing teenage childbirth, policy makers would do well to redirect efforts toward confronting the fundamental causes of poor health.

Table 3.1 Weighted Means for CDS-II Data

	Blacks (n=982)				Whites (n=1199)			
	All	Teens	Non-Teens		All	Teens	Non-Teens	
	1	2	3		4	5	6	
Health Outcomes								
Low Birthweight	0.12	0.09	0.12		0.05	0.10	0.04	
Asthma	0.31	0.35	0.31		0.26	0.29	0.26	
Obesity[1]	0.25	0.20	0.25		0.17	0.19	0.17	
Chronic Condition	0.28	0.25	0.29		0.28	0.31	0.28	
Maternal Background Factors								
Average Age at Birth	25.61	17.47	27.18	*	27.76	18.04	28.37	*
%		0.16	0.84			0.06	0.94	
Maternal Age (Prop.)		.						
15 to 17	0.06	0.40	-	-	0.02	0.27	-	-
18 to 19	0.10	0.60	-	-	0.04	0.73	-	-
20 to 24	0.30	-	0.36	-	0.20	-	0.22	-
25 to 29	0.25	-	0.30	-	0.35	-	0.37	-
30 to 34	0.22	-	0.27	-	0.30	-	0.32	-
35 to 40	0.07	-	0.08	-	0.08	-	0.09	-
Pre-Pregnancy Pov. Level	190%	170%	193%		351%	222%	359%	*
Grandmother's Educ (Prop.)								
Less than high school	0.49	0.44	0.50		0.21	0.32	0.20	*
High School	0.34	0.34	0.34		0.44	0.43	0.44	
Some College	0.13	0.21	0.11		0.21	0.22	0.21	
College or above	0.05	0.02	0.05		0.15	0.04	0.15	*
Gov. Assistance and Pregnant	0.74	0.90	0.71	*	0.22	0.73	0.18	*
Married at Birth	0.40	0.07	0.47	*	0.92	0.47	0.94	*
Child Characteristics								
Age	12.72	12.03	12.86		12.23	11.75	12.26	
Female	0.42	0.38	0.43		0.52	0.56	0.52	
Birth Order	1.94	1.18	2.09	*	1.75	1.13	1.79	*
Breastfed	0.24	0.19	0.25		0.65	0.44	0.66	*
Uninsured	0.09	0.08	0.10		0.06	0.15	0.05	*
Vaccinated	0.98	0.98	0.98		0.98	0.98	0.98	
Current Family Characteristics								
Adults in Family	1.74	1.47	1.79	*	2.05	1.93	2.06	
Family Income (Prop.)								
Less than $15,000	0.19	0.22	0.18		0.02	0.04	0.02	
$15,000 to $39,999	0.46	0.52	0.45		0.16	0.32	0.16	*
$40,000 and up	0.31	0.22	0.33		0.79	0.58	0.80	*
Economic Strain (#)	1.07	1.03	1.08		0.72	1.25	0.69	*
Social Support	5.42	5.14	5.48		5.15	5.18	5.15	

Note: All variables are coded in the direction of the label.

[1]*N* for obesity is 1946: 1049 whites & 897 blacks

*Teen/Non-Teen differences are significant at p<.05 or below.

Table 3.1 Weighted Means for CDS-II Data (continued)

	Blacks (n=982)				Whites (n=1199)			
	All	Teens	Non-Teens		All	Teens	Non-Teens	
	1	2	3		4	5	6	
Intergenerational Residence CDS-I	0.08	0.25	0.04	*	0.02	0.17	0.01	*
Intergenerational Residence CDS-II	0.06	0.19	0.04	*	0.01	0.07	0.01	
Family History of Asthma	0.12	0.12	0.12		0.15	0.20	0.14	

Note: All variables are coded in the direction of the label.

[1] *N* for obesity is 1946: 1049 whites & 897 blacks

*Teen/Non-Teen differences are significant at p<.05 or below.

Table 3.2 Odds Ratios of Low Birthweight and Asthma by Race, Maternal Age, and Covariates

	LBW						Asthma					
	Whites (n=1199)			Blacks (n=982)			Whites (n=1199)			Blacks (n=982)		
Maternal Age at Birth												
15-17	0.57	0.53	0.44	1.07	1.12	0.87	1.20	1.06	0.95	0.79	0.85	0.66
18-19	1.76	1.76	1.52	1.06	1.02	0.86	1.10	1.02	1.00	1.69	1.75	1.58
25-29	0.69	0.73	0.79	1.1	1.15	1.44	1.14	1.19	1.45+	0.89	0.82	0.78
30-34	0.31*	0.33*	0.39+	1.85	1.99	2.87*	0.80	0.85	1.19	1.21	1.15	1.04
35-40	0.19	0.21	0.28	4.82**	4.69**	8.27***	0.94	1.02	1.79+	1.45	1.35	1.42
Female	0.95	0.94	0.95	0.95	0.97	1.01	0.82	0.82	0.81	1.33	1.37	1.31
Child's Age							1.00	1.00	1.01	0.96	0.95+	0.96
Mother's Pre-Poverty Level		0.99	0.95		1.03	0.94		1.00	0.98		0.91	0.92
Grandmother's Education												
Less than High School		1.33	1.46		1.20	0.94		1.29	1.35		0.61	0.61
High School		1.10	1.13		1.35	1.08		1.22	1.21		0.53	0.59
Some College		1.26	1.29		0.83	0.68		1.11	1.19		0.36	0.38
Gov. Assistance & Pregnant		1.09	1.27		1.28	1.41		1.22	1.01		0.72	0.69
Mother Married at Birth			1.50			1.04			1.14			1.33
Child's Birth Order												
2nd			0.32**			0.86			0.77			0.50**
3rd or more			0.66			0.28**			0.53**			0.77
Breastfed									0.79			0.85
Uninsured									0.95			0.43*
Immunized									1.27			0.33
Current Family Income												
Less than $15k									3.30+			1.60
$15 to $39k									0.83			1.57
Current Economic Strain									1.35***			0.83+

Note: All variables are coded in the direction of the label name. Reference category in parentheses.
+p<0.10, *p<0.05, **p<0.01, ***p<0.001 (two-tailed test)

Table 3.2 Odds Ratios of Low Birthweight and Asthma by Race, Maternal Age, and Covariates (continued)

	LBW		Asthma	
	Whites (n=1199)	Blacks (n=982)	Whites (n=1199)	Blacks (n=982)
Adults in Family			0.90	1.00
Social Support			1.09	1.00
Intergenerational Residence				
CDS-I			0.54	0.75
CDS-II			2.16	1.43
Family History of Asthma			1.84**	3.14***

Note: All variables are coded in the direction of the label name. Reference category in parentheses.
+p<0.10, *p<0.05, **p<0.01, ***p<0.001 (two-tailed test)

Table 3.3 Odds Ratios of Obesity and Chronic Illness by Race, Maternal Age, and Covariates

	Obesity						Chronic Illness					
	Whites (n=1049)			Blacks (n=897)			Whites (n=1199)			Blacks (n=982)		
Maternal Age at Birth												
15-17	0.56	0.50	0.19+	0.39*	0.38+	0.44+	1.01	0.84	1.01	0.61	0.64	0.61
18-19	0.88	0.88	0.76	1.03	1.00	1.17	1.06	0.94	1.03	0.85	0.84	0.79
25-29	0.51**	0.57*	0.58*	0.91	0.81	0.73	0.98	1.12	1.19	0.80	0.70	0.73
30-34	0.56*	0.65	0.71	1.66	1.68	1.53	0.61*	0.71	0.79	0.99	0.82	0.92
35-40	0.59	0.70	0.82	0.31*	0.34*	0.32*	1.11	1.32	1.60	0.95	0.93	0.90
Female	0.66*	0.68*	0.66*	0.96	1.04	1.10	0.76+	0.76+	0.76+	0.70	0.80	0.80
Child's Age	1.03	1.01	1.02	1.01	1.02	1.00	1.03+	1.04+	1.04+	0.97	0.97	0.97
Mother's Pre-Poverty Level		0.89*	0.88*		1.04	1.04		0.98	0.97		1.13	1.06
Grandmother's Education					0.37	0.31						
Less than High School		0.98	0.94		0.35	0.27+		1.19	1.15		0.39	0.65
High School		1.23	1.19		0.69	0.44		1.16	1.18		0.45	0.73
Some College		1.13	1.09		1.14	1.25		1.42	1.45		0.55	0.87
Gov. Assistance & Pregnant		1.02	0.85			1.14		1.45+	1.10		0.65	0.74
Mother Married at Birth			0.63						0.85			1.81*
Child's Birth Order												
2nd			1.07			1.03			0.96			0.56*
3rd & up			1.08			1.17			0.82			0.47*
Child Breastfed			0.77			0.58+			0.78			1.29
Child Currently Uninsured			1.05			1.23			1.56			0.79
Child Immunized			1.35			0.64			0.72			0.83
Current Family Income												
Less than $15k			0.99			0.56			4.59*			1.30
$15 to $39k			0.78			0.58			0.84			1.99*
Current Economic Strain			1.06			1.26*			1.21*			1.05
Adults in Family Unit			0.82			1.03			1.06			1.14
Mother's Social Support			0.96			1.05			0.95			1.03

Note: All variables are coded in the direction of the label name. Reference category indicated in parentheses.
+p<0.10, *p<0.05, **p<0.01, ***p<0.001 (two-tailed test)

Table 3.3 Odds Ratios of Obesity and Chronic Illness by Race, Maternal Age, and Covariates (continued)

	Obesity		Chronic	
	Whites (n=1049)	Blacks (n=897)	Whites (n=1199)	Blacks (n=982)
Intergenerational Residence				
CDS-I	2.71+	0.78	0.08**	0.71
CDS-II	2.94	1.01	1.27	1.00

Note: All variables are coded in the direction of the label name. Reference category indicated in parentheses.
+p<0.10, *p<0.05, **p<0.01, ***p<0.001 (two-tailed test)

Table 3.4 Odds Ratios of Health Outcomes by Race, Maternal Age, and Covariates for First-Born

	LBW[1]				Asthma			
	Whites		Blacks		Whites		Blacks	
Maternal Age at Birth								
15-17	0.43	2.68	0.90	0.69	1.45	1.45	0.51	0.56
18-19	1.19	2.46	0.91	0.71	1.26	1.31	1.54	1.45
25-29	0.59	1.74	0.50	0.52	1.42	1.44	1.08	1.20
30-34	0.39	0.94	4.19*	4.95**	1.23	1.36	2.86*	2.16
35-40			7.88*	10.8**	1.93	0.73	0.26	0.24
Female	0.53	0.53+	0.94	1.20		0.55+	0.70	0.60+
Child's Age						1.00	0.95	0.95
Mother's Pre-Poverty Level		0.98		0.92		1.16		0.95
Grandmother's Education								
Less than High School		0.56		0.54		1.19		1.14
High School		1.08		0.47		0.87		2.56
Some College		1.30		0.43		2.35		1.75
Gov. Assist./Pregnant		1.38		1.64		1.00		0.61
Married at Birth		0.94		0.53		1.00		1.24
Child Breastfed						0.69		0.66
Child Uninsured						1.85		1.00
Child Immunized						0.94		0.76
Family Income								
Less than $15k					0.75	0.69		0.41
$15 to $39k					1.00	0.88		1.00
Current Economic Strain						1.43**		0.83
Adults in Family						0.81		0.55+
Social Support						1.00		0.93
Intergenerational Residence								
CDS-I						0.35		0.62
CDS-II						2.91		3.62*
Family Asthma						1.61+		2.90**
Sample Size	546	546	414	414	546	546	414	414

Table 3.4 Odds Ratios of Health Outcomes by Race, Maternal Age, and Covariates for First-Born (continued)

	Obesity				Chronic Illness			
	Whites		Blacks		Whites		Blacks	
Maternal Age at Birth								
15-17	0.52	0.21	0.56	0.5	0.73	1.58	0.37*	0.58
18-19	1.02	0.7	1.47	1.32	1.66	1.67	0.46	0.56
25-29	0.42*	0.55	1.34	1.93	1.43	1.47	0.43+	0.42+
30-34	0.50+	0.76	1.12	2.09	0.85	0.87	1.22	0.84
35-40	0.27	0.92	1.18	1.11	2.15	0.88	0.3	0.3
Female		1.22	1.42	1.44		0.95	0.78	0.79
Child's Age		1.09*	1.01	1.04		1	0.97	0.95
Mother's Pre-Poverty Level		1.68		0.99		0.59		1.27+
Grandmother's Educ.								
Less than High School		2.46+		0.52		0.75		0.79
High School		2.92+		0.45		0.78		1.19
Some College		0.32		0.5		2.26		0.98
Gov. Assist./Pregnant		0.91		1.37		0.97		0.81
Married at Birth		0.97		0.51		0.84		1.87+
Child Breastfed		0.74		1.02		0.59*		1.51
Child Uninsured		0.68		0.77		1.38		1.7
Child Immunized		5.96		0.99		0.5		0.65
Family Income								
Less than $15k	0.92	0.65		0.33	0.84	0.25		0.84
$15 to $39k	1.07+	1.15		0.36+	1.01	0.41*		1.48
Current Economic Strain		1.17		1.30+		1.26*		1.03
Adults in Family		0.75		0.62		0.59		0.91
Social Support		0.81*		0.97		0.94		1
Intergenerational Residence								
CDS-I		4.75+		1.26		0.068*		0.52
CDS-II		0.69		0.83		5.32+		1.67
Family Asthma								
Sample Size	489	489	387	387	546	546	414	414

[1]In the 35 and above age group, no white children were born LBW. Since the cell contains no variation, this category was combined with 30-34 for whites only.

+p<0.10, *p<0.05, **p<0.01, ***p<0.001 (two-tailed test)

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CHAPTER 4

DOES PLACE MATTER? NEIGHBORHOOD CONTEXT AND ASTHMA

The initial aim of this study was to estimate racial/ethnic disparities in asthma and to analyze how these disparities related to the areas in which people live. Unfortunately, because of the relatively low number of asthma cases within each neighborhood cluster, the data lacked sufficient power to decompose racial/ethnic disparities in asthma prevalence into within- and between-area components. For instance, of the 343 neighborhood clusters (NCs) sampled in the CCAHS dataset, none had at least two black residents and two white residents with asthma; only three NCs had one or more blacks and one or more whites with asthma. As such, the study was redirected from a focus on racial disparities toward a more general exploration of the relationship between neighborhood context and asthma.

The revised research aim conceptually relates to the initial aim, since asthma disproportionately burdens ethnic minorities and the poor who live in urban areas (Akinbami 2006). Understanding why neighborhoods may contribute to asthma prevalence could prove informative in understanding the source of racial disparities in asthma, if, say, minorities are more likely to live in asthma-inducing neighborhoods. Thus, while the redirected research question does not directly address the issue of racial disparities, it may serve to focus future research in exploring whether and through what mechanisms racial disparities in asthma potentially relate to neighborhood conditions.

* * * * *

Introduction

Asthma prevalence and morbidity are currently on the rise in the United States, with the trends disproportionately affecting individuals living in poor, urban areas (Gold and Wright 2005). The increases appear steepest in large inner cities, such as Chicago, New York, and Phoenix (Weiss and Wagener 1990).

The causes of the excess burden in these areas are not fully understood. Traditionally, the literature has concentrated on individual and family-level factors, such as indoor allergens, genetics, inadequate access to medical care, and associated poverty. However, recent research has begun to look to broader environmental exposures as a way to understand asthma (Aligne, Auinger, Byrd, and Weitzman 2000; Gold and Wright 2005; Wright and Fisher 2003). Individual-level health risks and behaviors appear to be spatially and socially distributed across neighborhoods (Fitzpatrick, LaGory, and La Gory 2000; Gee and Payne-Sturges 2004), with asthma-inducing pathogenic risk factors, such as indoor allergens or particulate matter concentrated in poor, segregated neighborhoods (Aligne, Auinger, Byrd, and Weitzman 2000; Byrd and Joad 2006; Federico and Liu 2003; Pearlman, Zierler, Meersman, Kim, Viner-Brown, and Caron 2006; Wright 2006). Researchers, utilizing an ecologic perspective on health, have sought to understand the role of social and geographic contexts specific to these neighborhoods in patterning such individual behaviors and risks (Subramanian, Belli, and Kawachi 2002; Williams and Jackson 2005; Wright and Subramanian 2007).

Recent experimental (Katz, Kling, and Liebman 2001) and observational studies (Cagney and Browning 2004; Juhn, Sauver, Katusic, Vargas, Weaver, and Yunginger

2005; Pearlman et al. 2006) support the theory that living in disadvantaged neighborhoods may be related to asthma prevalence and its associated morbidity, independent of individual factors. However, because asthma is a developmental disease primarily arising during childhood, these studies predominately focus on childhood asthma. Adult asthma—and its relationship to neighborhood context—remains an important but understudied area of research.

The current study contributes to the existing literature by examining differences in currently active asthma at the neighborhood level among adults living in Chicago. Through multilevel techniques, the paper assesses the extent to which neighborhood sociodemographic characteristics contribute to asthma prevalence, net individual-level confounders.

Background

Evidence of the importance of neighborhood conditions for active asthma comes from experimental and observational data.

Experimental Data

Due to their cost and difficulty implementing, experimental designs (i.e. mobility programs) are scarce. Nevertheless, one experiment has examined the relationship between neighborhoods and asthma morbidity. The Moving to Opportunity (MTO) study, sponsored by the Department of Housing and Urban Development (HUD), randomized families from high poverty areas (i.e., census tracts with more than 40% of the population at or below poverty level) and public housing to receive vouchers to pay

for rental housing from private landlords in census tracts with less than 10% poverty (Katz, Kling, and Liebman 2001). The Boston-based study found that families with children with asthma who moved to apartments in less poor neighborhoods experienced a 50% reduction in asthma attacks.

Observational Evidence

While most observational studies on asthma and neighborhoods are not multilevel—and thus pose substantive and statistical difficulties— the few studies utilizing multilevel techniques to disentangle neighborhood and individual-level contributors have generally found significant relationships between neighborhoods and health.[16] For instance, Juhn and colleagues (2005) examined the incidence of childhood asthma among all children born in Rochester, Minnesota, between 1976 and 1979, applying a multilevel survival model. Controlling for individual-level covariates (e.g. gender, birth weight, mother's age at birth, parental SES and parental educational level at birth), they found an increased risk of asthma among children living in census tracts that face intersections with highways or railroads, compared to those living in tracts that did not face intersections.

Similarly, Pearlman and colleagues (2006) drew on data from the 2001 Rhode Island Health Interview Survey (RI HIS), a statewide representative sample of 2,600 Rhode Island households, and the 2000 U.S. Census. Generalized estimating equations

[16] Observational studies consist primarily of ecological designs that fail to control for individual-level confounders or, more commonly, single-level studies that attempt to capture contextual influences (i.e. pollution, neighborhood violence) through individual- level measures. Substantively, it is difficult to tease part the separate contributions of individual- and neighborhood-level factors to asthma without using multilevel analyses. Statistically, single-level analyses fail to control for the clustering of individuals within neighborhoods (leading to a potential underestimation of the standard errors).

(GEE) with multivariate analyses were used to estimate the effects of race/ethnicity and SES on doctor-diagnosed asthma among 1,769 white, black and Hispanic children <18 years old. They found that both black and white children living in poverty neighborhoods had substantially higher odds of asthma than their counterparts in moderate- and high-income neighborhoods.

Finally, Cagney and Browning (2004) explored differences in asthma and other respiratory diseases among adults living in 338 Chicago neighborhoods, using multilevel techniques. Interestingly, they found no relationship between neighborhood structural characteristics (concentrated disadvantage, neighborhood stability) and respiratory diseases; however, neighborhood-level measures of collective efficacy were negatively associated with asthma and breathing difficulties.

Taken together, the above studies offer some evidence that neighborhoods may affect asthma; nevertheless, the work predominately focuses on childhood prevalence. Adult asthma remains understudied. This is problematic because hospitalization and mortality rates are at least twice as high for adult ethnic minorities (Apter 2003). Moreover, adult asthma can be a serious financial burden. Asthma-related costs increase monotonically with disease severity (Cisternas, Blanc, Yen, Katz, Earnest, Eisner, Shiboski, and Yelin 2003).

While Cagney and Browning's study analysis focused on adult asthmatics, their study contained serious methodological flaws. The outcome measure (respiratory diseases) failed to distinguish between asthma and COPD and instead categorized all respiratory conditions as a single outcome. Because these conditions differ in etiology, the role of neighborhoods in the onset of these respiratory conditions and the extent to

which neighborhoods influence each may vary in important ways. Moreover, the outcome measure captured lifetime, rather than current, asthma prevalence. Given that most cases of asthma are diagnosed by age 4, the majority of the subjects were likely diagnosed years before they moved into their current neighborhoods. As such, the category of asthmatics could include individuals who no longer experienced any symptoms, leading to a potential over or underestimation of neighborhood effects.

In light of the above limitation, understanding the causes of adult asthma, and its connection to neighborhood context, remains an important area of research. The current study attempts to address this gap by using single- and multi-level models to study the associations between a set of neighborhood structural characteristics (neighborhood affluence, concentrated disadvantage, immigrant concentration, and age composition) and currently active asthma. Similar to Cagney and Browning, the study utilizes multilevel techniques to examine neighborhood context in a Chicago-based adult population; however, it improve upon their measures of asthma/respiratory illness by operationalizing and modeling 12-month prevalence rates for asthma rather than lifetime prevalence rates, and by controlling for other respiratory illnesses. Furthermore, I estimate models on both the full sample as well as a sub-sample of asthmatics (an "at-risk" population.). This yields methodological and conceptual advantages elaborated in the methods section.

Methods

Data

The study's data come from the Chicago Community Adult Health Study (CCAHS), a multi-stage probability sample designed to investigate neighborhood effects

on individuals. From 2001 to 2003, the CCAHS collected neighborhood data on 3105 adults, aged 18 and older, who lived in 343 Chicago-based neighborhood clusters (NCs) previously defined by the PHDCN (Sampson, Raudenbush, and Earls 1997). One individual was interviewed per household, with a response rate of 71.8%. Each NC included two census tracts (approximately 8,000 individuals) with meaningful physical and social boundaries (Sampson, Raudenbush, and Earls 1997). Individuals in 80 focal areas identified by the PHDCN were sampled at twice the rate of those in others. The sample contains an average of 9.1 subjects per NC. All data and analyses were weighted to account for sample design, differential selection rates, household size, and differential coverage and non-response across NCs so that the weighted sample matches the 2000 Census population estimates for the city of Chicago in terms of age, race/ethnicity and sex.

The sample weight used in this analysis is a multiplicative combination of (1) a weight to adjust for the oversampling of cases in focal vs. non focal areas at a ratio of 2:1, (2) a weight to adjust for whether a respondent was selected for intensive non-response follow-up at the end of the survey vs. those eligible but not so selected at a ratio of 1:2, and (3) a combined non-response and post stratification weight, which was the inverse of the ratio of the proportion of respondents in each NC to the proportion of the eligible population in each NC by age, sex, and race/ethnicity. The weight was centered to have a mean of 1.0, with a standard deviation of 0.7, a minimum of 0.2, and a maximum of 5.4. Robust standard errors were used throughout the analysis.

Measures

Outcome Variables

Currently Active Asthma: The primary outcome is currently active asthma, a self-reported dichotomous variable. Respondents were first asked: "Has a physician or medical professional ever diagnosed you with asthma?" Those who responded "yes" to this question were then asked whether, in the last 12 months, they a) had asthma, b) saw a doctor or other health professional about asthma, or c) took prescription medicines or other prescribed treatments for asthma. An affirmative response to *any* of the follow-up categories was defined as active asthma. Survey questions using self-reported asthma based on physician diagnoses have among the highest specificity[17] (99%) and reliability rates in validation studies and are commonly used in epidemiological studies of adult-onset asthma (Toren 1993). Because the research focus is on currently active asthma (as opposed to previously diagnosed asthma), individuals with self-reported asthma but no self-reported symptoms (i.e., shortness of breath or trouble breathing) were excluded from this category. Sensitivity analyses revealed no substantial difference between these individuals and those with no asthma diagnosis and therefore were included in the non-asthma category.

Individual Covariates

Sociodemographic Factors. Epidemiological evidence suggests a patterning of asthma by gender, marital status, age, generational status, and socioeconomic status (Homa, Mannino, and Lara 2000; Mannino, Homa, Akinbami, Moorman, Gwynn, and Redd 2002; Rose, Mannino, and Leaderer 2006). The following demographic factors

[17] Specificity is the proportion of the truly health subjects found to be healthy using the questionnaire. A low specificity increases the risk of false positives, which could dilute the overall risk estimate.

were therefore included: *race/ethnicity* (categorized into [1] non-Hispanic black, [2] Hispanic, [3] other races, with non-Hispanic white as the omitted category), *age* (categorized into [1] ages 30-39, [2] 40-49, [3]50-59, [4] 60-69, [5] 70 or more, with ages 18-29 as the omitted category), *gender* (male=1), *marital status* (dummy variables categorized into [1] separated/ divorced, [2] widowed, [3] never married, with married as omitted category), *current family income* (dummies categorized into [1] less than $10,000, [2] $10,00-29,999, [3] $30,000-$49,999, with $50,000 and up as the omitted variable), *generational status* (categorized into [1] first generation, [2] second, with third or more as the omitted category) and *educational attainment* (categorized into [1] non-high school graduate, [2] high school graduate, [3] some college, [4] college degree, with some graduate school as the omitted category). Additionally, parental home ownership may influence subsequent currently active asthma through selection into future neighborhood residence. Though only retrospective measures are available, adult recall of parental home ownership has been shown to be accurate among middle-aged and older adults (Berney and Blane 1997; Krieger, Okamoto, and Selby 1998). Home ownership is a well-established indicator of income and adults can typically recall whether their parents owned or rented their homes. I coded parental homeownership as 1 if the respondent's parents owned their home during his/her childhood.

Health Behaviors/Health Care. Because obesity is an identified risk factor for adult asthma (Arif, Delclos, Lee, Tortolero, and Whitehead 2003; Camargo, Weiss, Zhang, Willett, and Speizer 1999; Chen 2002; Levy, Welker-Hood, Clougherty, Dodson, Steinbach, and Hynes 2004; Shaheen, Sterne, Montgomery, and Azima 1999), analyses adjusted for *obesity*, a dichotomous variable based on the respondent's body weight and

height (measured by the interviewer). Residents with a body mass index (BMI) of 30% or above were coded as obese. Residents in disadvantaged areas have higher smoking rates (Arif et al. 2003; Colilla, Nicolae, Pluzhnikov, Blumenthal, Beaty, Bleecker, Lange, Rich, Meyers, and Ober 2003; Shohaimi, Luben, Wareham, Day, Bingham, Welch, Oakes, and Khaw 2003; Thorn, Brisman, and Toren 2001; Toren 1993; Tseng, Yeatts, Millikan, and Newman 2001) and cigarette smoking is a risk factor for asthma (Thorn, Brisman, and Toren 2001; Toren 1993). As such, analyses controlled for *cigarette use* (categorized into [1] ever smoked 100 cigarettes, [2] smokes less than 6 cigarettes per day, [3] smokes 6-10 cigarettes per day, [4] smokes 11 or more cigarettes per day, with no longer smokes as the omitted category). Analyses also included dummies for active *chronic obstructive pulmonary disease*[18] (coded as 1 if the respondent experienced COPD in the last year), given its symptom similarities to asthma and risk of misdiagnosis (Tinkelman, Price, Nordyke, and Halbert 2006). Finally, dichotomous variables for *health insurance* (1 coded as "insured") and *regular source of medical care* (where 1 represents a "having a regular doctor or clinic") were included to account for the potential underdiagnosis of asthma due to differential access to medical care.

Neighborhood-level Exposures

Sociodemographic Structure. Building on previous research (Morenoff 2003; Morenoff, House, Hansen, Williams, Kaplan, and Hunte 2007; Sampson, Morenoff, and Earls 1999; Swaroop and Morenoff 2006), I used four census-based neighborhood level variables to characterize the sociodemographic structure of Chicago neighborhoods.

[18] Because COPD is a nonspecific term generally referring to a class of respiratory diseases (most commonly chronic bronchitis and emphysema), respondents with COPD, chronic bronchitis, or emphysema were considered, for the purposes of these study, as having COPD.

These variables were previously constructed from a principal components factor analysis with an orthogonal varimax rotation of 20 variables 2000 Census items that include NC-level measures of racial/ethnic composition, socioeconomic status, age composition, family structure, owner-occupied housing, and residential stability.[19] The goal of the factor analysis was to capture the shared variance for a broad range of neighborhood structural characteristics in a parsimonious set of factors.[20] All of the resulting factor scores were standardized to have a mean of zero and a standard deviation of one.

The first factor, interpreted as *concentrated disadvantage,* is characterized by strong positive loadings on the percentage of families with incomes of less than $10,000, the percentage of families in poverty, the percentage of families on public assistance, the percentage of unemployed adults in the civilian labor force, the percentage of families that are female-headed, and the percentage of adults who have never been married; and negative loadings on percentage of families with incomes of $50,000 and over and the percentage of owner-occupied homes. The second factor, *neighborhood affluence/gentrification,* has strong positive loadings for percentage of adults with 16 or more years of education, in professional or managerial occupations, and percentage of people ages 18-29 and 30-39; it has negative loadings on the percentage of people who lived in the same residence in 1995 and the percentage under age 18. The third factor,

[19] The advantage of utilizing factor loadings as opposed to specific variables (percent poor, percent black) to capture neighborhood structural characteristics is that it avoids the problem of collinearity. For instance, in urban segregated neighborhoods, the percentage of female-head households, the percent black, and the percentage of families below the poverty line are highly interrelated. The high collinearity of these variables renders it nearly impossible to tease out the unique contribution of any particular variable on the asthma outcome. Factor analysis addresses this methodological difficulty by grouping variables that have common characteristics into factors or dimensions. Each factor represents the latent characteristic of the environment measured by the interrelated set of variables. Moreover, each factor is as distinct as possible from other factors in the model. Therefore, factor analysis is a parsimonious way to captures the unique dimensions of neighborhood variance.

[20] The four factors together account for 96 percent of the variance in these variables. The first three factors have eigenvalues over 1.0 and the fourth has an eigenvalue of 0.86.

racial/ethnic/immigrant composition, has strong positive loadings for percent Hispanic and percent foreign-born and negative loader for percent non-Hispanic black. The final factor captures *older age composition*, with positive loadings for percent over the age of 70 and percent between ages 50-69, and negative loadings for the percent between ages 18-29 and the percent never married.

Analytic Strategy

The analysis aimed to assess whether neighborhood context contributed to currently active asthma. Means and standard deviations (SD) described the study population (Table 4.1). The primary analysis consisted of a series of hierarchical logit models (Table 4.2). An initial model regressed asthma on individual-level factors. Next, neighborhood-levels covariates were added to the model, according to equation (1):

$$(1)\ y_{ij} = \beta_{0j} + \beta_1 BLACK_{ij} + DX_{ij} + \alpha W_j + \mu_j$$

where y_i represents the natural logarithm of the odds ratio of person *i* in neighborhood *j* having asthma, β_{0j} is the average log odds of having active asthma for non-Blacks across all neighborhoods, β_1 represents the average differential risk of asthma associated with being black, *X* is a vector of individual-level covariates (excluding being black), *D* is the partial association between *X* and *y, W* is a vector of neighborhood-level covariates, α is the partial association between *W* and *y* (the likelihood of having asthma) and μ_j is the neighborhood-level variation not explained by *W*.

Finally, to assess whether potential neighborhood effects varied by the respondent's race, cross-level interactions between neighborhood factors and the black covariate were added, illustrated below.

$$(2)\ y_{ij} = \beta_{0j} + \beta_1 BLACK_{ij} + DX_{ij} + \alpha W_j + \gamma_1 W_j BLACK_{ij} + \mu_j$$

In this model, α is the partial association between *W* and *y* (the likelihood of having asthma) among non-Blacks, and γ_1 is the differential association between *W* and *y* for Blacks.

In addition to estimating these models for the full sample, additional analyses examined a restricted sample that included only those who experienced asthma in their lifetime (responded "yes" to lifetime prevalence) (Table 4.3). While such a procedure substantially reduces the sample size, the motivation for conducting these additional analyses relates to the etiology of asthma and the age of the study population. Asthma is a developmental disease, with the overwhelming number of cases diagnosed during childhood. It is highly unlikely that individuals in the study sample with no previous asthma diagnosis would develop asthma or express asthma symptoms, *regardless* of their residential location. Therefore, including the non-asthmatics in the sample—those whose results are presumably "fixed" at zero—may introduce unnecessary statistical noise into the analysis and potentially bias the effects of neighborhoods downward to non-significance. Since any presumed effect of neighborhood conditions on currently active asthma would most likely *only occur* among those previously diagnosed with asthma, the additional analyses were restricted to this "population-at-risk."

Results

Table 4.1 presents the individual-level summary statistics. About 6% of the full sample reported currently active asthma, rates comparable to the national average. Approximately a third of the sample was non-Hispanic white, 40% non-Hispanic black,

26% Mexican, 9% other Hispanics, and 3% other races. Over half the sample was female, below age 40, and attended at least some college. The average respondent was married, born in the United States, never smoked, had insurance, and access to medical care. Almost 40% earned less than $30,000 a year, grew up in rented homes, and 30% qualified as obese. Finally, about 5% had COPD in the last year.

[Table 4.1 about here]

Multivariate Analyses

Table 4.2 shows a series of models that introduce individual-level and neighborhood-level covariates. According to model 1, women exhibited an elevated risk of asthma compared to men, and those with less than a high school education or some college education had a higher risk than those with a college degree or more. Model 2 introduced a more extensive set of individual-level covariates, including health behaviors, obesity, and access to medical care. COPD was correlated with a higher risk of asthma, as was access to medical care. Blacks and Hispanics did not have a significantly higher asthma prevalence compared to whites; also individuals 50-59 were significantly less likely than 18-30-year-olds to have asthma. Model 3 incorporated neighborhood covariates with the individual-level models. Notably, measures of concentrated disadvantage, affluence, age composition, and immigrant concentration were not significant. Male gender, education, age, COPD, access to medical care, and obesity all remained significantly associated with asthma. Model 6 added cross-level interaction terms between neighborhood covariates and being black to determine whether the relationship between neighborhood factors and asthma varied by race. Interestingly, with

the exception of age concentration, the direction of the coefficients for all neighborhood covariates differed for blacks from other races. For instance, neighborhoods with a high proportion of Hispanic immigrants appeared to be protective against asthma for whites; the coefficient for blacks, though not significant, was in the opposite direction.

[Table 4.2 about here]

Table 4.3 presents results from the sub-sample of those with a lifetime prevalence of asthma. Similar to the full sample, education, COPD, and access to medical care were positively associated with asthma in the restricted sample. The effects for gender, significant in model 1, attenuated to non-significance in model 2.

The inclusion of neighborhood covariates (model 3) revealed no significant relationship between concentrated disadvantage, affluence, and immigrant composition, and asthma. Age concentration was positively associated with increased asthma risk, though the results were of borderline significance.

Model 4 added cross-level interaction terms between black race and neighborhood covariates. Once again, divergent neighborhood effects by race emerged. Neighborhood affluence and concentration of Hispanic immigrants appeared protective against asthma among whites, while, for blacks, affluence increased the risk of asthma and immigrant concentration had no effect on asthma for blacks.

[Table 4.3 about here]

Discussion

The aim of the current study was to explore differences in prevalence of currently active asthma at the neighborhood level among adults living in Chicago, while concurrently controlling for a range of individual-level confounders. I improved upon past work by using a more precise measure of asthma (based on 12-month prevalence rates that adjust for COPD), by focusing on an understudied population (adult asthmatics), and by analyzing both the full sample and a "population-at-risk."

Interestingly, none of the neighborhood characteristics predicted asthma, either when looking at the full sample or the subsample of those with a history of asthma. The absence of a significant effect for asthma, though striking in its consistency, is congruent with Cagney and Browning (2004), who found no relationship between neighborhood structural characteristics (concentrated disadvantage, residential stability) and breathing difficulties and asthma. Notably, the authors did identify a significant protective effect of health-related collective efficacy on breathing difficulties, and hypothesized that the social organization of the neighborhood—rather than poverty itself—may be the most salient predictor of asthma. While the current study did not look at collective efficacy, future research could examine such neighborhood domains to discern whether neighborhood social organization may be relevant for asthma.

A somewhat more complex picture emerged with the inclusion of the cross-level interactions. These models, which examined the differential effects of neighborhoods by race, revealed contrasting trends between blacks and the remaining sample. For whites and Hispanics, neighborhoods with a high concentration of Hispanic immigrants were less likely to have asthma than those with a lower concentration. Similarly, neighborhood affluence was highly associated with a reduced risk of asthma for whites

and Hispanics in the restricted sample. For blacks, on the other hand, neighborhood immigrant concentration had no effect on asthma prevalence, and affluence appeared to *increase* the risk of asthma. Likewise blacks living in disadvantaged neighborhoods (in the full sample alone) had a borderline *decreased risk* of asthma.

One possibility for the disparate findings is that blacks and whites residing in similar neighborhoods may experience these neighborhoods differently. For instance, blacks in affluent, predominately white settings may face greater exposure to discrimination and feelings of isolation or inferiority than blacks in racially integrated or predominately black neighborhoods. Moreover, they may continue to face individual-level risk factors for asthma, such as indoor allergens or occupational hazards, despite residing in more affluent areas. Though the neighborhood physical toxins may be fewer, the neighborhood social stressors and individual-level pathogen may be equal, if not greater. In such cases, the lived experience of blacks—and their relationship to neighborhoods—may differ importantly from other ethnic groups.

At the same time, it is important to interpret these findings cautiously. First, the results could be due to unmeasured heterogeneity. For example, blacks living in highly affluent neighborhoods may have a lower rate of under diagnosis, whereas those in highly disadvantaged areas may face substantial barriers to medical care (either because of reduced access to medical clinics or fear of leaving one's home). Since asthma was derived from a physician diagnosis, the negative association may reflect underdiagnosis or unreported conditions. Second, and more seriously, the small sample sizes of black and/or white asthmatics used in the analysis, especially in the restricted sample, may have generated unstable interaction terms, leading to potential "false positives." Given the

unexpected direction of the neighborhood coefficients for blacks, the notable non-significance of any main effects, and the small cell size of the interaction terms, a far more likely explanation is that the results are statistical artifacts and should therefore not be trusted.

Limitations of the study should be noted. First, and perhaps most significantly, the data are cross-sectional. As such, no causal inferences can be made. At best, the analysis can identify associations between individual or neighborhood factors and asthma. Second, because the asthma prevalence is quite low in the sample relative to the overall number of neighborhoods, insufficient statistical power may explain the lack of a neighborhood effect. Third, there are no questions about the respondent's housing unit or household characteristics (e.g., potential allergens, pet ownership, and housing quality).[21] Since a neighborhood effect may simply reflect unaccounted for household-level predictors, significant results (i.e., in the interaction terms) may be due to omitted variable biases. Finally, the use of multilevel methods raises the issue of endogeneity (Jencks and Mayer 1990). Some argue that segregation may be the result of poor economic outcomes or reflect omitted city characteristics, rather than its cause. These factors could result in spurious associations between segregation and black outcomes. Likewise, individuals prone to poor health may self-select into neighborhoods; ignoring these selection factors may overestimate the negative effects of neighborhood residence on health (Jencks and Mayer 1990; Kling, Liebman, and Katz 2007; Oakes 2004).

[21] For example, a person residing in a newly renovated, dust-free apartment will presumably have a lower risk of asthma than his/her neighbor living in a moldy, rodent-infested apartment, regardless of overall neighborhood quality. The data provide no direct way of differentiating between asthma caused by neighborhood factors (i.e., pollution) versus asthma caused by poor household conditions.

Future research could build on this study in several ways. First, a priority should be placed on understanding whether and how specific neighborhood attributes increase the risk of asthma. As noted above, because of the small sample size of asthmatics, the null effects in this study may be due to insufficient statistical power. Epidemiological data designed specifically to study asthma could more appropriately address this question.

Second, further research could also explore the potential role of wider geographic context and the spatial interdependencies between neighborhoods as a factor in asthma. An emerging body of research has shown that surrounding neighborhoods may be important for an individual's health (Morenoff 2003; Morenoff, Sampson, and Raudenbush 2001; Sampson, Morenoff, and Earls 1999). For instance, if a neighborhood has a low crime rate but the areas around it all have high crime rates, then the elevated crime rates in the adjacent areas could still cause significant stress for residents living in the low-crime neighborhoods, as demonstrated by Morenoff (2003). A spatial-based framework may elucidate why concentrated disadvantage was not significant in the current analysis. Since this study only focused on internal neighborhood properties, it ignored any "spillover effect" that may emerge from surrounding neighborhoods, thus underestimating the more extended contextual effects. By combining multilevel hierarchical models with spatial regression models, future research could consider the potential role of the wider spatial context within which a neighborhood is embedded in affecting asthma.

Finally, additional research should explore in greater depth the complex interplay between race, neighborhood context, and respiratory illness. The current study is a first

step toward understanding the role that "place" may or may not have in the etiology of asthma prevalence. To the extent that the pathways (both proximal and distal) to asthma pathogenesis vary by ethnicity/race, tailoring health interventions to each population may be the most effective means to reduce the excess asthma burden. To this end, further research on a larger sample size or a broader neighborhood unit of analysis would prove useful in identifying whether and how race mediates the relationship between neighborhoods and health.

Table 4.1 Descriptive Statistics (n=3105)

	N	Mean	SD
Asthma			
In Last 12 Months	207	0.06	(0.01)
Not in Last 12 Months	2898	0.94	(0.01)
Lifetime Prevalence	384	0.16	(0.01)
Race			
White	983	0.32	(0.02)
Black	1240	0.4	(0.02)
Hispanic	802	0.26	(0.02)
Other	80	0.04	(0.01)
Gender			
Male	1870	0.47	(0.01)
Female	1235	0.53	(0.01)
Age			
18-29	800	0.28	(0.01)
30-39	748	0.23	(0.01)
40-49	608	0.19	(0.01)
50-59	402	0.13	(0.01)
60-69	286	0.09	(0.01)
70+	261	0.09	(0.01)
Education			
No High School Degree	792	0.23	(0.01)
High School Graduate	759	0.24	(0.01)
Some College	817	0.25	(0.01)
College Graduate +	737	0.28	(0.02)
Income			
Less than $10,000	365	0.1	(0.01)
$10,000-$29,999	876	0.26	(0.01)
$30,000-$49,998	581	0.18	(0.01)
$50,000 and above	698	0.26	(0.01)
Missing	585	0.19	(0.01)
Generational Status			
1st Generation	773	0.27	(0.02)
2nd Generation	378	0.14	(0.01)
3rd Generation +	1954	0.59	(0.02)
Marital Status			
Married	1091	0.42	(0.01)
Sep/Divorced	584	0.15	(0.01)
Widowed	257	0.07	(0.01)
Never Married	1173	0.37	(0.01)
Weight			
Not Obese	2039	0.68	(0.01)
Obese	1066	0.32	(0.01)

Table 4.1 Descriptive Statistics (continued) (n=3105)

	N	Mean	SD
Smoking			
Never Smoked	1675	0.54	(0.01)
Past Smoker (>100 cig)	615	0.2	(0.01)
Smokes <6 cig daily	273	0.09	(0.01)
Smokes 6-10 cig	250	0.07	(0.01)
Smokes >10	292	0.09	(0.01)
COPD			
In Last 12 Months	155	0.05	(0.01)
Not in Last 12 Months	2950	0.95	(0.01)
Access to Medical Care			
Yes	2363	0.75	(0.01)
No	742	0.25	(0.01)
Insured			
Yes	2466	0.79	(0.01)
No	659	0.21	(0.01)
Parental Home Ownership			
Rented	1373	0.4	(0.01)
Owned	1732	0.6	(0.01)

Table 4.2 Hierarchical Logit Models of Active Asthma on Full Sample (n=3105)

Predictors	1		2		3		4	
				Individual-Level Factors				
Race (White)								
Black	0.14	(0.24)	0.26	(0.30)	0.15	(0.43)	0.04	(0.38)
Hispanic	0.09	(0.38)	0.08	(0.45)	0.17	(0.45)	-0.04	(0.46)
Other	-0.96	(0.77)	-0.77	(0.82)	-0.76	(0.83)	-0.88	(0.88)
Male	-0.82**	(0.21)	-0.72**	(0.24)	-0.69**	(0.24)	-0.70**	(0.24)
Age (18-29 ref)								
30-39	-0.17	(0.29)	-0.40	(0.32)	-0.41	(0.32)	-0.42	(0.33)
40-49	0.28	(0.26)	-0.06	(0.26)	-0.08	(0.26)	-0.09	(0.27)
50-59	-0.37	(0.33)	-1.14**	(0.35)	-1.20**	(0.36)	-1.23**	(0.36)
60-69	0.15	(0.36)	-0.43	(0.46)	-0.54	(0.45)	-0.53	(0.45)
70+	-0.48	(0.41)	-0.73	(0.54)	-0.80	(0.53)	-0.81	(0.54)
Income (50+ ref)								
<10k	0.17	(0.36)	-0.17	(0.36)	-0.11	(0.36)	-0.09	(0.35)
10-<30k	0.37	(0.29)	0.17	(0.29)	0.21	(0.29)	0.21	(0.30)
30-<50	-0.24	(0.31)	-0.23	(0.31)	-0.20	(0.32)	-0.22	(0.31)
Education (College+ ref)								
< High School Grad.	1.02**	(0.33)	0.93*	(0.36)	0.95**	(0.36)	0.98**	(0.36)
High School Grad	0.31	(0.30)	0.16	(0.34)	0.17	(0.34)	0.17	(0.34)
Some College	0.76*	(0.31)	0.54	(0.34)	0.51	(0.34)	0.54	(0.34)
Obesity	-		0.43+	(0.22)	0.41+	(0.22)	0.43+	(0.22)
Smoking (never ref)	-							
Past Smoker (>100 cig)	-		-0.08	(0.30)	-0.06	(0.30)	-0.09	(0.30)
Smokes <6 cig daily	-		0.27	(0.33)	0.27	(0.34)	0.25	(0.34)
Smokes 6-10 cig	-		-0.14	(0.37)	-0.16	(0.37)	-0.18	(0.37)
Smokes >10	-		0.59+	(0.32)	0.58+	(0.32)	0.53	(0.31)
COPD	-		2.88**	(0.23)	2.92**	(0.24)	2.95**	(0.24)
Access to Medical Care	-		0.64*	(0.29)	0.67*	(0.30)	0.64*	(0.29)
Insured	-		0.12	(0.28)	0.09	(0.27)	0.11	(0.27)
Parents Rented Home	-		-0.17	(0.22)	-0.16	(0.21)	-0.14	(0.21)
Intercept	-2.91**	(0.33)	-3.80**	(0.49)	-4.54**	(0.53)	-4.24	(0.54)
				Neighborhood-Level Factors				
Constant								
Disadvantage	-		-		-0.08	(0.12)	0.15	(0.16)
Affluence	-		-		-0.02	(0.11)	-0.19	(0.14)
Hispanic Immigrant	-		-		-0.13	(0.16)	-0.37*	(0.19)
Age Concentration	-		-		0.12	(0.11)	0.07	(0.13)
Black*Interaction								
Disadvantage	-		-		-		-0.37+	(0.21)
Affluence	-		-		-		0.30	(0.25)
Immigrant Concentration	-		-		-		0.42	(0.32)
Age Concentration	-		-		-		0.13	(0.23)

Note: Standard errors in parentheses.

** $p \leq 0.01$, * $p \leq 0.05$ + $p \leq 0.10$

All models control for generational status and marital status

Table 4.3 Hierarchical Logit Models of Active Asthma Among Those with Lifetime Prevalence of Asthma (n=384)

Predictors	1		2		3		4	
				Individual-Level Factors				
Race (White)								
Black	0.37	(0.32)	0.54	(0.36)	-0.02	(0.10)	-0.05	(0.09)
Hispanic	0.45	(0.51)	0.66	(0.51)	0.14	(0.10)	0.09	(0.11)
Other	-0.45	(1.03)	-0.00	(1.25)	-0.01	(0.26)	-0.03	(0.26)
Male	-0.65*	(0.30)	-0.50	(0.34)	0.08	(0.06)	0.07	(0.06)
Age (18-29 ref)								
30-39	0.21	(0.38)	0.17	(0.42)	-0.01	(0.07)	-0.04	(0.07)
40-49	0.72+	(0.40)	0.27	(0.49)	0.01	(0.08)	-0.04	(0.08)
50-59	-0.32	(0.43)	-1.20+	(0.68)	-0.21*	(0.10)	-0.24*	(0.10)
60-69	0.32	(0.53)	-0.38	(0.78)	-0.13	(0.11)	-0.14	(0.11)
70+	-0.03	(0.51)	-0.47	(0.68)	-0.09	(0.13)	-0.09	(0.12)
Income (50+ ref)								
<10k	0.28	(0.52)	-0.22	(0.65)	-0.04	(0.09)	-0.06	(0.10)
10-<30k	0.32	(0.40)	0.36	(0.47)	0.04	(0.08)	0.05	(0.07)
30-<50	-0.13	(0.44)	-0.10	(0.51)	-0.02	(0.09)	-0.04	(0.09)
Education (College+ ref)								
< High School Grad.	1.10*	(0.44)	1.04*	(0.52)	0.15	(0.09)	0.13	(0.09)
High School Grad	0.66	(0.43)	0.70	(0.45)	0.15+	(0.08)	0.14+	(0.08)
Some College	0.87*	(0.41)	0.67	(0.45)	0.12	(0.09)	0.11	(0.08)
Obesity	-		-0.34	(0.34)	-0.05	(0.05)	-0.04	(0.05)
Smoking (never ref)	-							
Past Smoker (>100 cig)	-		0.16	(0.42)	0.01	(0.07)	0.00	(0.07)
Smokes <6 cig daily	-		0.04	(0.59)	0.02	(0.10)	0.03	(0.10)
Smokes 6-10 cig	-		-0.37	(0.53)	-0.03	(0.09)	-0.04	(0.09)
Smokes >10	-		0.12	(0.52)	0.00	(0.08)	0.00	(0.08)
COPD	-		3.99**	(0.86)	0.54**	(0.05)	0.54**	(0.05)
Access to Medical Care	-		1.01*	(0.46)	0.20*	(0.08)	0.19*	(0.07)
Insured	-		-0.03	(0.42)	-0.05	(0.07)	-0.04	(0.07)
Parents Rented Home	-		-0.15	(0.28)	-0.01	(0.05)	-0.01	(0.05)
Intercept	-0.74	(0.48)	-1.65*	(0.73)	0.22	(0.14)	0.38**	(0.14)
				Neighborhood-Level Factors				
Constant								
Disadvantage	-		-		-0.03	(0.03)	0.02	(0.05)
Affluence	-		-		-0.05	(0.03)	-0.12**	(0.03)
Hispanic Immigrant	-		-		-0.06	(0.04)	-0.16**	(0.04)
Age Concentration	-		-		0.05+	(0.03)	0.04	(0.04)
Black*Interaction								
Disadvantage	-		-		-		-0.07	(0.06)
Affluence	-		-		-		0.23**	(0.07)
Immigrant Concentration	-		-		-		0.16+	(0.09)
Age Concentration	-		-		-		-0.04	(0.07)

Note: Standard errors in parentheses.
** $p \leq 0.01$, * $p \leq 0.05$ + $p \leq 0.10$
All models control for generational status and marital status

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CHAPTER 5

CONCLUSION

The goal of this dissertation is to examine the contextual determinants of racial disparities in health across the life course. Though racial disparities persist across multiple measures of health status, the sources of these differences are not well understood. I hoped to shed light on this question by conducting three studies progressing from "downstream" to "upstream" processes linking race to health disparities. As noted in the introduction, the three papers are not intricately related, each examining a different dataset, sample population, and health outcome. However, together, these papers explore the role of spatial and temporal contexts in initiating, perpetuating, and perhaps exacerbating health disparities.

Key Findings

Chapter 2 examines the relationship between lifetime exposure to abuse among 643 pregnant women in the Boston area and elevated cord blood IgE. Results from this prospective study show a graded association between lifetime exposure to violence and cord blood IgE, a biological marker for atopic asthma. Greater exposure to violence throughout one's life course is independently associated with increased risk of offspring elevated IgE after simultaneously adjusting for maternal and family-level confounders. In contrast, no association is found between proximal abuse (during pregnancy) and

elevated IgE in the unadjusted and adjusted model suggesting that the cumulative exposure to violence over all ages, rather than any specific abuse experience, may be the most salient factor in fetal effects. The results indicate that the detrimental effects of violence may a) accumulate over the life course and b) transmit across generations through the fetal environment.

Chapter 3 continues the exploration of the intergenerational transmission of disadvantage by examining the relationship between teen childbearing and offspring health among a nationally representative sample of children ages 5-19. In this paper, I test two competing theories: the "social causation/developmental" hypothesis, which posits that teenage childbirth causes poor offspring health, and the "culturally adaptive" hypothesis, which argues that teenage childbirth can be protective. Logistic regression models reveal no increased risk of low birthweight, chronic illness, obesity or asthma among offspring of teen mothers versus non-teens, and a slight decrease in obesity among offspring of teens. These results indicate that the timing of one's pregnancy, may matter far less than other contextual factors in influence offspring health, and may not be a salient factor in causing poor health outcomes.

Finally, Chapter 4 shifts from a temporal to a spatial context. This paper uses multilevel methods to examine differences in currently active asthma at the neighborhood level among adults living in Chicago. The paper assesses the extent to which neighborhood sociodemographic characteristics contribute to asthma, net individual-level confounders. Interestingly, I find no association between neighborhood factors and currently active asthma, either when examining the full sample or a subsample of previously diagnosed asthmatics. Though cross-level terms interacting black race with

neighborhood affluence and immigrant concentration emerge as significant, the unexpected direction of the coefficients, along with the extremely small cell size, cast doubt on the reliability and validity of these findings.

Taken together, these three studies present a complex and dynamic picture of how contextual factors affect health outcomes. Their relative importance, and when and how they influence health, may be contingent upon key conditions (demographics, SES, race, ethnicity). For instance, the findings in Chapter 2 support both a "critical period model" and a "cumulative effect" life course framework. The "critical period model" posits that exposure during a specific period has lasting or lifelong effects on the structure or function of organs, tissues and body systems. In Chapter 2, the fetal environment operates as the critical period since this is when the fetal immune system is primed toward an allergic phenotype. However, the findings also reveal the importance of the "cumulative effects model," which posits that factors that raise disease risk may accumulate gradually over the life course. It is the maternal *cumulative* exposure to violence throughout her life course, rather than any direct experiences of abuse during her pregnancy, which most potently creates a harmful gestational environment, the very critical period in which the asthma phenotype is shaped. Thus, a complex interplay emerges between cumulative effects and critical periods theories in the intergenerational transmission of asthma risk.

Chapter 3, in contrast, finds little support for either life course perspective, since maternal age at birth is unrelated to offspring health. The two competing hypotheses on teen childbearing each assume a specific life course perspective. The developmental hypothesis, which argues that mothers are biological unfit to bear children, presupposes a

critical period model since its claim regarding the mother's physiologic immaturity is based on the notion that events occurring *in utero* (e.g., nutritional deprivation, hormonal imbalance) are critical for offspring health. The culturally adaptive hypothesis, which argues that *early* fertility-timing may be a culturally adaptive means of maximizing offspring wellbeing, presupposes a cumulative effects model since it posits that an accumulation of health insults beginning in infancy leads to early health deterioration and excess mortality. In finding that teen pregnancy is neither protective nor harmful; the study therefore provides little support for either life course perspective. Irrespective of the physiological maturity of teens versus non-teen moms, their offspring's health is equivalent, and as such, there is no reason to think that the critical period model applies. Similarly, any cumulative deterioration that might accompany the aging process does not transmit into poor offspring health. Therefore, in contrast to Chapter 2, Chapter 3 provides little evidence in favor of life course models.

The varying complexity of the temporal context is paralleled by similar complexities in the spatial context. Chapter 4 suggests that neighborhood context (i.e., where respondents live) is unrelated to currently active asthma, at least when looking at all races in aggregate. The cross-level interactions, however, reveal potentially differing effects of neighborhoods on blacks and whites. While these results must be viewed cautiously, they nevertheless raise the possibility that the pathways (both proximal and distal) linking neighborhoods to health vary importantly by ethnicity/race.

What emerges from these three studies is a complex picture of how and when contextual factors may affect health disparities. Overall, the findings confirm the value of incorporating contextual factors into analyzing racial disparities in health. At the same

time, they underscore the pitfalls in overlooking the diversity in age, ethnicity, life stage, exposure, and health outcomes in understanding health disparities—and the critical need to account for this diversity in future research and policy.

Limitations

The studies contain several limitations. These limitations can be divided into those relating to individual papers, and those relating to the thesis as a whole.

Limitations of Individual Papers

Each of the papers exhibit data limitations. Chapters 2 and 3 (using the ACCESS data and PSID data, respectively) both face sample attrition and missing data on key predictor variables, resulting in potential selection biases and non-generalizable results. Chapter 4 uses cross-sectional data, and, as such, no causal inferences can be derived from the findings. Moreover, the asthma rates are extremely low, presenting substantive problems with statistical power in disentangling neighborhood- and individual-level effects.

There are also limitations with the measurements in all three studies. The measures of lifetime abuse history in Chapter 2 are self-reported and retrospective, leading to potential recall bias (especially in recounting childhood abuse) and, social desirability response bias, given the sensitivity of the topic. In Chapters 3 and 4, the key outcome variables (with the exception of birth weight in Chapter 3) are based on physician diagnoses. Given that disadvantaged populations—those most likely to have teen mothers or reside in poor neighborhoods—tend to underutilize medical services, the

measures may have overlooked sick individuals who either lacked access to medical care or failed to see a doctor. Moreover, Chapter 4 has no questions about the respondent's housing unit or household characteristics (e.g., potential allergens, pet ownership, and housing quality) and therefore can not detect important within-neighborhood, between-household variation in housing quality or asthma-related risk factors.

Finally, the statistical techniques in Chapters 3 and 4 raise methodological concerns. Chapter 3 uses standard regression models, instead of fixed effect sibling comparisons, to assess the health effects of teen pregnancy, because of the insufficient number of sibling pairs. Since standard controls cannot control for all sources of unobserved heterogeneity, the results may be biased by this residual confounding. Chapter 4 employs hierarchical linear models, which, despite its strengths, faces issues of endogeneity and neighborhood selection (Jencks and Mayer 1990). Ignoring these selection factors may over or underestimate the effects of neighborhood residence on health (Jencks and Mayer 1990; Kling, Liebman, and Katz 2007; Oakes 2004).

Limitations of Thesis as a Whole

Though my dissertation provides foundational research by looking at ecological (neighborhood), proximal, and physiological processes, it does not full integrate these pieces into a single analysis. Rather, each chapter explores contextual factors at specific levels: Chapter 2, at the proximal (e.g. stress) and biological (IgE) level; Chapter 3, at the proximal level (fertility behavior); and Chapter 4, at the macro/meso level (segregation, neighborhood social and environmental conditions). When combined, the three studies suggest at potential avenues of interconnection between the upstream and downstream

factors. However, no empirical analysis directly tests the pathways linking distal and proximal causes of health disparities. Thus, the dissertation study does not provide an empirically tested conceptual framework for how distal factors may "get under the skin (i.e. illuminating whether and how neighborhood segregation may produce elevated IgE).

Similarly, though time and place are individually addressed in one or more of the dissertation chapters, they are never examined simultaneously. This is an important omission because each may inform the other's relationship to health. For instance, residence in poor neighborhoods may damage one's health, but the *extent* of this damage may vary considerably by the duration of tenure, the age of the individual (whether s/he is a child or adult), and the individual's residential history. Likewise, *in utero* exposure to violence may adversely affect offspring IgE, but the effects may be substantially worse for mothers living in polluted or unsafe neighborhoods. In both instances, accounting for the timing of exposure and the broader social environment would our understanding for how contextual factors influence health.

Finally, each chapter examines individuals at distinct life course stages (fetuses, children, teens, and adults); however, no single analysis tracks individuals throughout their life course. While the current presentation can provide "snap shots" for how people at different life stages differentially respond to contextual factors, the samples are distinct demographically, geographically, and financially. Findings from one study are therefore not generalizable to the other two sample populations.

Future Research

Given the existing limitations and the current state of evidence on health

disparities, there are several avenues for future research. First, further research is necessary to understand the various upstream and downstream factors causing health disparities, and the mechanisms traversing these levels. At the ecological level, additional attention should be paid to whether and what neighborhood factors lead to racial disparities in asthma, given that the current data were not equipped to address this question. At the more proximate level, further research could explore alternate pathways through which violence exposure may lead to asthma pathogenesis. My existing work has only tested the "direct effects" hypothesis—whether exposure to violence causes asthma by triggering asthma-inducing physiological processes. However, violence exposure may also operate indirectly by encouraging health behaviors (smoking, staying indoors) that heighten exposure to other known environmental risk factors. It may therefore be helpful to examine the relationship between *in utero* violence, health behaviors, and IgE.

Second, future research should simultaneously consider temporal and neighborhood contexts in understanding health disparities. Specifically, this entails bringing a life course approach into multilevel research on neighborhoods and asthma, and asking questions such as: do the adverse effects of neighborhoods on asthma accumulate over time or are they specific to a particular life stage? Do past neighborhood effects interact multiplicatively (as opposed to independently) with current neighborhood exposures, such that a previous tenure in a toxic neighborhood magnifies the health effects of current tenure? Does the relationship vary by race (i.e., are blacks more vulnerable to neighborhood exposures during childhood than whites)? It also means incorporating multilevel methods into the examination of the cumulative effects of

abuse and/or other stressors and intergenerational transmission of health disadvantage. For example, does *in utero* exposure to family violence operate synergistically with neighborhood violence such that victimized pregnant women living in socially toxic areas have offspring with the highest IgE or stress levels? Does a mother's residential history affect fetal IgE levels, independent of current exposure? If so, are there specific critical periods in the mother's life course (childhood, adulthood) in which neighborhood influences would be most potent?

Third, future research could employ additional statistical techniques to expand the scope of multilevel and temporal analyses. For instance, additional work could explore the potential role of wider geographic context and the spatial interdependencies *between* neighborhoods as a factor in health disparities. An emerging body of research has shown that surrounding neighborhoods may be important for an individual's health (Morenoff 2003; Morenoff, Sampson, and Raudenbush 2001; Sampson, Morenoff, and Earls 1999). By combining multilevel hierarchical models with spatial regression models, future research could consider the potential role of the wider spatial context within which a neighborhood is embedded in affecting racial disparities in asthma.

Additionally, growth curve analysis could more effectively capture the dynamic relationship between maternal exposures (abuse, or teen pregnancy) and offspring health trajectories. For instance, findings from Chapter 2 indicate that IgE levels are elevated among children of abused mothers, but it is unknown whether/how their respiratory functions develop as they age, whether they differ notably from children with no abuse history, and how current life stressors differentially affect their health. Likewise, Chapter 2 revealed no health differences between offspring of teen versus non-teen mothers

(Chapter 3), but it is possible that the adverse health effects only emerge as the children reach adulthood. Growth curves are the most appropriate statistical models to test for these possibilities since they estimate the full trajectory of change both within an individual's measurement points (the same person at different ages), and across groups of individuals. Since the PSID and the ACCESS are longitudinal studies that have or intend to collect multiple waves of data, they possess the necessary data points to conduct such analyses.

Finally, future research could explore psychological factors related to health disparities. The current dissertation addresses the physical health domain solely. Chapter 2, for instance, considers the adverse effects of physical and sexual abuse, but not psychological abuse. Psychological abuse, however, has been linked to post-traumatic stress disorder (PTSD) (Basile, Arias, Desai, and Thompson 2004) and other adverse physical outcomes (Coker, McKeown, and Alerts 2000), and may be just as damaging as other types of violence (Claussen and Crittenden 1991; Egeland, Sroufe, and Erickson 1983). Similarly, Chapter 3 examines a range of physical, but not psychological, consequences, of teen childbearing. It is possible that teen motherhood damages offspring's psychological health, even if their physical health remains intact. Additional research is necessary to explore these possibilities.

These avenues for future research will build on the findings of this dissertation by offering greater insight into the role of temporal and spatial contextual factors. Further study will bring greater clarity about how contextual factors initiate and/or perpetuate racial disparities in health to the complex picture presented here.

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