# CHAPTER 1
# INTRODUCTION AND STUDY RATIONALE

## Overview of the problem

Racial inequalities in health are widely documented, their causes and solutions discussed and debated, and attempts to eliminate them implemented; yet the Black-White gap in health persists (Geronimus & Thompson, 2004; Williams, 1999).  The National Center for Health Statistics (2011) reported that African Americans have a greater risk of dying from 7 out of the 10 leading causes of death in the United States .  Researchers theorize that more frequent, persistent, and severe experiences of lifetime stress may result in the higher incidence of mortality and morbidity for Black versus White Americans (George & Lynch, 2003; Geronimus, 2001; Geronimus & Thompson, 2004; Jackson, Knight, Carstensen & Schaie, 2006; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Pearlin, Schieman, Fazio, & Meersman, 2005; Turner & Turner, 2005).  This differential exposure to stressors and unequal distribution of psychological and physiological stress may be a largely overlooked fundamental cause of Black-White health inequalities.  The studies described in this dissertation will address this hypothesis by focusing on causes of stressor exposure and stress for Black Americans.

Compared to Whites, Black Americans experience more frequent chronic and acute stressors, stressful and traumatic life events, after controlling for indicators of socio-economic position (Jackson et al., 2006; Kessler & Neighbors, 1986; Turner & Lloyd, 1995).  Frequent and cumulative experiences of stress may be related to increases

in chronic disease (Geronimus & Thompson, 2004; Wright, Rodriguez & Cohen, 1998), lowered immune functioning (Friedman & Lawrence, 2002), poor psychological and mental health (Schulz et al., 2000; Turner & Turner, 2005; Williams, Yu, Jackson, & Anderson, 1997), and deleterious coping behaviors (Jackson et al., Friedman & Lawrence, 2002; 2006).  Early studies on stress and health focused disproportionately on recent life events and acute stressors, which may vastly underestimate stress exposure. Pearlin (1989) described the social patterning of stress and noted that it is deeply rooted in structural contexts that affect health prior to birth.  A cumulative and life course perspective suggests that Black Americans are likely to experience more frequent and intense stressors than their non-Black counterparts.

Although the historical and contemporary background of racism, segregation, and concentrated poverty in the United States is expansive, a detailed review is beyond the scope of this dissertation and can be found elsewhere (Conley, 1999; Massey & Denton, 1993; Wilson, 1987).  Numerous researchers and scholars cite, however, that race and racism has shaped U.S. social systems and has resulted in discriminatory policies and practices, which limit the opportunities and social mobility for African Americans (Oliver & Shapiro, 2006; Williams, et al., 1997).  This research will focus on indirect pathways between racism and stress via the physical and social environment (neighborhood), which is shaped largely through residential segregation and resulting concentrated poverty (Clark, Anderson, Clark, & Williams, 1999; Williams, et al., 1997).  More specifically, this series of three studies will explore the ways in which the social and physical environment, which is shaped by a historical context as well as the current context of racism and segregation (Conley, 1999; Massey & Denton, 1989), differentially influences

the prevalence of psychological and physiological stress in Black versus White Americans.

## Conceptual Model

Figure 1.1 displays the conceptual model connecting neighborhood factors (shaped by the aforementioned fundamental social and political structures) to stress, as well as several important mediators and moderators of this relationship.

**Stress**

Despite the ordinary nature of the word, the meaning of *stress* is ambiguous. The stress response, for instance, may be reflected in single cells or organs; in biochemical, physiological and emotional functioning; in entire body systems like the endocrine, immunologic and cardiovascular systems; or in the incidence of physical or psychological distress (Pearlin, et al., 1981). Research frequently interchanges the stimuli (stressor) with the outcome (stress), which results in a muddled literature connecting neighborhood-level stressors to a psychological and physiological stress response. For the purposes of this research, *stress* is defined as the physiological or psychological (perceived stress) outcome and *stressor* as the stimulus of the outcome (i.e., neighborhood disadvantage, crime). This research will examine both psychological and physiological stress (cortisol) to more fully capture the connection between context and stress.

Recently, researchers have focused on physiological indicators of stress (biomarkers), as these physiological responses may more adequately represent the connection between chronic stressor exposure and health, and may therefore enable researchers to study a lifetime of stressor exposure and *wear and tear* on the body

(Geronimus, Hicken, Keene, & Bound, 2006; Geronimus & Thompson, 2004; McEwen & Seeman, 1999). Allostatic load represents one way in which to assess this wear and tear. When healthy, the body maintains a state of equilibrium or allostasis; the physiological stress response system becomes activated during periods of stress and deactivated when the threat of stress declines (McEwen & Seeman, 1999). The stress response system becomes hyper-activated under conditions of chronic stressor exposure, resulting in allostatic load, (McEwen & Seeman, 1999) which is associated with harmful cardiovascular, cognitive, neuronal, immunosuppressive, metabolic and immune disorders (McEwen & Seeman, 1999). Cortisol, which is one biomarker included in the assessment of allostatic load, will be examined in this research as an indicator of physiological stress.

**Neighborhoods and stressor exposure**

One context in which Black Americans may be disproportionately exposed to chronic stressors is within the neighborhood. Researchers have a growing awareness of the role neighborhoods[1] play in influencing health and health behavior (Browning & Cagney, 2003; Diez Roux et al., 2001; Diez Roux, 2001; 2003; Ellen, Mijanovich, & Dillman, 2001; Freudenberg, 2005; Morenoff, 2003; Morenoff et al., 2007; Wen, Cagney, & Christakis, 2005). Research indicates that neighborhoods characterized by segregation, poverty, crime, violence, residential instability, social isolation, and unemployment have a detrimental effect on the health and behavior of the residents (Browning & Cagney, 2003; Ellen, et al., 2001; Hill, Ross, & Angel, 2005; Kruger,

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[1] The definition of neighborhood is widely debated in the literature. In this paper neighborhood and community will be used interchangeably, and refer to administrative (Census) boundaries of an area. I assume that residents of a neighborhood and community have frequent contact with each other and the physical area.

Reischl, & Gee, 2007; Steptoe & Feldman, 2001; Wen, et al., 2005; Wright, Bobashev & Folson, 2007). These harmful effects persist after controlling for individual level factors (i.e. socioeconomic status, education), which suggests that the social and physical environment is a crucial point of intervention (Morenoff, 2003).

Youth in particular are at risk for stressor exposure (Cicchetti & Rogosch, 2002; Deardorff, Gonzales, & Sandler, 2003; Leventhal, 2003), as their family, peer, and school context are often tied to their neighborhood context. Youth who live in worse neighborhoods are at increased risk for physical abuse, family conflict, attending ineffective schools, environmental hazards, and exposure to community violence (Landis, 2007), which puts them at risk for experiencing more acute, chronic and traumatic stressors. The intersection of multiple contexts may thus impose a greater effect on young people than on adults who are less constrained to the home environment.

One avenue through which neighborhoods may influence health is through a stress pathway. Living in a poor and segregated neighborhood may increase the potential for greater exposure to chronic and acute stressors, traumatic events and daily hassles (Latkin & Curry, 2003; Ross, Reynolds, & Geis, 2000; Steptoe & Feldman, 2001). Residents living in neighborhoods with high rates of poverty and unemployment, or disadvantaged neighborhoods[2], may experience greater exposure to stressful circumstances. Neighborhood disadvantage is often a direct outcome of segregation and concentrated poverty, which contributes to physical deterioration of neighborhoods, crime, violence, vandalism, and fear (Ewart, 2002; Latkin & Curry, 2003; Steptoe, 2001).

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[2] Neighborhood disadvantage is commonly used as a measure of the objective socio-economic condition of an area, and typically includes factors like racial composition, the percent of residents living in poverty, percent unemployment and the percent of single-parent families (Latkin & Curry, 2003; Yen & Kaplan, 1999).

These socially disordered neighborhoods lack elements of social control and cohesion,[3] and have higher rates of crime and violence (Perkins & Taylor, 1996; Ross & Mirowsky, 1999; Sampson, Raudenbush, & Earls, 1997). Symptoms of neighborhood disadvantage may contribute to the chronic stress and strain that threatens the health of residents living in poor neighborhoods. Additionally, neighborhoods fraught with physical, social, and economic decay, and crime, may undermine neighborhood, family and peer social support systems, as well as the social and economic resources that help buffer residents against the noxious stressors of disadvantage (Latkin & Curry, 2003).

Crime and violence are stressors that affect neighborhoods directly, via physical and psychological harm to the victims, and indirectly, via avenues of fear and mistrust (Wen, et al., 2005). Like other indicators of social disorder, crime and violence are direct products of concentrated poverty (via racism and segregation). Communities with high rates of crime often experience a spiral of disinvestment from individuals and businesses; abandoned buildings, fires, deterioration; and feelings of resentment, hopelessness and alienation; all of which may increase the stress of residents (Blau & Blau, 1982; Massey & Denton, 1993; Ross, Mirowsky, & Pribesh, 2001).

Disadvantaged neighborhoods, however, are not necessarily devoid of resources. Neighborhoods high in poverty may also exhibit social control and social cohesion (Burgard & Lee-Rife, 2009; Ungar & Wandersman, 1985), and may have resources available (particularly for youth) (Wandersman & Nation, 1998) to offset the negative effects of disadvantage (e.g., youth centers and programs). In addition, churches in disadvantaged neighborhoods may offer residents a sense of community and provide

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[3] The combination of social control and social cohesion is referred to as collective efficacy, which will be discussed in a following section.

social support to buffer them against the detrimental effects of poverty. Despite the

potential for disadvantaged neighborhoods to have resources for residents, in

communities where social disorder – particularly violence and crime – is present,

residents may not utilize these resources. Researchers have found, for instance, that

people living in high crime neighborhoods tend to stay inside more often, have fewer

social ties and higher levels of social isolation, and more fear and anxiety (Wright,

Bursik, 1988; Perkins & Taylor, 1996; 2006). Therefore, it may be the utilization of

community resources, and not just the presence of them that matters most to the health of

residents.

The Transactional Model of Stress and Coping is applied in the conceptual model

and helps clarify the process by which neighborhood stressors may affect psychological

and physiological stress outcomes and coping efforts. The conceptualization of the stress

process as a transactional process between individuals and their environment (Folkman,

1984; Wenzel, Glanz, & Lerman, 2002) is particularly relevant to the understanding of

population inequalities in psychological and physiological stress, which are rooted in

context. A more detailed discussion of the application of the Transactional Model of

Stress and Coping is left to the individual papers. In general, however, the Model of

Stress and Coping directs our understanding about how people react to stressful stimuli

based on their evaluation of the susceptibility, severity, controllability and personal

relevance of the stressor (Lazarus & Folkman, 1984; Wenzel, et al., 2002). The model

also suggests that the coping response depends on the severity and risk of the stressor

(Wenzel, et al., 2002). These reactions and responses are shaped by an individual's

access to intrapersonal (e.g., self-efficacy) and interpersonal (e.g., social support) coping

resources (Folkman, 1984). The research in this dissertation will include an analysis of important coping and social support factors as outlined by the Model of Stress and Coping.

## Rationale

Few researchers have looked at stress (either psychological/perceived or physiological) as an endpoint. This research will explore the ways in which the social, economic, and physical environment differentially influence the prevalence of psychological and physiological stress in Black versus White Americans. Although this research will examine both psychological and physiological stress, I hypothesize that measures of physiological stress will more accurately reflect chronic exposure to neighborhood-level stressors (which also capture more broad structural stressors of institutionalized racism and segregation; and low wealth and poverty) than psychological stress measures. Psychological stress, while a valid assessment of a person's current psychological state, may not capture an accumulation of exposure to stressful experiences over the lifecourse. To better address this theoretical and analytical issue in this research, the first and third papers in my dissertation will use a broadly accepted measure of perceived stress (Cohen and colleague's daily hassles scale) (Cohen, Kamark, & Mermelstein, 1983), and the second paper will examine cortisol, a physiological indicator of *wear and tear*, often used as a stress biomarker.

## Roadmap of the dissertation

Each paper in this dissertation will address a portion of the conceptual model represented in Figure 1.1. Some pathways are addressed in more than one paper; due to data limitations, the entire model could not be examined in one study. The following

section presents an overview of the goals, data, methods and unique contributions of each paper.

**Paper 1**

Adolescents who grow up in disadvantaged neighborhoods are constantly exposed to economic, social and environmental stressors (Allison, 1999; Deardorff, et al., 2003). Disadvantaged neighborhoods are characterized by high rates of unemployment, poverty, and single-parent households, which often lead to socially disordered neighborhoods and a greater experience of daily stressors, major life events and traumatic events (Landis, 2007). Several researchers have investigated the relationship between neighborhood disadvantage and mental health, behavior (e.g., delinquency and substance use), and physical health (Dubow, Edwards, & Ippolito, 1997; Harding, 2003; Meyers, 2004; Seidman et al., 1998). Few studies use longitudinal designs, however, which make it difficult to establish causality between stressor exposure and psychological stress. In addition, most researchers assume a stress mechanism to explain significant relationships between living in a disadvantaged neighborhood and the health outcome; researchers, however, have not directly addressed the connection between neighborhood stressors and psychological stress, while considering the mediating and moderating effect of intra- and interpersonal risk and promotive factors (Dubow, et al., 1997; Gutman, 2005; Seidman, et al., 1998).

The overall aim of the first paper, presented in chapter 2, is to examine the relationship between neighborhood disadvantage and perceived (psychological) stress in a low-income African American sample, using longitudinal data and a multi-level analysis. Several mediating and moderating factors including high effort coping,

substance use, social support, and neighborhood perceptions are also examined (Figure 2.1), to investigate the role of intra- and interpersonal risk and promotive factors on the perceived stress trajectory, as well as interactions between neighborhood disadvantage and these factors. To test my models, data from the Flint Adolescent Study (FAS), a longitudinal study of youth development and the transition to adulthood are employed. The study began with 850 youth entering high school in 1994; these youth are now young adults in their late twenties and early thirties, and many have young children who are also included in the more recent waves of data collection. The current study focuses on 665 self-identified Black or African American youth who were enrolled in the second wave of the study (1995). I extend my analysis through Wave 8 (2002), and I conduct a multi-level growth curve model to examine how neighborhood stressor exposure (operationalized as neighborhood disadvantage) shapes initial stress levels as well as the change in stress over time, accounting for individual-level control variables and risk and promotive factors.

Results from this research will help better understand the relationship between neighborhood disadvantage and psychological stress in an at-risk sample of African American youth, as well as the role of risk and promotive factors and their contribution to perceived stress. The FAS data are ideal for testing this model because I am able to examine changes in stress, substance use, social support, high effort coping (John Henryism), and neighborhood perceptions over time during early adolescence and the transition to adulthood, which represent two important periods in an individual's development.

**Paper 2**

Evidence suggests that the consequences of chronic exposure to stressors extend beyond psychological effects (Jackson, Knight, & Rafferty, 2009; McEwen & Seeman, 1999). Researchers believe that an accumulation of stressor exposure wears down both the psychological and physical systems in the body, resulting in hyper-activation of the stress response and allostatic load. Despite the dearth of research in humans, allostatic load is associated with cardiovascular health, cognitive dysfunction, neuronal atrophy, immunosuppressive effects, insulin resistance and increased risk of autoimmune disorders (McEwen & Seeman, 1999). Paper 2 (Chapter 3) will address the relationship between exposure to stressors at the neighborhood level and salivary cortisol, which is a physiological biomarker of stress included in measures of allostatic load, and a critical intermediary between stress and health (McEwen & Seeman, 1999).

Paper two examines a subset of youth in the FAS who consented to saliva sampling during the sixth wave of data collection. The overall aim of the second paper is to examine the relationship between the same neighborhood stressors considered in Paper 1, and physiological stress, measured by cortisol reactivity. I also examine moderating pathways including individual-level risk and promotive factors that are associated with neighborhood stressor exposures and may be related to cortisol (Figure 3.2). Data from the FAS (details described above) will be used to examine these study aims, in addition to neighborhood disadvantage indicators from the 2000 U.S. Census. A cross-sectional, multi-level analysis using the sixth wave of FAS data is conducted to test my hypotheses. This paper will help abate some of the confusion in the literature surrounding the importance of psychological versus physiological stress measures, as well as the relationship between neighborhood exposures and risk and promotive factors on cortisol.

**Paper 3**

Despite the growing evidence in support of the association between neighborhood exposures and health, much of the research does not include a thorough test of the processes through which neighborhood stressors affect health. Individuals who live in neighborhoods with more socio-economic disadvantage, crime, violence, social isolation, and less social support and social cohesion in their neighborhoods, experience worse health outcomes (Diez Roux, 2001; Diez Roux, 2003; Diez Roux & Mair, 2010; Diez Roux, 1997; Pickett & Pearl, 2001; Yen & Kaplan, 1999). Yet, we are not clear exactly how neighborhood context influences these health outcomes. Many researchers propose that neighborhoods affect health through a stress mechanism (Elliott, 2000), which includes exposure to stressors, stressor appraisal, and mediators and moderators of the stress process (Pearlin, et al., 1981), but few researchers specifically examine stress in their models.

Paper 3 (Chapter 4) will extend research on the stress process at the neighborhood level, to examine the pathways through which neighborhood socio-economic disadvantage influences self-rated health. I test a structural equation model through which I examine mediating factors at both the neighborhood and individual level that connect neighborhood disadvantage to health (Figure 4.1). At the neighborhood level I examine the relationship between neighborhood disadvantage and psychological stress, including crime and safety, social capital, and neighborhood attitudes as mediating pathways. I also test perceived stress as a mediator of the relationship between neighborhood stressor exposure and global self-rated health.

To test my model and hypotheses I use data from multiple sources including the FAS, the *Speak to Your Health!* Community Survey, Flint police crime data, and 2000 U.S. census data. By using several sources of data I am able to combine objective and subjective neighborhood measures for the neighborhoods in which FAS participants live, while avoiding biases that may result from using subjective neighborhood ratings from the same participants who self-reported behavioral, psychological and health information (same source bias). I test cross-sectional structural equation models using data from 2009 (and violent crime data averaged over five years between 2004 and 2008) to specifically consider psychological stress as a critical intermediary in the relationship between neighborhood context and health.

## Summary

This dissertation will convey a clearer and more nuanced understanding of the way neighborhood context affects individuals' perceptions of stress and cortisol reactivity, which will offer some insight into the contribution of stress to racial health inequalities. While many researchers have examined neighborhood influences on health, proposing that they function through a stress mechanism, few researchers have specifically examined the stress mechanism, or grounded their research in theory to better understand the relationships between neighborhood stressor exposure, intra- and interpersonal risk and promotive factors, and stress. To address these gaps and better connect the sociological, psychological, health, and physiological literatures, in this dissertation I will focus on the stress mechanism to connect neighborhood context and stress.
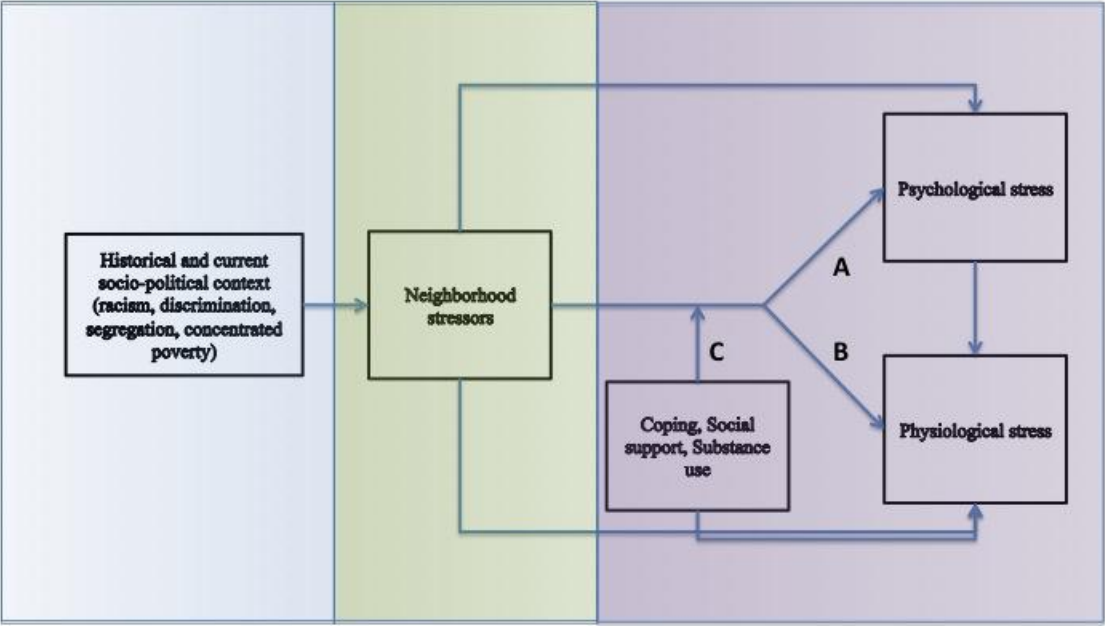
**Figure 1.1 - Conceptual model for the dissertation**

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**CHAPTER 2**

**NEIGHBORHOOD CONTEXT AND PERCEPTIONS OF STRESS OVER TIME: AN ECOLOGICAL MODEL OF NEIGHBORHOOD STRESSORS AND INTRAPERSONAL AND INTERPERSONAL RESOURCES AND COPING**

**Introduction**

Health status is patterned by social factors including race, sex, education and social class (Geronimus & Thompson, 2004; Jackson, Knight & Rafferty, 2009; Williams, Yu, Jackson & Anderson, 1997). Compared to White Americans, Black Americans have fewer individual economic advantages and live in poorer; more segregated; and more socially and environmentally disadvantaged neighborhoods (Williams, 1997; Williams & Collins, 2001). Many researchers attribute the fact that Black Americans experience higher rates of mortality, morbidity, chronic and infectious disease, and poorer birth outcomes than their White counterparts to these economic, social, and environmental inequalities (Geronimus, 1996; Link & Phelan, 1996; Mays, Cochran, & Barnes, 2007; Morello-Frosch & Shenassa, 2006).

Despite social and economic inequalities between Black and White Americans, and the widely documented health disparities in the United States, researchers continue to debate the causal pathway connecting these factors. Stress has been suggested as a potential cause of Black-White health inequalities, and a mechanism that may link social and economic inequalities to health

(Aneshensel, Rutter & Lachenbruch, 1991; George & Lynch, 2003; Myers, 2009; Pearlin, Schieman, Fazio & Meersman, 2005; Schulz et al., 2000). Black Americans are exposed to more hassles, chronic, acute and traumatic life stressors than Whites, and experience greater psychological stress across the life course (George & Lynch, 2003; Geronimus, Hicken, Keene, & Bound, 2006; Jackson, Knight, Carstensen & Schaie, 2006; Turner & Lloyd, 1995).

**Stress**

The meaning and measurement of *stress* is complex. The stress response may be reflected in single cells or organs; in biochemical, physiological and emotional functioning; in entire body systems like the endocrine, immunologic and cardiovascular systems; or in the incidence of physical or psychological distress (Pearlin, Menaghan, Lieberman & Mullan, 1981). For the purposes of this research, I will refer to *stress* as the psychological outcome and *stressor* as the stimulus of the outcome (e.g., poverty). Measures of acute life events, for instance, assess the stressful stimuli (stressor) and not the outcome (stress). In this case, it is impossible to determine whether a health outcome is truly caused by exposure to the stressful experience, or due to psychological or physical mediators of the exposure. These mediators may be the key to understanding a causal pathway between lifetime exposure to stressors and Black-White health inequalities.

Much of the literature on stress and health examines the relationship between exposure to stress and distress (Cutrona, Wallace, & Wesner, 2006; Elliott, 2000; Evans & English, 2002; Hammack, 2003), but few researchers examine perceived stress as an endpoint (or even test a mediating model). Perceived stress is a more subjective endpoint

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than a measure of depression or a physiological biomarker of stress, but understanding trends in this psychological state over time (or the trajectory of perceived stress) will improve our understanding of the relationships between exposure to stressors and psychological states; and psychological states and physical and mental health endpoints (Schmeelk-Cone & Zimmerman, 2003). Schmeelk-Cone & Zimmerman (2003) found, for instance, that adolescents who experience low levels of perceived stress over time exhibit less anxiety and depression at the end of the time period. Examining stress as an endpoint is also crucial in light of the non-specific, multiple pathways that link stress to physical and mental health (Aneshensel et al., 1991). Finally, some of the null and inconsistent findings for the relationship between exposures (e.g., neighborhood disadvantage) and health outcomes may be due to the long lag time between exposure to stressors and the manifestation of health outcomes, as well as a lack of sufficient longitudinal data that includes information on lifetime stressor exposure and health (Ellen, Mijanovich, & Dillman, 2001). Using perceived stress as an intermediary endpoint enables researchers to examine more immediate relationships between stressor exposure and mental health.

The perceived stress scale (Cohen et al., 1983) is a global measure of perceived stress, which assesses the degree to which a person's life feels stressful, and whether an individual feels in control of his or her life. Although a measure of perceived stress is likely to reflect current feelings of stress instead of cumulative life stress, the perceived stress scale avoids confounding of stressor exposure and stress. Many researchers include life event scales, or other checklists of acute stressors as their measure of stress (versus stressor exposure); this fails to differentiate the exposure (e.g., death of a loved

one) from the psychological reaction to the exposure (e.g., feeling out of control in one's life), and may contribute to the mixed results in the literature (Stancil, Hertz-Picciotto, Schramm, & Watt-Morse, 2000). Although it seems reasonable that an individual who lost a loved one would experience psychological stress due to the stressor, exposure to a stressor does not necessarily lead to stress. The experience of stress is mediated or moderated by interpersonal (e.g., social support) and intrapersonal (e.g., high effort coping) resources, as well as shaped by social, economic and structural context (Pearlin, 1989). Research on stress indicates that it is necessary to study the exposure (stressor), mediating and moderating processes and the stress outcome.

Several factors are likely to contribute to the unequal distribution of stressor exposure in the population, but differences in neighborhood context may capture multiple sources of stressor exposure as well as historical and structural contributors to stress. This paper, therefore, will examine the ways in which the neighborhood context influences an individual's perceived stress trajectory from mid-adolescence through early adulthood. I draw from the Transactional Theory of Stress and Coping (Folkman, 1984; Wenzel, Glanz, & Lerman, 2002), neighborhood structural and stress models (Aneshensel, 2008), and a larger ecological framework (Bronfenbrenner, 1979) to develop hypotheses and guide the multilevel analysis.

**Stress and coping theories**

The Transactional Model of Stress and Coping describes the process by which stressors affect psychological stress outcomes, social resources and coping efforts. Early work on stress and health focused on stress as an isolated physiological response to an external or internal stimuli appraised as stressful (Wenzel, et al., 2002). More recent

theories hypothesize a transactional process (Folkman, 1984; Wenzel, et al., 2002) where stress is explained by the relationship between a person and his/her environment. This conceptualization of the stress process is particularly relevant to the understanding of population inequalities in psychological and physiological stress, which are rooted in contexts such as the neighborhood.

The Transactional Model of Stress and Coping includes four components, which describe appraisal of the stressor and the mobilization of coping processes. During primary appraisal individuals initially evaluate whether the stressor is threatening based on susceptibility, severity, controllability and personal relevance, and then initiate a coping response depending on the severity and risk of the stressor (Lazarus & Folkman, 1984; Wenzel, et al., 2002). Concurrently, individuals consider and assess their options for alleviating stress based on their perceptions of control and their available coping resources; this step is referred to as the secondary appraisal stage (Folkman, 1984; Lazarus & Folkman, 1984; Wenzel, et al., 2002). Coping resources include both intrapersonal (e.g., self-efficacy; high effort coping) and interpersonal (e.g., social support) resources, and the coping process mediates or moderates the relationship between the stressor (exposure) and health outcome (Folkman, 1984; Wenzel, et al., 2002).

Coping efforts can be active or passive. Active coping, for example, may include efforts to modify a stressful situation (Pearlin & Schooler, 1978), and is most likely used when an individual determines that the stressor is controllable or that they feel capable to tackle the stressor. These strategies typically increase positivism and self-control (Lazarus & Folkman, 1984; Wenzel, et al., 2002). One active coping strategy is using

social support.  The role of social support as a means to alleviate and control stress is widely documented in the literature (Ensel & Lin, 1991; Lyons, Mickelson, Sullivan, & Coyne, 1998; Thoits, 1995).  Pearlin, Menaghan, Lieberman and Mullan (1981) define social support as "access to and use of individuals, groups, or organizations in dealing with life's vicissitudes (p. 340)."  Social support from family and friends may diminish the detrimental effects of living in a highly disadvantaged neighborhood, as suggested by the literature and models of stress and coping (Lazarus & Folkman, 1984; Thoits, 1995).  This research will examine perceived social support from family, peers and significant others.  Although some researchers suggest that the use of, and not just the perception of social support is important, most find that the perception of support is more strongly related to mental health outcomes (Thoits, 1995).  I hypothesize, therefore, that individuals who report sources of social support will be better able to cope with stressors.

Another active coping strategy that will be examined in this research is John Henryim.  James (1994) presents the notion of John Henryism as a metaphor for high effort coping, which is a prolonged coping process in response to extreme psychosocial environmental stressors and barriers to success (James, 1994; Sellers & Neighbors, 2008). The John Henryism hypothesis suggests that Black Americans are exposed to chronic stressors (i.e. financial strain, racism, unemployment), which require excessive amounts of active coping to endure.  The extended coping efforts, therefore, are hypothesized to slowly break down the body, both mentally and physically (James, 1994).  John Henryism particularly applies to Blacks who are exposed to highly salient and personally relevant stressors (Sellers & Neighbors, 2008).  Individuals who engage in high effort coping believe that they can control stressors by hard work and determination;

this active style of coping may decrease current perceptions of stress, but may also result in physical harm and wear on the body later in adulthood or old age.

Conversely, avoidant coping strategies (a type of passive coping), which are often used to combat highly uncontrollable stressors, can increase psychological distress and the probability of engaging in harmful health behaviors to alleviate stress (Jackson, et al., 2006; Wenzel, et al., 2002). Substance use may operate as an avoidant coping method and may moderate the relationship between living in a stressful environment and psychological stress (Jackson, et al. 2009; Lambert, Brown, Phillips, & Ialongo, 2004; Schmeelk-Cone & Zimmerman, 2003). Jackson et al. (2009) hypothesize that when people are confronted with chronic and uncontrollable stressors, such as those reflected by measures of neighborhood disadvantage, they may engage in unhealthy behaviors like substance use to achieve an immediate relief of the psychological strain (Jackson, et al. 2009). Unlike more active coping methods that are used to alleviate the psychological strain associated with stressor exposure (Wenzel, et al., 2002), substance use may actually intensify the detrimental effects of the exposure (Jackson, et al. 2009).

Residents living in highly stressful, disadvantaged neighborhoods may have less access to coping resources such as tangible (e.g., economic resources), intrapersonal resources (e.g., self-efficacy) and interpersonal resources (e.g., support system) (Myers, 2009). In the case of neighborhood disadvantage as a source of chronic stressor exposure caused by complex and distal factors, it is likely that individuals will evaluate stressors as uncontrollable and rely more heavily on avoidant coping strategies like substance use, which may contribute to or accentuate their perceptions of stress and result in negative behavioral and health outcomes. I examine three unique coping methods in this study:

John Henryism, or high effort coping, social support, and substance use. Each of these coping strategies, supported by stress and coping theories, may play an important role in shaping an individual's perception of stress, and moderating the pathway between neighborhood stressor exposure and stress.

**Socio-ecological framework**

I apply the socio-ecological framework to explain how interactions across multiple environments may affect an individual's perception of stress. Specifically I examine the ways in which neighborhood stressors may interact with an individual's intra- and interpersonal resources (e.g., coping, social support and substance use) as a part of the stress and coping process, to affect their perceived stress over time. The socio-ecological framework includes several levels of influence including the intrapersonal, interpersonal, organizational, community and environmental/policy level, and describes how these levels interact to affect health (Glanz, Rimer, & Lewis, 2002). Although this study does not specifically address the environmental/policy level, structural factors embedded in society (e.g., institutionalized racism in education and jobs leading to economic inequalities; discriminatory lending practices in banking and in real estate, leading to segregation) shape the residential choices of the individuals living in the neighborhoods I examine.

The socio-ecological framework suggests that both intra- and interpersonal aspects of peoples' lives are affected by their neighborhood (organizational/community level factor). Although many researchers studying neighborhood effects on health include individual-level control factors, few actually test the moderating role of contextual and individual-level factors (Latkin, German, Hua & Curry, 2009). This

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research applies an ecological framework and examines the ways in which individual-level intra- and interpersonal factors are shaped by the larger neighborhood environment to affect individuals' psychological stress.

**Neighborhoods and Stress**

Research on the importance of residential context is not new. Sociologists have examined the detrimental effects of residential segregation and urban disorder on health and well-being for decades (Massey & Denton, 1989; Yinger, 1995). Research on neighborhood effects seems to have resurfaced with new vigor over the past five years, (Diez Roux, 2003; Diez Roux & Mair, 2010), a trend that can be partially attributed to an increasing domestic and international focus on health inequalities and the social determinants of health. Evidence clearly indicates an economic and racial patterning in place of residence, which may contribute to widening health inequalities (Diez Roux & Mair, 2010; LaVeist, Pollack, Thorpe, Fesahazion, & Gaskin, 2011; Morenoff et al., 2007). In addition, individual-level models of health do not adequately explain disease etiology or reasons for the large racial and ethnic health gap (Diez Roux & Mair, 2010), which may be further explained by contextual effects.

Neighborhood socioeconomic characteristics and social processes have been linked to several indicators of health including cardiovascular disease, mortality, morbidity, chronic diseases, psychological health, and health behaviors, even after accounting for individual-level socioeconomic status (Diez Roux, 2003; Pickett & Pearl, 2001; Williams, et al. 1997; Yen & Kaplan, 1999). The association between neighborhood factors and psychological stress, however, has not been as thoroughly examined, despite an abundance of literature indicating that living in poor and segregated

neighborhoods exposes residents to more stress (Dupéré, 2007; Ellen, et al., 2001; M. Elliott, 2000; Kruger, Reischl, & Gee, 2007; Latkin & Curry, 2003), and a separate literature that connects stress to various negative health outcomes (Herbert & Cohen, 1993; Wright, Rodriguez, & Cohen, 1998).

Black Americans, who are on average poorer and have less wealth than their White counterparts, are also more likely to live in poor and segregated neighborhoods (Massey & Denton, 1993; Merkin et al., 2009). These neighborhoods are often characterized by economic and social disadvantage, and may lack many of the resources necessary to counter these stressors (e.g., strong social networks, economic resources, recreation) (Burgard & Lee-Rife, 2009; Latkin & Curry, 2003; Wen, Cagney, & Christakis, 2005). Several conceptual frameworks have been discussed in the literature to explain the relationship between neighborhoods and stress including structural models, environmental stress models, and models of social disorder or disorganization (Latkin & Curry, Browning & Cagney, 2003; Dupéré, 2007; Kruger, et al., 2007; 2003; Wen, et al., 2005; Yen & Kaplan, 1999). The structural and social disorder models are most relevant for the present study.

Structural Model. Structural neighborhood models characterize the neighborhood as a cluster of individual characteristics. These models are often tested using multi-level modeling techniques, which examine the association between socioeconomic indicators of the neighborhood (e.g., poverty, unemployment, racial composition) and health via social, cultural and psychological mediating processes (Aneshensel, 2008; Wandersman & Nation, 1998). Although structural models generally include census-based aggregates of individual-level variables, these indicators are not simply summary measures of

individual characteristics. Contextual effects are greater than the sum of their parts

because they include person-place interactions, as well as complex social interaction

(Aneshensel, 2008; Yen & Kaplan, 1999).

      <u>Social Disorder Model</u>. Social disorder models of neighborhoods connect

structural and economic characteristics of a neighborhood (neighborhood disadvantage)

to health via social pathways (Aneshensel, 2008). Mirowsky (1999) and Ross & Jang

(2000) characterize social disorder as visible signs of lack of order and social control in

an area. These signs may include deviance and crime, graffiti, noise, drug use,

vandalism, litter and the presence of abandoned buildings (Latkin & Curry, 2003; Ross &

Jang, 2000; Ross & Mirowsky, 1999). Disordered neighborhoods may have direct and

indirect effects on an individual's experience of stress. Neighborhoods fraught with

crime and violence, for instance, may directly evoke stress for residents who witness

these acts, or who live in fear of the crime in their communities (Diez Roux & Mair,

2010; Kruger, et al., 2007). Socially disordered neighborhoods may indirectly influence

stress by reinforcing unhealthy social norms (e.g., drug use), by exposing residents to

poor role models, and may lack the resources necessary to buffer residents from harmful

effects of disorder (Latkin & Curry, Burgard & Lee-Rife, 2009; Diez Roux & Mair,

2010; Dupéré, 2007; 2003).

      Although neighborhood disadvantage is criticized as a measure of neighborhood

context (Culhane & Elo, 2005; Morenoff, 2003; Yen & Kaplan, 1999) for its focus on

aggregated characteristics and neglect of social processes, neighborhood disadvantage is

a precursor of social disorder. A neighborhood with high levels of economic and social

disadvantage is not necessarily socially disordered, but researchers have found that

disadvantage and disorder are empirically and theoretically related (Aneshensel, 2008; Ross, Mirowsky, & Pribesh, 2001). Aneshensel (2008) characterizes neighborhood disorder as a secondary stressor that mediates the relationship between disadvantage (the primary stressor) and health.

Neighborhood disadvantage is a direct product of larger social and economic structures that have resulted in spatially, socially and economically isolated urban neighborhoods (Wilson, 1987; Yinger, 1995). Although researchers make distinctions between the different neighborhood models, I focus on neighborhood disadvantage for several reasons. First, neighborhood disadvantage is a precursor to social disorder, and social disorder is a direct result of factors like poverty and residential instability, which limit access to resources, and weaken support structures and communication (Aneshensel, 2008; Browning & Cagney, 2003). Neighborhood disadvantage may also precede environmental stress, as advantaged communities are less likely than disadvantaged ones to be situated in areas near environmental stressors like power plants, highways and bridges, and away from parks and safe space (Bullard, Mohai, Saha, & Wright, 2007).

In addition, neighborhood disadvantage is a concept that captures the accumulation of multiple neighborhood stressors across the lifecourse (e.g., exposure to disorder and environmental stressors; cumulative economic stressors), as well as the larger structures of racism and segregation that result in chronic stressor exposure for many Black Americans (Williams & Collins, 2001). As described in the Transactional Model of Stress and Coping, these stressors are likely appraised as highly uncontrollable and personally relevant, as they are institutionalized in American society and reinforced

daily, and thus shape the lives of Black Americans living in highly segregated, urban areas.

Finally, one of the common critiques of the neighborhood literature is the potential for selection bias (Diez Roux, 2001; Diez Roux & Mair, 2010; Ellen, et al., 2001; Wheaton & Clarke, 2003). Researchers argue that it is difficult to rule out reverse causation, where people chose their neighborhoods based on individual characteristics. I argue, however, that although this is most certainly true, several of the predominant characteristics that influence this decision for people living in disadvantaged neighborhoods like socioeconomic status and race, are partially accounted for by indicators of neighborhood disadvantage.

I also include subjective measures of neighborhood context, measured at the individual level, which may related to a neighborhood's level of disadvantage or may assess different aspects of the neighborhood: neighborhood attitudes, fear of neighborhood violence, and worry about being physically hurt in the neighborhood. Residents who feel more favorably towards their neighbors and neighborhood, and who are not afraid of violence in their neighborhoods, may experience lower levels of perceived stress than residents living in highly disordered and violent neighborhoods (Diez Roux & Mair, 2010; Ross & Jang, 2000; Ross, et al., 2001). In addition, residents who live in highly disadvantaged neighborhoods may not perceive their neighborhood as disadvantaged, and thus may better characterize residents' perceptions and subsequent stress.

**Study Objectives**

Researchers recommend that future work in the area of neighborhoods and health include an analysis of the processes through which behavioral, psychological and social processes interact to shape health across different neighborhoods (Aneshensel, 2008; Diez Roux & Mair, 2010). This study addresses this gap and examines the relationship between several intra- and interpersonal factors and perceived stress, as guided by the Transactional Model of Stress and Coping. This study also tests cross-level interactions between these factors and neighborhood disadvantage stressors, which situates the neighborhood stress process in a larger ecological framework (Figure 2.1).

Specifically, I examine the change in perceived stress during middle adolescence and early adulthood. In addition, I investigate the effect of intra- and interpersonal coping resources including high effort coping, social support and substance use, on perceived stress during young adulthood. I also examine the effect of neighborhood attitudes and perceptions on perceived stress over time. Finally, I consider the moderating effect of neighborhood disadvantage on the stress trajectory and on the coping factors and perceived stress over time. Although neighborhood stressors are hypothesized to have direct effects on individuals' stress, they may also interact with coping and the availability of social support in order to jointly influence an individual's perceptions of stress across early adulthood.

## Methods

### Design and Sample

Data from the Flint Adolescent Study (FAS), a longitudinal study of 850 youth at risk for substance use and school dropout were used for this study. Eligible students

included ninth graders enrolled in one of four public high schools in an urban city in Michigan, and who had an 8th grade grade point average (GPA) of 3.0 or below upon entering high school. Youth who were diagnosed by the schools with emotional or developmental impairments were excluded from the study sample. Youth self-identified as African American (80%), White (17%) or Bi-racial (3%). Males and females were equally represented in the sample.

The FAS consists of four waves of data collected during the high school years (Waves 1-4; 1994-1997), four waves of data collected after high school and during young adulthood (Waves 5-8; 1999-2002), and two waves of data collected in young adulthood (Waves 9-10; 2008-2009). Retention rates were generally high (90% from Waves 1 to 4 and 75% from Waves 4 to 8). Data were collected during structured face-to-face interviews conducted either at school or at alternative community locations (University of Michigan Institutional Review Board approval, UMIRB# H03-0001309).

The interview portion of the study included most of the psychosocial variables in the data, as well as information on employment, education, family and friends. A self-administered questionnaire assessed more sensitive information (e.g. substance use, sexual risk behavior, discrimination) and was distributed at the conclusion of each interview to facilitate confidentiality. The individual-level data collected in Waves 1 through 4 (1994-1997) was linked to 1990 census data based on geo-coded home address information, and individual-level data from Waves 5 through 8 (2000-2003) was linked to 2000 census data using the same methods.

The current study uses data from Waves 2 through 8 of the study. I exclude the first year of data collection because the dependent variable examined in this paper was

not included in the first wave.  The analysis focuses on a sub-sample of 665 Black youth

who had at least one wave of non-missing data for the dependent variable, as well as

corresponding census block group data in the 2000 Census.  On average, youth were 15.9

years old (*SD=0.64)* in Wave 2 and 23 years old (*SD* =0.68) in Wave 8, and over half

(51%) of the sample were female.

**Measures**

In this research I focus on neighborhood measures of disadvantage (census

based), substance use, social support, high effort coping (John Henryism), neighborhood

perceptions, and perceived stress, while controlling for sex and mother's socioeconomic

status (occupation and education).  The measures are described below, and descriptive

data for the sample by wave are displayed in Table 2.1.

**Individual-level measures**

Dependent Variable: Perceived stress

Perceived (or psychological) stress was assessed using Cohen and colleagues'

Perceived Stress Scale (PSS) (Cohen et al., 1983), which was originally a 14-item scale

designed to measure subjective (psychological) stress.  The scale assesses the degree to

which people believe their lives to be "unpredictable, uncontrollable, and overloading"

(p. 387) (Cohen, et al., 1983).  In the FAS, however, the Perceived Stress Scale was

shortened to 11 items based on a principal components analysis in order to reduce the

length of the measure and retain the best psychometric properties.  Sample items include,

"In the past month, how often have you…": been upset because of something that

happened that you didn't expect, felt that you had so many problems that you could not

deal with them, and felt able to handle your personal problems (positive items were

reverse coded). The items were averaged and used as a continuous measure ranging from 1=low to 5=high perceived stress ($\alpha$ = 0.71 to 0.83).

Demographic factors

Several demographic factors are included in the analysis as control variables. Youth's *sex* was coded as 0=female and 1=male. Although the participants range from adolescents to young adults in this study, and many of them have families of their own by Wave 8, I use the family's SES from Wave 1 in analyses. In a study of cumulative exposure to stressors, early life circumstances may be more important predictors of psychological stress than current SES and the participant's education. *Family socioeconomic status* (SES) was assigned based on the highest occupational prestige score for either parent, using codes developed by the National Opinion Research Center and then standardized to facilitate interpretation (Nakao & Treas, 1990). The score was assigned based on 20 occupational classifications, ranging from private household work (scored as 29.28) to professional (scored 64.38). The mean prestige score in our sample is 39.82 (*SD* =9.80) which corresponds to a blue-collar occupation (Nakao & Treas, 1990). *Maternal education* was used as an additional measure of SES status. Mother's highest education level was assessed using a seven-level interval response (1=Grade school or less to 7=Graduate/professional school), and was used as a continuous variable in the analysis. The mean education level in our sample is 4.40 (*SD*=1.88), which corresponds roughly to an average vocational/training school education.

Substance Use

Substance use is a composite measure that includes items about smoking (number of cigarettes smoked in the past 30 days), alcohol use (composite measure of past 30 day

alcohol use, binge drinking over the past two weeks and drinking to get high), and marijuana use (past 30 day marijuana use). Each item or scale was standardized (mean=0, SD=1.00), and the average of the three standardized variables was computed to create a substance use scale ($\alpha = 0.62$ to $0.81$). Higher values denote more substance use.

High effort coping

High effort coping or John Henryism is a measure that was originally conceptualized to assess high effort coping, which is a prolonged coping process in response to psychosocial environmental stressors and barriers to success (James, 1994). High effort coping was measured using an eight-item scale that asks respondents to rate items like: "Hard work is the best possible way for someone to get ahead in life," and "sometimes I feel that if anything is going to be done right, I have to do it myself." These items are rated on a 5-point Likert scale (1=Not true to 5=Very true) and then averaged to create a measure of high effort coping ranging from 1=low coping to 5=high coping ($\alpha = 0.70$ to $.84$ across waves).

Social Support

Three sources of perceived social support were assessed in this study. Procidano and Heller's perceived support scales were adapted slightly and used in the FAS questionnaire (Procidano & Heller, 1983). Mother's support was measured by five items using a 5-point Likert Scale. Items include the degree to which the adolescent's mother gives emotional and instrumental support, and the closeness of the mother-child relationship. The support scale ranges from 1=low to 5=high support ($\alpha = 0.88$ to $0.93$). Peer support was also included in Waves 2-8, and includes items assessing emotional and instrumental support, which are averaged to create a composite measure. Like the

measure of mother support, the scale ranges from 1 to 5 ($\alpha$ = 0.87 to 0.94). Finally, the questionnaire asks the respondent to identify a person in their lives to whom they feel closest. This person is likely to be a significant other, but could be another family member or friend. These items were added to the questionnaire in Wave 5 and were thus assessed only in Waves 5-8. Relationship support was measured as a mean score of 6-8 items (this scale was changed slightly between waves of the study) and included items such as: "how much the person provides you reassurance and encouragement when needed", "shows that he/she cares about you as a person", and "gives you useful information or advice when you need it." The scale ranges from 1=low to 5=high support ($\alpha$ = 0.82 to 0.86).

Neighborhood perceptions

Three measures of perceptions of the neighborhood were included in this analysis. The first measure, *neighborhood attitudes,* assesses the way people feel about their neighborhood. Sample items include: "I like living in my neighborhood", "If I needed advise about something, I could go to someone in my neighborhood", and "I believe my neighbors would help me in an emergency." Each of the five items was assessed using a Likert Scale (1=Strongly Disagree to 4=Strongly Agree). An average of the five items was computed and the scale ranges from 0=unfavorable to 3=very favorable neighborhood attitudes ($\alpha$ = 0.72 to 0.76). The second neighborhood perception measure asks respondents to rate their *fear of the level of violence in the neighborhood* (1=no fear to 4=high fear). A dichotomous indicator of neighborhood fear was created because of a highly skewed distribution towards reporting no fear of violence. This indicator was based on the distribution around the mean of fear, where 0=low fear (below the mean)

and 1=high fear (above the mean).  Finally, participants reported on the degree to which

they worried that someone in their neighborhood would physically hurt them.  This item

was initially assessed on the same four-point scale as the neighborhood fear item, and a

dichotomous indicator of worry was similarly created to reduce skewness towards

reporting little worry, where 0=low worry and 1=high worry.

**Neighborhood-level measures**

Census data

The neighborhood level *disadvantage* variables were created from 2000 census

data, as this census year is closer to the majority of FAS waves (1995, 1996, 1997, 1999,

2000, 2001, 2002) than the 1990 census data.  Although there were some changes in

levels of block group disadvantage between the 1990 and 2000 Census, I conducted a

sensitivity analysis to determine how many census block groups experienced a significant

change in socio-economic disadvantage.  Only about 5% of the block groups experienced

changes in disadvantage between the two census years that were greater than 1.5 standard

deviations in either direction (e.g., lower or higher disadvantage).  Due to this

considerably small number of changes, which is not expected to change my results

significantly, I use the 2000 census data in this analysis.  The census data were linked to

the individual data by geocoding techniques.

Neighborhood was conceptualized at the census block group-level (N=162) for

this analysis, as the block group is small enough that it contains limited variation in

important census indicators of socio-economic disadvantage, but allows for examination

of variation across neighborhoods (Figure 2.2).  Four census items were used to create a

composite neighborhood socioeconomic disadvantage measure.  A principle components

analysis using Varimax rotation indicated that the following four census items loaded

onto a single factor of neighborhood disadvantage that explained 70% of the variance:

percent of families in the census block group at or below 1.5 times the Federal Poverty

Level (FPL), percent of female single-headed households in the block group below the

FPL with at least one child under 18 years of age, percent of unemployment in the block

group and percent of households with a head of household who has less than a high

school education level ($\alpha= 0.83$).  Standardized (mean=0, standard deviation=1.0) values

of each item were computed and summed to create the measure of neighborhood

disadvantage.  In addition to the composite measure, individual disadvantage items were

also tested in the analysis.  Higher values on the individual neighborhood disadvantage

items and the composite measure denote greater levels of neighborhood disadvantage in

the census block group.

**Analytic strategy**

To account for both the longitudinal and nested structure of the data I employed a

three-level analysis using Hierarchical Linear and Nonlinear Modeling software (HLM 6)

(Li et al., 2006).  Multilevel modeling accounts for the intra-individual variation

(multiple measurements for each person) and the intra-group correlation (multiple

participants within each census block group), and partitions the variance and covariance

between and within groups (Raudenbush & Bryk, 2002).  Multilevel modeling has

several benefits including that participants who do not have complete data for every wave

of the study are retained in the models (Raudenbush & Bryk, 2002), it enables cross-level

interactions between neighborhood-level factors and the individual-level time varying

covariates, and it allows for the investigation of random effects at the cluster level.   In

this analysis I examined changes in an individual's perceived stress over time as a function of individual, interpersonal and neighborhood characteristics. Growth curve models represent an individual's development by generating a unique growth trajectory for each individual, which is estimated by a set of growth parameters that are based on these characteristics.

**Missing data**

Although it is not necessary for participants to have complete data for the time varying covariates, HLM cannot handle missing data at the individual (level-2) or neighborhood (level-3) levels. Participants who did not have data at the census block group (neighborhood) level were excluded from this analysis (N=15). The excluded cases did not differ from included cases on any of the time-varying predictors or the demographic variables. I employed the expectation maximization (EM) algorithm to impute missing data at level-2 for family SES and mother's education (West et al., 2006) and conducted an attrition analysis comparing participants with missing (N=88) and complete data (N=593). All missing data is assumed to be missing at random. Participants did not experience a specific event or external factor that would contribute to their propensity to leave the study.

Individuals who had missing data for family SES reported more perceived stress at baseline (Wave 2) (t/df=-2.6/644; $p<0.05$) than individuals with complete SES data. There were no differences at baseline between individuals with complete and missing SES data for: peer support, relationship support, substance use, John Henryism, neighborhood attitudes or neighborhood fear. Only two participants had missing data for mother's education so the sample size was too small for reliable comparisons, although

Fischer's Exact tests and tests of the sample means suggest that the two groups were not different.

## Results

**Change in perceived stress over time**

First I examined a null growth model, which contained a term for the average initial perceived stress (intercept) and the growth parameters (linear and curvilinear change in stress over time). I used this model to determine whether perceived stress varies for individuals over time, and the shape of the trajectory by which it varies. In addition, I determined that initial stress levels and the stress trajectory varied between neighborhoods (census block groups), which indicates that a significant portion of the variance in the dependent variable is attributable to differences between neighborhoods.

Thus, the first model in this analysis can be represented by Equation 1, where $i$ represents the individual, $t$ is the age at time t for an individual, $\pi_{0i}$ is the average perceived stress for individual $i$ at baseline, $\pi_{1i}$ is the linear growth trajectory parameter for stress, and $\pi_{2i}$ is the quadratic growth trajectory parameter for stress. The growth parameter for change in perceived stress used in the model ($\pi_{1i}$) was centered on the minimum age of the sample at the beginning of Wave 2 (15.9 years).

Equation 1

$$Stress = \pi_{0i} + \pi_{1i}(Age)_{ti} + \pi_{2i}(Age^2)_{ti} + e_{ti}$$

The change in perceived stress between baseline (beginning of high school) and early adulthood (approximately three to four years post highs school) was modeled using linear and quadratic growth parameters.  The dependent variable is interpreted as the change in perceived stress for each additional year after baseline.  At baseline, the average perceived stress for an individual was moderate ($\beta$=1.65, [$SE$=0.03], $p$<0.01), and decreased linearly at a rate of 0.05 units per year of age ($SE$=0.01, $p$<0.01).

The linear model of change in perceived stress was tested against a quadratic model and the results of the model comparison indicated that the stress trajectory was, in fact, quadratic ($\chi^2$=10.00[1], $p$<0.01).  The quadratic component of time can be considered as the curvilinear change, or acceleration in an individual's stress trajectory. On average in this sample, perceived stress increased quadratically over time ($\beta$=0.004, [$SE$=0.00], $p$<0.01).  Although the absolute difference in the minimum and maximum stress level between baseline and Wave 8 was not large (~0.15), the change in stress over time was significant in both linear and curvilinear growth parameters.

**Change in perceived stress and neighborhood stressors**

After specifying the basic growth model and determining that the growth parameters for perceived stress vary by census block group, I examined whether the between-neighborhood differences were a function of neighborhood-level disadvantage. The neighborhood disadvantage measure was added to the level-3 intercept and growth parameter equations, and the error term (sigma) was allowed to vary at random for the equations, addressing the hypothesis that the initial perceived stress level (intercept) and changes in perceived stress over time (slope and acceleration) are neighborhood-specific.

Neighborhood disadvantage did not explain any of the variance in the quadratic growth

term and was therefore dropped from this equation.

The final mixed model for the initial perceived stress level for an individual in a

neighborhood of a specific disadvantage level ($\gamma_{001}$), as well as for the linear change in

perceived stress ($\gamma_{101}$) for an individual in that neighborhood, is presented in Equation 2.

$$Stress = \gamma_{000} + \gamma_{001}Disadvantage + \gamma_{100}Age + \gamma_{101}Disadvantage *$$
$$Age + \gamma_{200}AgeSq + r_0 + \mu_{00} + \mu_{10}Age + e$$

(Equation 2)

The model displayed in Equation 2 indicates that an individual's perceived stress level is

a function of the average perceived stress level across all neighborhoods (block groups;

$\gamma_{000}$), the contribution of the neighborhood's specific disadvantage level ($\gamma_{001}$) on the

overall perceived stress level, a person's linear change in perceived stress over time

($\gamma_{100}$), the overall effect of a specific level of disadvantage on a person's linear change in

stress ($\gamma_{101}$), the curvilinear change in stress ($\gamma_{200}$), the unique effect of a particular

neighborhood on an individual's intercept and slope for perceived stress ($u_{00}, u_{10}$), and

the residuals ($r_0$, e).

Although I included only the composite neighborhood disadvantage measure in

the model I also examined individual indicators of disadvantage. Although several

indicators were significant neighborhood-level predictors of perceived stress, the

magnitude and direction of the effects were similar to that of disadvantage; thus the

composite measure is included in all subsequent analyses to simplify the interpretation.

The addition of neighborhood socioeconomic disadvantage improved the model fit ($\chi^2$=8.44[2], p<0.05) over the null model previously presented.  Living in a more disadvantaged neighborhood increased an individual's initial perceived stress levels ($\beta$ = 0.05 [0.025], $p$<0.05).  In addition, individuals living in more disadvantaged neighborhoods reported a steeper decrease in perceived stress (slope) over time than individuals in less disadvantaged neighborhoods ($\beta$ = -0.013 [0.004], $p$<0.01).  Overall, living in neighborhoods with higher levels of disadvantage increased baseline stress levels and accentuated the trajectory of perceived stress over time (Figure 2.3).  This pattern, however, was reversed approximately four years after baseline (~age 20) when individuals who live in neighborhoods with the lowest levels of disadvantage experienced steeper increases in perceived stress than individuals who lived in the most disadvantaged neighborhoods (Figure 2.3).

Next, individual-level controls were added to the model at level-2 to determine whether the relationship between neighborhood disadvantage and perceived stress remained significant after controlling for an individual's SES, sex and mother's education (Equation 3).  The level-2 equation models the baseline perceived stress level (intercept from level-1) and the growth parameters ($\pi_{1i}$ and $\pi_{2i}$) as a function of the static individual-level factors.  A general level-2 equation for baseline stress ($\pi_{0i}$) is displayed below in Equation 3.

$$\pi_{0i} = \beta_{00} + \beta_{01}(Sex) + \beta_{02}(occ.prestige) + r_{0i}$$

(Equation 3)

In the level-2 equation, $\beta_{00}$ is the mean stress level for individual $i$ based on that

individual's sex and family occupational prestige. These demographic variables were

included in models for the intercept only, as they were measured at baseline (Wave 2)

and thus theoretically more closely related to initial stress levels. In addition, sex and

SES were not associated with either growth parameter, and maternal education was not

associated with the intercept or growth terms. These variables were dropped from

following models. Adding the individual controls improved the fit of the model and

these variables are retained in further analysis ($\chi^2$=36.02[2]. $p$<0.01). Individuals who

had higher family SES at baseline reported lower perceived stress levels at baseline ($\beta$ = -

0.004 [0.002], $p$<0.05), and men reported lower initial perceived stress levels than their

female counterparts ($\beta$ = -0.184 [0.033], $p$<0.01).

**Effect of time-varying covariates on perceived stress over time**

Finally, to examine the relationship between exposure to neighborhood

disadvantage and perceived stress over time, while considering intra- and interpersonal

factors, I examined a full three-level model. I examined whether the intra- and

interpersonal covariates were associated with initial levels of perceived stress and

changes in stress over time, while accounting for the level-2 control variables and for

exposure to neighborhood disadvantage. This model also indicates whether exposure to

neighborhood disadvantage affects the stress trajectory above and beyond the effect of

individual-level factors. The results of these models are presented in Table 2.2. I

included the following covariates in the model: high effort coping, maternal, peer and

relationship support, neighborhood attitudes and perceptions of being physically hurt in

the neighborhood. The time-varying covariates were entered in blocks of theoretically

meaningful constructs and the final model is presented (Table 2.2, Models 2-4). All of the level-1 terms remained uncentered, as zero has a meaningful value for each variable.

Risk model

First, substance use was added to the model containing neighborhood disadvantage and individual controls (SES and sex) to examine the hypothesis that using substances may alter a person's perceptions of stress (Model 2). Inclusion of substance use improved the model fit ($\chi^2$=89.22[1], $p$<0.001). Individuals who used more substances reported more perceived stress over time than individuals who used fewer substances ($\beta$ = 0.07 [0.01], $p$<0.01).

Protective model

Next, three promotive factors were added to the risk model to determine whether they counteract the effects of stressors and risk on perceived stress (Model 3). Friend support was not significant and was dropped from the model. Mother and relationship support were inversely associated with an individual's perceived stress over time ($\beta$ = -0.04 [0.01], $p$<0.01; $\beta$ = -0.10 [0.02], $p$<0.01). High effort coping was also inversely associated with perceived stress over time, such that individuals, who reported higher levels of John Henryism, had lower levels of perceived stress over time ($\beta$ = -0.26 [0.02], $p$<0.001).

Addition of the promotive factors to the model reduced the effect of neighborhood disadvantage on perceived stress to non-significance. Inclusion of the promotive factors also eliminated the associations between linear and quadratic growth and stress. Thus, this model indicates that all of the between-neighborhood variation in the linear change in

perceived stress is accounted for by the level-1 and level-2 factors included in the present model ($var(\pi_{10})$=0.00, $\chi^2$=140.38(146), $p<0.5$).

<u>Full model</u>

Three neighborhood perception variables were added to the risk and promotive model, although only two were retained (Model 4). The indicator of an individual's fear of violence in their neighborhood was not significant and was dropped from the final model. People who reported more favorable attitudes toward their neighborhood also reported less perceived stress over time ($\beta$ =-0.04 [0.02], $p<0.05$) and those who were worried about being physically hurt in their neighborhood reported more stress over time than individuals who felt safe ($\beta$ =0.13 [0.03], $p<0.01$). Addition of the two neighborhood perception measures improved the overall fit of the model ($\chi^2$=79.32(2), $p<0.01$) and the other predictors remained unchanged from the previous model.

**Neighborhood variation in time-varying covariates**

Neighborhood disadvantage was not associated with any of the slopes of the time varying covariates at level-3, which indicates that living in a disadvantaged neighborhood does not affect the relationship between intra- or interpersonal factors and perceived stress. I did find, however, that the relationship between high effort coping and perceived stress over time varied based on census block group-level differences. Despite significant random variation in the slope of high effort coping, this variation was not explained by neighborhood disadvantage.

**Discussion**

My study addresses a critical gap in the literature by responding to the recommendations of scholars to include interactions between behavioral, psychological, and social factors in neighborhood research to more thoroughly explore the mechanisms through which neighborhoods influence health (Aneshensel, 2008; Diez Roux & Mair, 2010). I therefore examined the effect of intra- and interpersonal factors on perceived stress, and their interaction with neighborhood disadvantage to influence the trajectory of perceived stress during adolescence and early adulthood. I found that while neighborhood disadvantage was associated with the stress trajectory, this relationship became non-significant when individual risk and promotive factors were included in the model. This may indicate that proximal influences on adolescents and young adults have a stronger effect than more distant neighborhood factors, or it may suggest that contextual factors other than socio-economic disadvantage are more important.

Several intra- and interpersonal factors were associated with baseline stress and the change in perceived stress over time, as expected based on theories of stress and coping. First, I tested a risk model including substance use, and found that individuals who used more substances reported more perceived stress over time than youth who used fewer substances. This is consistent with the tension-reduction hypothesis, suggesting that alcohol and drugs are used to reduce tension (Conger, 1956) due to chronic stressor exposure. The directionality of this relationship is not clear; individuals who experience more psychological stress in their lives may use substances to relieve stress, or individuals who use substances may feel more stressed due to the effects of substance use (e.g., lack of control, withdrawal) (Jackson et al., 2006). Regardless of directionality, it is clear that substance-using individuals may be at greater risk for experiencing higher

levels of psychological stress and resulting health problems related to stress. This suggests that residents of disadvantaged neighborhoods, who experience more chronic stressors than residents of less disadvantaged neighborhoods, are more likely to use substances as a means of relieving stress and tension, but that increased use of substances also contributes to increasing psychological stress over time.

Social support from a mother and another important relationship was associated with less perceived stress over time as expected based on models of stress and coping. Although researchers have found consistent support for beneficial effects of social support on reducing stress and other behavioral and mental health problems during adolescence and adulthood (Cohen, 1988; Turner & Turner, 2005; Weigel, Devereux, Leigh, & Ballard-Reisch, 1998), results for the effect of social support as a buffer against neighborhood stressors on health and well-being are mixed (D'Imperio, Dubow, & Ippolito, 2000; M. Elliott, 2000; Gonzales, 2001; Landis, 2007; Stockdale et al., 2007; Zimmerman & Brenner, 2009). Additionally, several researchers have found that social support is an effective stress buffer for individuals living in more advantaged neighborhoods, but not in neighborhoods with higher levels of socioeconomic disadvantage or under the most stressful conditions (Aneshensel2010; D'Imperio, et al., 2000; Elliott, 2000).

Results from my research, however, indicate that social support is an important resource for youth regardless of their neighborhood context. Individuals who reported receiving more social support from their mother or another relationship were not as strongly affected by living in a disadvantaged neighborhood. These results support the Transactional Model of Stress and Coping, which emphasizes the beneficial effects of

social support as a coping resource to buffer the harmful effects of stressor exposure. Although the literature on neighborhood stressors and health indicates that the availability and effect of social support may be dependent on context, as indicated by neighborhood stress models, I did not find this to be the case. Elliot (2000), for instance, found differential effects of social support on health based on the degree of neighborhood disadvantage, which suggests that disadvantage may inhibit the formation of social ties and reduce the effectiveness of social support in certain neighborhoods (Elliott, 2000). In high socioeconomic neighborhoods, social support was associated with physical and mental health, while it had no significant effect in the most disadvantaged neighborhoods. My research does not support this idea however, as neighborhood disadvantage did not moderate the effect of social support on the perceived stress trajectory.

I also found that having greater social support weakened the association between substance use and stress, although the relationship was still significant. This suggests that adolescents and young adults with strong support systems may rely less on avoidant coping methods like substance use to cope with neighborhood stressors. While future research may consider the limits of resources like social support, interventions to help adolescents and young adults avoid stress should utilize stress and coping frameworks. These models highlight the importance of social support, and focus on strengthening the family unit, in particular, as mother support is critical during all stages of adolescence and early adulthood (Weigel, et al., 1998).

As hypothesized in theories of stress and coping, individuals in our sample who reported more high effort coping also reported less perceived stress over time. Coping is likely to alleviate the effects of stressor exposure and help individuals avoid negative

health outcomes related to the exposure (Pearlin & Schooler, 1978; Thoits, 1995). Active coping, in particular, may be most effective in alleviating the effects of stressor exposure, although researchers suggest that active coping is mainly adaptive in situations in which individuals perceive greater control over the stressor (Compas, Banez, Malcarne & Worsham, B. Compas, Banez, Malcarne, & Worsham, 1991). Although stressors to which residents of disadvantaged neighborhoods are chronically exposed are likely perceived as less controllable, results from my study indicate that even in the face of neighborhood disadvantage, high effort coping was beneficial in reducing stress.

Despite potential immediate benefits in psychological stress reduction as demonstrated in our sample, researchers have found that engaging in high effort coping over extended periods of time may result in serious negative health consequences including hypertension (James, Keenan, Strogatz, Browning, & Garrett, 1992), early onset heart disease, general wear on the body, and poor health (Geronimus, et al., 2006; Warren-Findlow, 2006). Thus, although my study indicates that high effort coping was beneficial for individuals living in socioeconomically disadvantaged neighborhoods, this promotive factor may result in harmful physiological effects later in the life course. Future research could examine long-term effects of high effort coping on the body (e.g., stress biomarkers), in addition to current psychological outcomes. Despite a paucity of research that includes high effort coping as a means to protect against neighborhood stressor exposure, research on neighborhood context and health indicates that individuals who live in disadvantaged neighborhoods have a greater risk for coronary heart disease than their counterparts in advantaged neighborhoods (Diez Roux, 2001), as well as a lower life expectancy (Geronimus, 2001). Research that explores the relationship

between high effort coping and psychological and physiological health will help to better understand how individuals effectively and healthfully cope with stressors.

Finally, I found that an individual's perception of their neighborhood was an important predictor of psychological stress regardless of their neighborhood's economic disadvantage. Residents who had more favorable attitudes towards their neighborhood experienced less stress over time. Conversely, residents who worried more about getting hurt in their neighborhood reported more stress. These results support neighborhood models, which connect structural neighborhood disadvantage to health through psychological and stress pathways (Aneshensel, 2008). Although decreasing economic disadvantage in a community is difficult and often not easily accomplished, my results suggest that by improving individuals' perception of their neighborhoods through cultivating community resources (e.g., libraries, community centers and spaces), and strengthening social ties, it may be possible to decrease their psychological stress. Thus, intervention efforts aimed at building neighborhood assets and resources, and decreasing the presence of stressors like crime, alcohol, drugs and violence, may have the potential to reduce stress, despite a greater context of socioeconomic disadvantage.

Perceived stress is not a static characteristic of an individual (Schmeelk-Cone & Zimmerman, 2003). I found that stress in our sample of adolescents and young adults varied significantly over time. These results are consistent with literature on adolescence and development, which cites early and middle adolescence as a stressful time, characterized by physical, cognitive, social and emotional changes (Weigel, et al., 1998). It is not surprising, therefore, that perceived stress was higher during middle adolescence (ages 15-16), and proceeded to decrease over the next five years. Based on theories of

stress and coping I expect that stress is modified by coping processes, which also change during childhood and adolescence (Compas, Connor-Smith, Saltzman, Harding Thomsen & Wadsworth, 2001). Researchers believe that youth's reliance on emotion-focused coping increases during adolescence (Compas et al., 1991), which may suggest that perceptions of stress decrease during this period due to an improvement in specific coping strategies.

The gradual increase in perceived stress beginning around age 20 to 21 may be partially a result of role and life changes faced by participants in our sample. During this life transition, many individuals are stepping into adult roles and starting families. Research indicates that the onset of many psychological disorders begin during the transition from adolescence into adulthood (Wheaton & Clarke, 2003), which would also suggest that psychological stress may also increase during this transition. Thus, the general shape of the perceived stress trajectory is consistent with the literature and theories on stress and development. Based on the shape of the stress trajectory, which suggests that developmental stage influences stress, I tested a piecewise model that included middle and late adolescence (age 16-19) and early adulthood (20-23). Due to power limitations, however, I was unable to test my hypotheses using a piecewise model, although researchers could continue to consider developmental issues in future research on neighborhood stressors and psychological stress.

Neighborhood effects accounted for a portion of the variability in initial stress levels, and in the linear change in stress over time only when more proximal individual, peer and family influences were not included in the model. Youth living in neighborhoods with higher levels of socioeconomic disadvantage had higher stress levels

at baseline than youth living in less disadvantaged areas, although this relationship did not vary between neighborhoods in our sample. Youth who lived in more disadvantaged neighborhoods, however, experienced a faster decline in their perceived stress levels over time compared to their peers in more advantaged neighborhoods. Finally, an interesting qualitative relationship emerged around age 19, when participants were beginning the transition into adulthood. Individuals who lived in neighborhoods with the highest levels of socioeconomic disadvantage experienced continuing declines in perceived stress between age 19 and 22, while young adults living in neighborhoods with the lowest degree of disadvantage experienced slight increases in perceived stress during the same time period.

Although these trends seem counterintuitive, one possible explanation may be that the young adults who live in neighborhoods with the highest levels of disadvantage eventually begin to succumb to the effects of socioeconomic disadvantage; this may leave them emotionally and mentally numb to their environment, and they may become less reactive to exposure to chronic stressors. Some researchers, for instance, theorize that people may reach a state of emotional equilibrium, in which the effects of persistent poverty subside after the initial stressful effects have passed (Elder & Caspi, 1988). Although this example has been suggested specifically as it relates to families living in poverty, neighborhood disadvantage may exert similar effects.

Although overall, this research indicates that individual and interpersonal factors are more closely related to an individual's perceived stress trajectory in adolescence and early adulthood than neighborhood factors, several limitations of the measurement of neighborhood context in this study may contribute to the lack of findings. First, the

measure of neighborhood disadvantage used in this study was based only on census

indicators of social and economic disadvantage.  Although there are clear benefits of

using this measure (discussed earlier), it may not fully capture all of the important aspects

of the neighborhood social environment that relate to stressor exposure.  Neighborhood

disadvantage, for example, may not account for exposure to crime, violence, or substance

use in the neighborhood.  It may also fail to account for important social processes like

social capital, collective efficacy and social organization, as well as influences of the

built environment, access to resources, and environmental exposures, which are all

related to health, behavior and well-being (Diez Roux & Mair, 2010; Elliott, 1996;

Sampson, Raudenbush, & Earls, 1997).  The neighborhood stress model underscores the

importance of how residents perceive their neighborhood – including stressors like drugs

and crime, and resources like community gardens – on their health.   Including more

diverse and comprehensive measures of neighborhood context in research may result in

stronger associations between neighborhood stressor exposure and perceived stress, as

well as more significant interactions between intra- and interpersonal factors and the

neighborhood context.

In addition, few researchers (including I) examine changes in disadvantage over

time (e.g., some neighborhoods may experiences increases or decreases in socioeconomic

disadvantage from year to year), and the amount of time individuals spend in that

particular neighborhood context (e.g., do they live in the same neighborhood for 20 years

or did they move from neighborhood to neighborhood?) (Wodtke, Harding, & Elwert,

2011).  Wodtke et al. (2011) propose that weak or null neighborhood effects may often be

attributed to poor measurement of neighborhood disadvantage.  They also propose that

research using only a current measure of disadvantage, as I do in this study, may conflate this immediate exposure with lifetime exposure, greatly diluting the results (Wodtke, et al., 2011).

Although future research could more fully account for changes in socio-economic disadvantage over time, as well as residential moves and changes in neighborhood context, the results from my sensitivity analysis suggest that in our sample this may not have posed a significant problem. While participants in the Flint Adolescent Study frequently change residence, they typically move into new neighborhoods with similar levels of disadvantage. In fact, I found that less than 4% of participants who moved (within Flint) moved into neighborhoods with more than a 1.5 standard deviation change in disadvantage, in either direction. Additionally, I included controls for moving into higher or lower disadvantaged neighborhoods for those participants who moved, and these controls were not significant. Although I did not find many significant differences in the degree of disadvantage in an individual's neighborhood between study waves, and tested a model that controlled for residential moves, more elegant and advanced statistical techniques like inverse probability of treatment weighting used in a recent study (Wodtke, et al., 2011), may result in more accurate accounting of stressor exposure.

The lack of significance of neighborhood disadvantage may also suggest that the effects of living in a disadvantaged neighborhood are more important during earlier stages of development instead of during middle adolescence and adulthood, as well as cumulatively over the life course. Wodtke et al. (2011) found, for instance, that sustained effects of exposure to neighborhood disadvantage measured over 17 years on high school graduation were much greater than previously estimated cross-sectional effects. Their

research accounted for early exposure, cumulative exposure, and selection into neighborhoods (Wodtke, et al., 2011). Despite not finding continued effects of neighborhood disadvantage on perceived stress after accounting for risk and promotive factors, my research still contributes to this important body of literature, and could guide approaches to future research.

Finally, there was very little neighborhood-level variation in perceived stress to explain by the individual and neighborhood level factors included in my research. This may suggest that the effect of neighborhood context is small relative to other factors like social support and John Henryism, and that the predictors that explained a portion of this variation are not theoretically significant. The variation in the perceived stress trajectory due to neighborhood residence was significant, however, and may have been underestimated in the model due to over-controlling and selection effects (Wheaton & Clarke, 2003; Wodtke, et al., 2011).

Despite these limitations, several aspects of my study highlight the importance of this research and its implications. First, I examined changes in perceived stress over seven years, which included two important developmental periods: adolescence and the transition into adulthood. Using a growth curve modeling approach allowed me to determine the shape of the perceived stress trajectory for individuals in our sample. This approach addresses theories of development, and supports research that indicates that stress, coping and mental health vary based on age (Compas, Connor-Smith, Saltzman, Thomsen & Wadsworth, 2001; Wheaton & Clarke, 2003). Examining changes in stress over time and the effect of neighborhood disadvantage on the trajectory is essential for effectively translating research into practice, as interventionists should focus their efforts

during periods in development when stress is greatest and gains are likely to be largest. Similarly, examining perceived stress over time also enabled me to distinguish between immediate effects of neighborhood disadvantage on perceived stress, as well as the relationship between disadvantage and stress during development.

Another strength of this study is the use of multi-level modeling techniques to account for the variation in perceived stress at the intra-individual, individual and neighborhood level. This correctly partitions the variance in stress into each level of influence, resulting in more unbiased and accurate variance and covariance estimates. In addition, I was able to apply the ecological framework to my hypotheses, and to test cross-level interactions and their effect on the stress trajectory. Research that does not employ multi-level modeling techniques cannot correctly assess the moderating effect of neighborhood-level characteristics on the outcome of interest.

This research included a relatively high risk, all Black sample. Although stress is a universal construct, it is not equally distributed in the population (Turner & Turner, 2005), and thus examining neighborhood stressors and perceived stress in a more disadvantaged, all Black population is important. This research contributes to the literature on population-level inequalities in stressor exposure (George & Lynch, 2003; Geronimus, et al., 2006), and may indicate that living in disadvantaged neighborhoods play a role in an individual's perceptions of stress, and eventual health. Finally, my research was supported and guided by several theories linking neighborhoods to stress and future health inequalities. Integrating multiple theories into my research helped me more meaningfully connect neighborhood disadvantage to individual-level factors and perceived stress, and guided my statistical analysis.

Results from my research have several important implications for future research, intervention and policy. First, my results support a continued focus on neighborhood as a source of stressor exposure for adolescents and young adults. Although neighborhood disadvantage directly affected baseline levels of perceived stress as well as changes in perceived stress over time, it was not significant after individual-level risk and promotive factors were included in the model. This may suggest that neighborhood influences are not as important as individual and interpersonal influences (e.g., substance use and social support), but it more likely suggests that neighborhood socioeconomic disadvantage is not the best measure of neighborhood stressor exposure, and previously discussed. Future research could include measures of segregation, crime, collective efficacy, neighborhood social capital, and access to resources in addition to census-based measures of disadvantage.

Lastly, my research emphasizes the importance of considering developmental trajectories in planning interventions. Based on my results, intervening around ages 15-17 (middle adolescence) and then early adulthood (during the transition) may be most effective in reducing individuals' perceived stress, as this was when stress levels were highest. At an individual level, interventions can help adolescents and young adults learn how more effectively cope with stressors. At the neighborhood level larger structural, economic and policy changes could be aimed at increasing neighborhood social ties, increasing opportunities and resources available to cope with stressors, and most challenging (yet most necessary), improving the economic conditions of disadvantaged communities.

This study adds to the growing literature on neighborhood effects on adolescent development, and suggests that more research is still necessary. We may need, for instance, to develop new models of neighborhood effects in an effort to understand how neighborhoods may influence individual outcomes, as it may be that neighborhood characteristics are the beginning of a causal mechanism (Wodtke, et al., 2011) that is more complex than existing theory and most research has considered. This may help explain why more proximal family and individual level factors explain more variation in adolescent outcomes then the more distal effects of neighborhoods. My finding that neighborhood effects were washed out after more proximal factors were considered suggests that future research might examine neighborhood factors such as segregation, crime, collective efficacy, neighborhood social capital, and access to resources rather than more global measures of disadvantage. Nevertheless, the fact that neighborhood disadvantage did predict psychological stress when promotive factors were not in the model also suggests that it is too early to abandon efforts to understand how neighborhood context may influence adolescent and young adult development.

**Table 2.1: Means (SD)/Frequency(%) of time varying predictors**

| | Wave 2 | Wave 3 | Wave 4 | Wave 5 | Wave 6 | Wave 7 | Wave 8 |
|---|---|---|---|---|---|---|---|
| **Perceived stress** | 2.53 (0.57), N=646 | 2.48 (0.60), N=627 | 2.52 (0.62), N=611 | 2.37 (0.59), N=465 | 2.46 (0.59), N=504 | 2.44 (0.55), N=459 | 2.46 (0.56), N=470 |
| **Substance use[a]** | -0.03 (0.83), N=639 | 0.00 (0.79), N=620 | -0.03 (0.78), N=604 | -0.01 (0.79), N=473 | -0.01 (0.74), N=511 | -0.03 (0.74), N=461 | -0.00 (0.74), N=470 |
| **High effort coping** | 4.21 (0.57), N=649 | 4.23 (0.54), N=627 | 4.23 (0.62), N=613 | 4.23 (0.59), N=471 | 4.23 (0.59), N=514 | 4.23 (0.59), N=462 | 4.18 (0.61), N=470 |
| **Mother support** | 4.01 (0.96), N=639 | 4.10 (0.95), N=614 | 4.03 (0.99), N=595 | 4.03 (1.02), N=447 | 4.00 (1.01), N=483 | 3.96 (1.02), N=437 | 3.99 (1.05), N=436 |
| **Friend support** | 3.33 (0.97), N=648 | 3.28 (0.94), N=627 | 3.24 (0.98), N=613 | 3.27 (0.93), N=449 | 3.27 (0.93), N=508 | 3.12 (1.00), N=444 | 3.17 (1.02), N=456 |
| **Relationship support** | — | — | — | 4.33 (0.65), N=468 | 4.42 (0.60), N=503 | 4.38 (0.64), N=460 | 4.41 (0.66), N=467 |
| **Neighborhood attitudes** | 2.78 (0.78), N=646 | 2.76 (0.78), N=623 | 2.63 (0.77), N=613 | 2.53 (0.78), N=468 | 2.48 (0.77), N=510 | 2.52 (0.77), N=452 | 2.57 (0.75), N=463 |
| **Fear of neighborhood violence** | 226 (34.8%), N=649 | 206 (32.8%), N=628 | 153 (24.9%), N=614 | 180 (38.3%), N=470 | 191 (37.2%), N=514 | 153 (33%), N=463 | 163 (34.8%), N=469 |
| **Worry get hurt in neighborhood** | 136 (21%), N=649 | 102 (16.2%), N=628 | 85 (13.8%), N=614 | 96 (20.3%), N=472 | 107 (20.9%), N=513 | 91 (19.7%), N=463 | 100 (21.3%), N=469 |

[a] Numbers represent standardized values

**Table 2.2: Changes in perceived stress and time varying covariates and effect of neighborhood disadvantage on the stress trajectory**

| | Model 1 B (SE) | Model 2 B (SE) | Model 3 B (SE) | Model 4 B (SE) |
|---|---|---|---|---|
| **FIXED EFFECTS** | | | | |
| **Age predictors** | | | | |
| Mean stress at baseline $(\beta_0)$ | 1.65 (0.03) | 1.63 (0.03) | 2.53 (0.27) | 2.38 (0.27) |
| Neigh. disadvantage | 0.05 (0.02) | 0.04 (0.02)[c] | NS | NS |
| SES | -0.004 (0.00) | -0.003 (0.00) | -0.00 (0.00) | -0.00 (0.00) |
| Male | -0.18 (0.03) | -0.20 (0.03) | -0.12 (0.04) | -0.11 (0.04) |
| Mean growth $(\pi_1)$ | -0.05 (0.01) | -0.05 (0.01) | NS | NS |
| Neigh. disadvantage | -0.01 (0.00) | -0.01 (0.00) | NS | NS |
| Mean acceleration $(\pi_2)$ | 0.004 (0.00) | 0.004 (0.00) | NS | NS |
| **Risk factors** | | | | |
| Substance use | | 0.07 (0.01) | 0.04(0.02) | 0.04 (0.02) |
| **Protective factors** | | | | |
| John Henryism | | | -0.26 (0.03) | -0.25 (0.03) |
| Mother support | | | -0.04 (0.01) | -0.03 (0.01) |
| Friend support | | | NS | NS |
| Relationship support | | | -0.10 (0.02) | -0.09 (0.02) |
| **Neighborhood perceptions** | | | | |
| Neighborhood attitudes | | | | -0.04 (0.02) |
| Fear of neighborhood violence | | | | NS |
| Worry about getting hurt in neighborhood | | | | 0.13 (0.03) |
| **RANDOM EFFECTS OF MEAN GROWTH** | | | | |
| Variance (SD) | 0.0003 (0.02) | 0.003 (0.02) | NS | NS |
| $\chi^2$ (DF) [a,b] | | 89.22 (1) | 2932.96 (3) | 79.28 (2) |

[a] Degrees of freedom based on number of cases used in computation of random effects
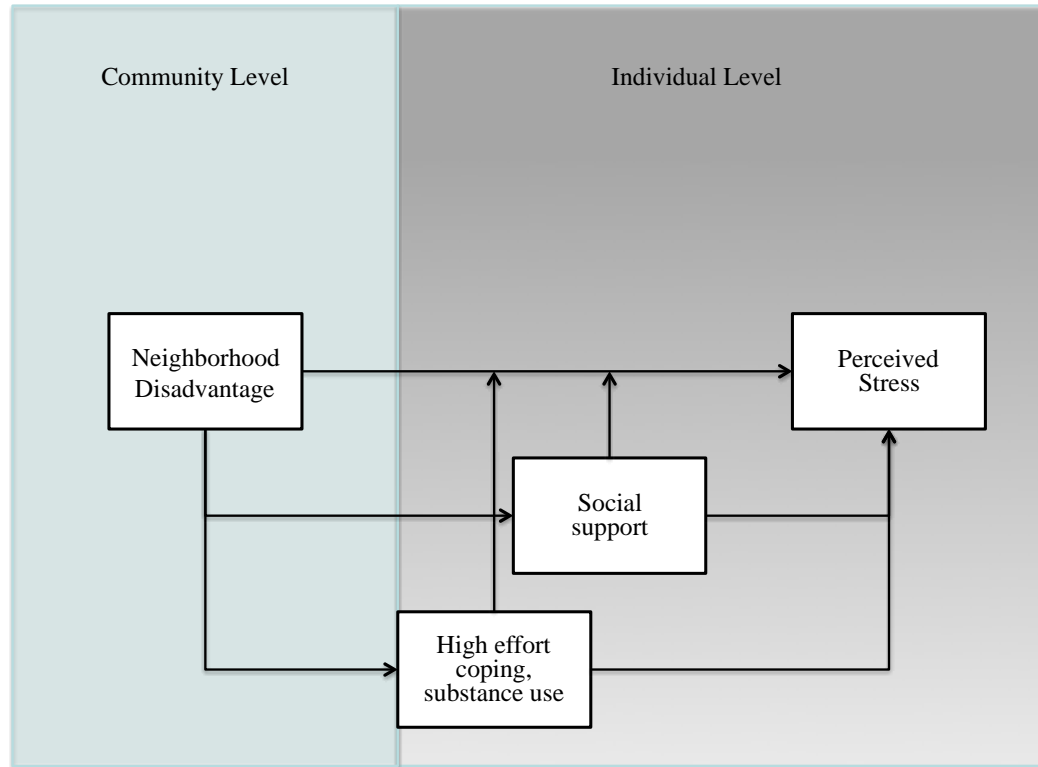[b] Model comparison based on nested models
[c] p=0.06

**Figure 2.2 - Neighborhood disadvantage and psychological stress model**
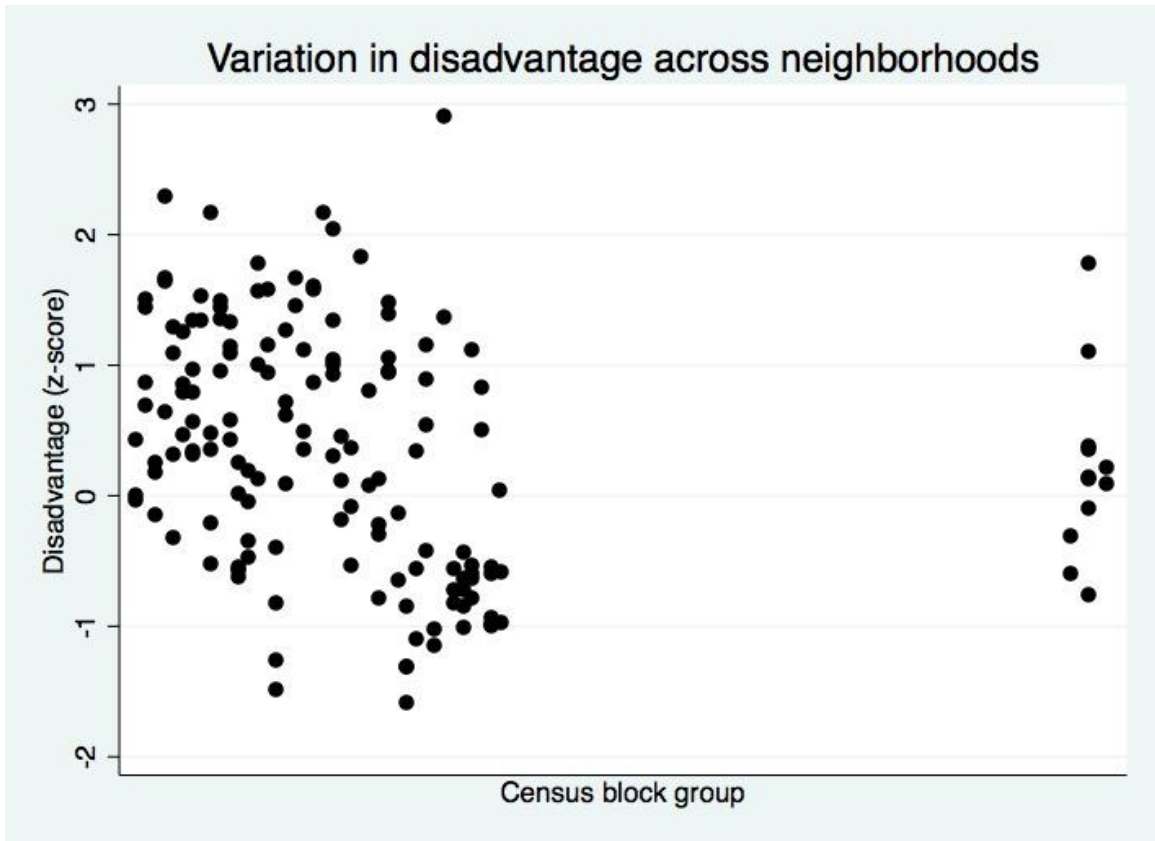
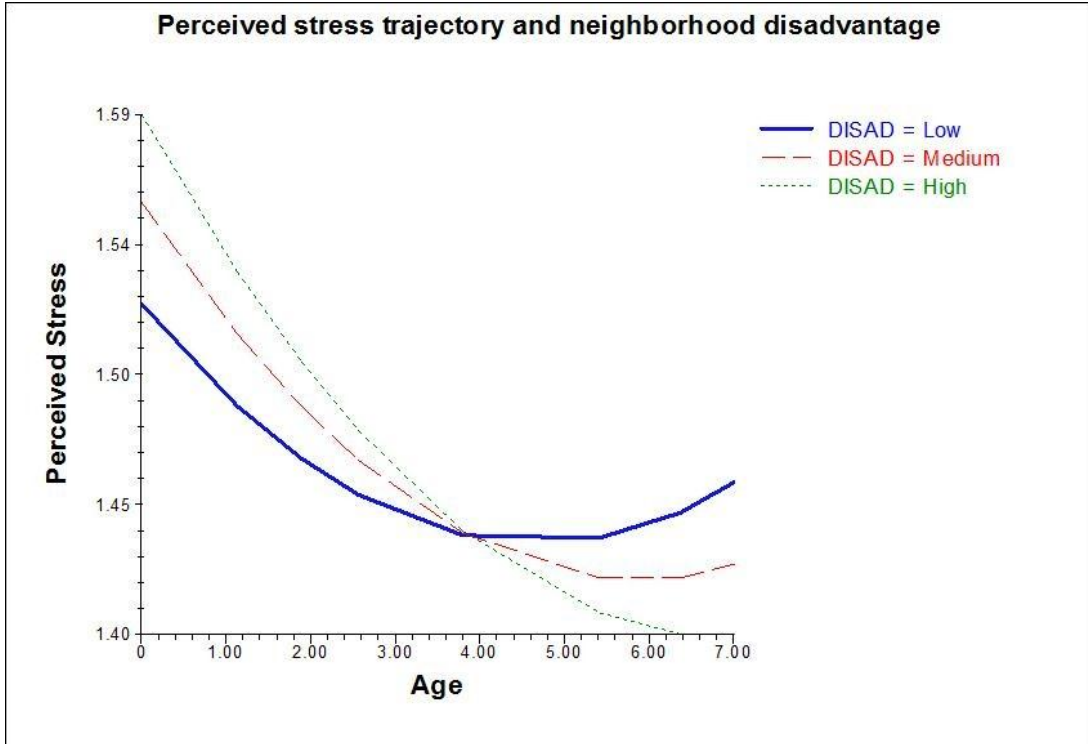**Figure 2.2: Variation in socio-economic disadvantage across neighborhoods**

**Figure 2.3 - Effect of neighborhood disadvantage on perceived stress trajectory**

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# CHAPTER 3

## STRESSFUL NEIGHBORHOODS AND HEALTH: THE EFFECT OF LIVING IN DISADVANTAGED NEIGHBORHOODS ON ADOLESCENT CORTISOL

## Introduction

### Overview

Racial and ethnic health inequalities persist across health outcomes, social groups, and levels of educational attainment (Mays, Cochran, & Barnes, 2007; Williams, 1999). Although the causal mechanisms behind these inequalities are likely multifaceted, one possible mechanism through which they may arise is unequal exposure to stressors and a resulting unequal distribution of stress (Adler et al., 1994; Baum, 1999; Pearlin, 1989). Thus, differential exposure to stress may partially explain population patterns of health and well being (George & Lynch, 2003; Turner & Lloyd, 1995); compared to Whites, Black Americans experience more frequent chronic and acute stressors, stressful life events, and trauma, after controlling for their socioeconomic status (Jackson, Knight, Carstensen & Schaie, 2006; Kessler & Neighbors, 1986; Turner & Lloyd, 1995). Individuals of lower socioeconomic status (SES), and who live in disadvantaged neighborhoods, are more likely to be exposed to episodic and chronic stressors (Friedman & Lawrence, 2002; Latkin & Curry, 2003; Steptoe & Feldman, 2001). The neighborhood context – which reflects larger societal structures including racism, discrimination, residential segregation and concentrated poverty, as well as an individual's access to economic resources, safety and social support – may help researchers better understand

how socio-cultural and economic factors get under the skin and result in health inequalities. **Neighborhoods and stressor exposure**

Pearlin (1989) argued that stress is socially patterned and deeply rooted in structural contexts that affect health across the entire life course, with effects beginning as early as development in utero. Research indicates that neighborhoods characterized by segregation, poverty, crime, violence, residential instability, social isolation, and unemployment exert negative influences on the physical and mental health, and behavior of its residents (Browning & Cagney, 2003; Ellen, Mijanovich, & Dillman, 2001; Hill, Ross, & Angel, 2005; Kruger, Reischl, & Gee, 2007; Steptoe & Feldman, 2001; Wen, Cagney, & Christakis, 2005; Wright, Bobashev, & Folson, 2007). Black Americans may be disproportionately exposed to these stressors within their neighborhood, as they are more likely than their non-Black counterparts to live in neighborhoods characterized by poverty; crime and violence; and physical and social deterioration (Browning & Cagney, 2003; Merkin et al., 2009). These harmful neighborhood effects persist after controlling for individual level factors (i.e. socioeconomic status, education), although they are likely to mediate and interact with individual and interpersonal factors including demographics, coping efforts and social support (Aneshensel, 2008; Diex Roux, A. Diez Roux, 2001; Morenoff, 2003).

One avenue through which neighborhoods may influence health is through a stress pathway. Residents living in disadvantaged neighborhoods may encounter more frequent, severe, and sustained stressful events than residents living in more affluent neighborhoods (Latkin & Curry, 2003). They may also experience this stressor exposure over many years (i.e., cumulative stressor exposure), as mobility from disadvantaged to

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more advantaged neighborhoods is limited (Browning & Cagney, 2003; Latkin & Curry, 2003). In addition, children and young adults who grow up in disadvantaged neighborhoods are likely to remain in similar neighborhoods as adults, which may amplify their lifetime exposure to contextual stressors like disadvantage (Garmezy, 1991). Neighborhood disadvantage is often a direct outcome of residential segregation and concentrated poverty, which contributes to physical deterioration of neighborhoods, crime, violence, vandalism, and fear (Ewart, 2002; Latkin & Curry, 2003; Steptoe & Feldman, 2001). These symptoms of disadvantage contribute to the chronic stress and strain that threatens the health of residents living in poor neighborhoods. Additionally, neighborhood disadvantage and its correlates may undermine neighborhood, family, and peer social support systems; personal resources; and the social and economic resources that help buffer residents against the noxious stressors of disadvantage (Gonzales, 2001; Latkin & Curry, 2003; Ross, Mirowsky, & Pribesh, 2001).

Neighborhood models help to explain the relationship between residential context and stress (Aneshensel, 2010). The structural model posits that socioeconomic disadvantages at the neighborhood level (e.g., census-level characteristics) interact with socioeconomic factors at the individual level (e.g., socioeconomic status) to jointly influence an individual's mental health in a manner that extends beyond the exclusive effect of an individual's socioeconomic status (SES). The structural model, for example, posits that individuals with higher SES, but who live in disadvantaged neighborhoods, will still face more risk for mental health problems than higher SES individuals who live in less disadvantaged areas (Aneshensel, 2010). The test of this model requires a multi-

level analytic technique to examine the effect of an individual nested within a neighborhood.

The neighborhood stress model also describes the relationship between residential context and stress. In this model, neighborhood disadvantage influences mental health via a neighborhood disorder pathway (Aneshensel, 2010). Disorder is characterized by factors such as crime, a drug presence, graffiti, litter, noise and abandoned buildings in the neighborhood (Ross & Jang, 2000). Although neighborhoods with greater disadvantage are not necessarily disordered, researchers report a strong relationship between disadvantage and disorder (Ross & Mirowsky, 2001), and the neighborhood stress model proposes disorder as a mediator of the relationship between neighborhood disadvantage and mental health, and a direct source of stress for residents (Aneshensel, 2010). Ross and Mirowsky (2001) found, for example, that the relationship between disadvantage and health is almost fully explained by neighborhood disorder and by fear, suggesting that the mechanism by which neighborhood disadvantage influences health is through perceptions of social and physical disorder in an area. In combination, both the structural and stress models guide the present study and connect a person's residential context to their exposure to stressors and thus to physiological indicators of stress.

**Stressors and stress**

The influence of residential context on psychological and mental health has been explored in depth (Diez Roux & Mair, 2010; Hill, et al., 2005; Latkin & Curry, 2003; Mair et al., 2009), and researchers frequently cite disadvantaged neighborhoods as stressful (Latkin & Curry, 2003; Steptoe & Feldman, 2001). The direct relationship between neighborhoods and stress (perceived or physiological markers of stress),

however, has received little investigation.  Although perceived stress is an important measure of an individual's psychological response to stressor exposure, it may not accurately represent an individual's chronic stress burden.  Individuals may not report perceived stress accurately or consistently, and psychological stress may primarily reflect current and not chronic or cumulative stressor exposure (Worthman & Costello, 2009).  In addition, individuals who are chronically exposed to stressful stimuli, as may be the case for residents of a highly disadvantaged neighborhood, may not perceive high levels of stress despite their exposures because they may psychologically acclimate to stressor exposure despite continuing harm to their physical health (Worthman & Costello, 2009).  While psychological stress measures are valuable indicators of individuals' current psychological state, physiological biomarkers like cortisol may more adequately represent the connection between chronic stressor exposure and health, and may therefore enable researchers to study prolonged stressor exposure and the resulting *wear and tear* on the body (Geronimus, Hicken, Keene, & Bound, 2006; Geronimus & Thompson, 2004; McEwen & Seeman, 1999).

**Physiological stress response**

When confronted with a stressful situation (stressor), the body initiates a stress response, which is mediated by the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (McEwen & Seeman, 1999).  During a stress response, hormones including cortisol and catecolamines are secreted by the body, beginning a cascade of events among receptors and cells (McEwen & Seeman, 1999).  Pearlin et al. (1981) note that the stress response may be reflected in multiple pathways including single cells and organs; biochemical, physiological and emotional functioning; entire

body systems like the endocrine, immunologic and cardiovascular systems; or in the incidence of physical or psychological distress. Although a stress response to an acute stressor is normal and helps regulate the body and maintain *allostasis* (maintaining stability through change, or maintaining homeostasis), repeated challenges to allostasis due to chronic stressor exposure can damage the feedback loops and wear down the body (Dowd, Simanek & Aiello, 2009; McEwen & Seeman, 1999). These constant challenges to allostasis, or the cumulative effect of failures in regulating allostasis over time is referred to as *allostatic load*. This study focuses on cortisol, which is one specific biomarker used to assess allostatic load.

Physiological biomarkers (e.g., cortisol, norepinepherine, DHEA) are objective (though not perfect) measures that are believed to reflect cumulative exposure to stressful conditions (Peek et al., 2007; Worthman & Costello, 2009). These biomarkers, which may mediate the association between stressful exposures and particular physiological health outcomes, allow researchers to connect social exposures to health. Biomarkers account for individual vulnerabilities, and interactions between individuals and their environment, as biomarkers reflect exposures, adaptation and response for an individual (Worthman & Costello, 2009). Worthman & Costello (2009) suggest "biomarkers can complement sociocultural analysis by probing biocultural pathways that shape health but lie outside of awareness, everyday observation, or cultural discourse (p. 284)." More generally, biomarkers may allow researchers to measure the complex socio-cultural and environmental pathway to specific health outcomes.

Cortisol

Cortisol is one of the primary hormones or stress mediators released during a stress response, which regulates the secondary and tertiary outcomes like heart rate and blood pressure (McEwen & Seeman, 1999). These secondary and tertiary outcomes are health indicators that are precursors to many chronic diseases including cardiovascular disease and diabetes. Thus the primary hormones, including cortisol, are important biomarkers of later disease that may relate back to the stress response and cumulative stressor exposure. Cortisol is largely responsible for regulation of the central nervous, metabolic and immune systems, which makes cortisol a critical intermediary between stressor exposure and disease (Miller, Chen, & Zhou, 2007). This relationship between a single biomarker (cortisol) and multiple health outcomes across several physiological systems makes cortisol desirable to study as an indicator of stressor exposure.

Researchers believe that cortisol is related to many poor health conditions; chronically elevated cortisol levels have been associated with cognitive decline, immunosuppression and insulin resistance (Dowd et al., 2009; Miller, et al., 2007). Despite the emerging and robust evidence of the health effects of cortisol dysregulation, the relationship between stressor exposure and cortisol is complex. Although earlier (and the majority of the) research on cortisol dysregulation cites elevated cortisol levels as hazardous, researchers have found that low cortisol levels (i.e., hypocortiosolism) may also be detrimental (Dowd et al., 2009; Miller, et al., 2007). In addition, cortisol reactivity varies based on the type of stressor to which an individual is exposed (Dowd et al., 2009). Dowd et al. (2009) concluded that a lower cortisol awakening response (CAR) (the increase in morning cortisol levels during the first waking hour) is associated with chronic health problems and post-traumatic stress disorder, but that people who

experienced higher job and social stress had a higher CAR.  Finally, cortisol reactivity

varies based on the time of day.  In a meta-analysis of exposure to chronic stressors and

HPA outcomes including cortisol, Miller et al. (2007) found that chronic stressor

exposure was associated with lower levels of morning cortisol, higher cortisol output

during the day, and a flattened diurnal rhythm.  Thus, the complex relationship between

stressor exposure and cortisol must be carefully examined in research.

Several researchers have examined the association between individual-level risk

or stressors (e.g., SES, experimental stress tests, and life events) and biomarkers or

allostatic load (Buske-Kirschbaum et al., 2003; Dowd et al., 2011; Dowd et al., 2009;

Gersten, 2008; Gustafsson, Anckarsäter, Lichtenstein, Nelson, & Gustafsson, 2010; Hajat

et al., 2010; Heim & Nemeroff, 2001; K. Schmeelk-Cone, Zimmerman, & Abelson,

2003).  Researchers have also examined demographic differences in allostatic load based

on population-based studies (Geronimus, et al., 2006; Peek, et al., 2007).  Few

researchers, however, have examined the association between contextual-level stressors

and cortisol (or allostatic load).  Evans et al. (2007) studied the relationship between

cumulative risk and allostatic load, and included a measure of housing quality as a

contextual risk factor.  The measure, however, was based on a rater's observation and

was included as an individual-level exposure variable.  Similarly, Kliewer et al. (2008)

included neighborhood quality in their composite risk score. Chen and Paterson (2006)

investigated the relationship between neighborhood census indicators and cortisol in a

sample of adolescents, and found that individuals in their sample who lived in lower SES

neighborhoods had lower basal cortisol levels.  Their study, however, used only a single

cortisol measurement, which is difficult to interpret in relation to health given the

complexity of the cortisol diurnal rhythm (Almeida, McGonagle, & King, 2009). Phuong

Do et al. (2011). Additional research that explicitly connects contextual stressor

exposure to stress biomarkers is critical in improving our understanding of residential

stressor exposures and health; this area of research is largely unexplored. This study will

address this gap in the literature, and examine the effect of neighborhood socio-economic

context on physiological stress.

**Connecting socio-economic exposures to physiological outcomes**

The biopsychosocial model was first used in medicine to situate a patient and

their ailments within a human, family, community, cultural and larger ecological context,

in contrast to the individual-centered biomedical model (Engel, 1980). More recently,

the biopsychosocial model has been used to guide research on racism and health

inequalities (Clark, Anderson, Clark, & Williams, 1999), as well as biological aging and

health (Crimmins & Seeman, 2004). Although this model is derived from the more

general stress and coping framework, researchers propose that the biosocial framework is

particularly relevant for understanding racism, stress, and health as it relates to Black

Americans (Anderson, 1991). The Transactional Model of Stress and Coping (Lazarus &

Folkman, 1984) and the biopsychosocial model (Engel, 1980) help understand the

mechanism through which exposure to stressors in the neighborhood may result in

physiological stress and health. These theories conceptualize the stress response as a

process that is dependent on individual's perception of the stressor(s), demographic and

psychological factors, coping processes, behaviors and availability of social support

(Folkman, 1984; Wenzel, Glanz, & Lerman, 2002).

The biopsychosocial model, displayed in Figure 3.1 (adapted from Anderson, Glaser and Glaser, 1994 and Clark et al. 1999), connects stressor exposure to health outcomes via a mediating/moderating pathway that includes psychological, behavioral and physiological factors, and supports the idea that an individual's perceptions of their neighborhood, and psychological stress, may moderate the relationship between neighborhood stressor exposure (disadvantage) and physiological stress. I include measures of perceived stress, neighborhood attitudes, and neighborhood fear, as individuals who report more psychological stress and more negative feelings towards their neighborhoods may also have atypical cortisol levels.

The Transactional Model of Stress and Coping and the biopsychosocial model also indicate that the relationship between neighborhood stressor exposure and cortisol may be moderated by coping, social support and behaviors. High effort coping (John Henryism), a prolonged coping process in response to extreme psychosocial stressors and barriers to success (James, 1994; Sellers & Neighbors, 2008), may be associated with an atypical cortisol response, and may moderate the relationship between neighborhood disadvantage and cortisol. Extended coping efforts required to overcome chronic stressors such as neighborhood disadvantage, may slowly break down the body, both mentally and physically (James, 1994). Although high effort coping may help individuals overcome stressors through hard work and determination, it can also cause physical damage to the body, as indicated by research by James (1994), who found that Blacks who engaged in high levels of John Henryism and who had low socioeconomic status, had higher blood pressure and higher rates of hypertension than Blacks who engaged in low levels of John Henryism. Thus, high effort coping may interact with

neighborhood disadvantage to increase wear and tear on the body, and thus may amplify

cortisol (Geronimus, et al., 2006).

Social support may also play an important role in affecting cortisol, as the

protective role of social support as a means to buffer individuals from the psychological

and physical results of exposure to stressors is well-documented, (Ensel & Lin, 1991;

Lyons, Mickelson, Sullivan, & Coyne, 1998; Thoits, 1995). Cohen and Wills (1985)

propose that received support may directly intervene in the neuroendocrine response to

stressful stimuli, and reduce physical reactivity to the stressor, resulting in reduced

cortisol reactivity. In addition, social support may reduce psychological reactivity to the

stressor or aid in healthy coping, which may then indirectly reduce physical reactivity and

resulting physiological markers of stress (Cohen & Wills, 1985). Despite these

hypothesized relationships, limited research includes social support as a mediator or

moderator between stressor exposure and physiological stress (Uchino, Cacioppo, &

Kiecolt-Glaser, 1996), and of the research, only a handful of researchers examine the

moderating or mediating effect of social support on cortisol (Uchino, et al., 1996). This

research will examine the association between perceived social support and cortisol

reactivity, as well as the interaction between neighborhood disadvantage and social

support on cortisol reactivity.

Finally, I include a measure of substance use as a pathway in the model

connecting neighborhood disadvantage to cortisol, as individuals living in stressful,

disadvantaged neighborhoods may be more likely than individuals living in less stressful

areas to engage in substance use to relieve stress or escape chronic stressors encountered

in daily life (Jackson et al., 2009). Jackson et al. (2009) hypothesize that when people are

confronted with chronic and uncontrollable stressors, such as those reflected by measures of neighborhood disadvantage, they may use substances to immediately relieve the psychological strain (Jackson, Knight & Rafferty, 2009). Substance use may therefore operate as an avoidant (type of passive) coping method, which may moderate the relationship between contextual stressors and stress (Jackson et al., 2009; Lambert, Brown, Phillips, & Ialongo, 2004; Schmeelk-Cone & Zimmerman, 2003). Although substance use may reduce anxiety from exposure to stressors because of its depressive effects on the HPA axis, hormone elevation and activation of the HPA axis may also contribute to physiological stress and allostatic load (Jackson et al., 2009).

**The current study**

To date few researchers have examined physiological biomarkers of stress as an endpoint (Hajat, et al., 2010; Kliewer, et al., 2008; Schmeelk-Cone et al., 2003). Fewer researchers have included neighborhood exposures in their analyses, and none have examined stress and coping or health behavior as a part of the model. The current study, will therefore address these gaps in the stress literature, by integrating stress and coping, biopsychosocial and neighborhood theories to better understand the relationship between neighborhood stressors and cortisol reactivity.

The overall aim of this study is to examine the relationship between intra- and interpersonal factors and physiological stress, measured by cortisol (baseline and reactivity), to examine the relationship between neighborhood stressors and cortisol (Figure 3.2), and to examine cross-level interactions, or the moderating pathways between the individual and neighborhood levels on cortisol. Research that focuses on contextual-level stressors and neuroendocrine biomarkers will help abate some of the

confusion in the literature surrounding the importance of psychological versus physiological stress, as well as the relationship of exposure to stressors and resulting stress. In addition, moderating factors like active coping and substance use may interact with neighborhood stressor exposure to affect cortisol differently than they interact with stressors to affect perceived stress.

## Methods

**Data design and sample**

To test the proposed hypotheses, data from the Flint Adolescent Study (FAS) will be used. The FAS is a longitudinal study of 850 youth at risk for substance use and school dropout. Eligible students included ninth graders enrolled in public high schools in an urban city in Michigan, and who had an eighth grade grade point average (GPA) of 3.0 or below upon entering high school. Youth who were diagnosed by the schools with emotional or developmental impairments were excluded from the study sample. Youth self-identified as African American (80%), White (17%) or Bi-racial (3%). Males and females were equally represented in the sample.

The FAS consists of four waves of data collected during the high school years (Waves 1-4; 1994-1997), four waves of data collected after high school (Waves 5-8; 1999-2002), and two waves of data collected in early adulthood (Waves 9-10; 2008-2009). Retention rates were generally high (90% from Waves 1 to 4 and 75% from Waves 4 to 8). Data were collected by structured face-to-face interviews and a self-administered portion, conducted either in a school or alternative community location. The interview included most of the psychosocial variables in the data, as well as

information on employment, education, family and friends. A self-administered

questionnaire assessed more sensitive information (e.g. substance use, sexual risk

behavior, discrimination) and was distributed at the conclusion of each interview in order

to facilitate confidentiality. The individual-level data was linked to census data (1990 or

2000 depending on the year of data collection) based on geo-coded home address

information. This study was approved by the University of Michigan Institutional Review

Board (UMIRB# H03-0001309).

Only the sixth wave of data collection, which occurred between 2000 and 2001

when the youth were in their early 20's, is used in this analysis. Saliva samples were

collected from a subset of youth (N=201) of those who were present in Wave 6 (N=573),

who consented to the procedure, and who had not eaten, drank or used tobacco in the

hour prior to the collection in Wave 6. Saliva was sampled at three different time points

during the interviews from participants who agreed to the collection, were not pregnant,

and were otherwise eligible for the saliva collection. Participants provided saliva

samples ten minutes into the interview, approximately mid-way through the interview,

and 30 minutes after the second collection. Samples were placed on ice prior to

transportation and assay. Cortisol levels were assayed by Salimetrics, Inc. The current

sample consists of 163 African American youth who provided saliva samples in Wave 6.

Youth who consented to saliva sampling were not different from the overall Wave 6

sample. Half of the sample is male, and participants are 21 years old on average

(SD=0.63).

**Measures**

Sample means, standard deviations, ranges and Cronbach's alphas for the

measures are included in Table 3.1 and a description of each measure is included below.

**Individual-level**

Cortisol is the dependent variable examined in this analysis. Cortisol was measured in

the thawed saliva samples (Salimetrics, Inc.) using a high-sensitivity salivary cortisol

enzyme immunoassay. The three measurements of cortisol (measured as µg/dl) are used

in the growth curve analysis to determine the change in an individual's cortisol during the

interview. Saliva samples were taken about 10 minutes into the interview, approximately

22 minutes after the first sample, and then approximately 30 minutes after that. All

samples were placed on ice and refrigerated after they were taken. Cortisol values were

log transformed to reduce the skewness of the distribution, and the transformed values are

used in the analysis. Saliva sampling time was included in the models as a control

variable, as the time of day at which the saliva was collected may affect cortisol readings

(diurnal patterns).

Several demographic factors are included in the analysis as control variables.

Youth's sex was coded as 0=female and 1=male. Family SES from Wave 1 and mother's

highest education are also included in order to control for a measure of family

socioeconomic status when the participant was a young adolescent, as early life

circumstances may be more important predictors of stress than current SES. Family

socioeconomic status (SES) is assigned based on the highest occupational prestige score

for either parent, using codes developed by the National Opinion Research Center and

then standardized to facilitate interpretation (Nakao & Treas, 1990). The score is

assigned based on 20 occupational classifications, ranging from private household work

(scored as 29.28) to professional (scored 64.38). The mean prestige score in our sample was 39.07 (*SD* =9.9), which corresponds to a blue-collar occupation.

Maternal education was used as an additional measure of socioeconomic status. Mother's highest education level was entered into the analysis as a continuous variable and was originally assessed based on a seven-level interval response format. The mean education level in our sample was 4.55 (*SD*=2.00), which corresponds roughly to an average vocational/training school education (using a scale from 1=less than high school to 9=graduate degree). Self-reported health status is also assessed as a covariate and possible confounder of the relationship between exposure to stressors and stress biomarkers. Health was measured by asking participants to compare their health to that of other people, and the answer choices include: healthier than others, equally healthy as others, less healthy as others. Health was included as a continuous variable in the analysis due to its relatively normal distribution. Higher values correspond to worse self-reported health. In our sample, 68.5% of participants rated their health the same as others, and about 21% rated themselves as healthier than others.

Substance use is a composite measure that includes items about smoking (number of cigarettes smoked in the past 30 days), alcohol use (composite measure of past 30 day alcohol use, binge drinking over the past two weeks and drinking to get high), and marijuana use (past 30 day marijuana use). Each item or scale was standardized (mean=0, SD=1.00), and the average of the three standardized variables was computed to create a substance use scale (Cronbach's alpha = 0.62). Substance use was entered into the models as a 3-level categorical variable (low, average and high use), because preliminary analyses indicated that the continuous measure was not linearly associated

with the dependent variable. The levels of the dummy variable were created based on tertiles of the continuous distribution, and "average" substance use was used as the reference category and omitted from the regression equation.

Active coping was operationalized using the John Henryism measure that was originally developed by James (1999) to assess high effort coping. High effort coping is a prolonged coping process used by African Americans in response to psychosocial environmental stressors and barriers to success (James, 1994). The measure of John Henryism is an eight-item scale that asks respondents to rate items like: "Hard work is the best possible way for someone to get ahead in life," and "sometimes I feel that if anything is going to be done right, I have to do it myself." These items are rated on a 5-point Likert scale (Not true to Very true) and then averaged to create a measure of high effort coping where higher values signify more coping (Cronbach's alpha = 0.85). As with the substance use predictor, a three-level categorical variable was created for John Henryism (low, average and high coping) based on quartiles of the continuous distribution to correct for the non-linear relationship between coping and cortisol, and "average" John Henryism was used as the reference category and omitted from the equation.

Three sources of perceived social support were assessed in this study. Procidano and Heller's perceived support scales were adapted slightly and used in the FAS questionnaire (Procidano & Heller, 1983). Mother's support was measured by five items using a 5-point Likert Scale. Items include the degree to which the adolescent's mother gives emotional and instrumental support, and the closeness of the mother-youth relationship. The support scale ranges from 1 to 5 and a higher value corresponds to

greater levels of social support (Cronbach's alpha = 0.92). A three-level categorical variable was created for mother support based on quartiles of the distribution (low, average and high support) because the continuous measure was not linearly related to the dependent variable. "Average" maternal support was used as the reference category and was omitted from the regression equation.

Like maternal support, the peer support measure includes items assessing the same types of emotional and instrumental support, which are averaged to create a composite measure. The scale ranges from 1 to 5 and a higher value corresponds to greater levels of social support (Cronbach's alpha = 0.91). The same categories were used in the models for peer support as were used for maternal support (low, average and high), and these were created based on tertiles of the distribution. Finally, the questionnaire asks the respondent to identify a person in their lives to whom they feel closest. This person is likely to be a significant other, but could be another family member or friend. Relationship support was measured as a mean score of 6-8 items (this scale was changed slightly between waves of the study to assess support one receives from an intimate partner). It includes items such as: "how much the person provides you reassurance and encouragement when needed", "shows that he/she cares about you as a person", and "gives you useful information or advice when you need it." The scale ranges from 1 to 5 and a higher value signifies more relationship support (Cronbach's alpha = 0.83). The three-level categorical version of relationship (low, average and high relationship support) was used as with the other social support predictors.

Daily hassles, which is a measure of perceived or psychological stress in the past month was assessed using Cohen and colleagues' Perceived Stress Scale (PSS) (Cohen,

Kamark & Mermelstein, 1983), which was originally a 14-item scale designed to measure subjective (psychological) stress.  The scale assesses the degree to which people believe their lives to be "unpredictable, uncontrollable, and overloading" (p. 387) (Cohen et al., 1983).  A shortened version of the PSS was used in the FAS; 11 items were included in the survey based on a principal components analysis.  Sample items include, "In the past month, how often have you…": been upset because of something that happened that you didn't expect, felt that you had so many problems that you could not deal with them, and felt able to handle your personal problems (positive items were reverse coded).  The items were averaged and used as a continuous measure.  The scale ranges from 1 to 5 and higher values denote more perceived stress (Cronbach's alpha = 0.82).  The relationship between daily hassles and cortisol was also non-linear, so a three-level categorical predictor was created based on quartiles of the linear distribution (low, average and high daily hassles), and "average" hassles was used as the reference category in the models.

**Neighborhood perceptions**

Two measures of perceptions of the neighborhood are included in this analysis. Fear of violence, assesses whether participants are afraid of violence in their neighborhood.  The item was measured on a scale of 1 to 5 with higher values representing more fear.  To reduce skewness the variable was dichotomized into a score for participants who reported low levels of fear (below or equal to the median) and another score for those who reported high levels of fear (above the median).  The second neighborhood perception measure, worry about getting hurt, asks respondents whether they worry that someone in their neighborhood will physically hurt them.  This is also measured using a scale of 1 to 5 where higher values correspond to more worry.  This

variable was also created into a dichotomous measure that divides participants into two categories of worry based on the sample median.  Higher values on this item therefore correspond to greater fear that they will be hurt in their neighborhood.

**Neighborhood-level measures**

Census data

The neighborhood level disadvantage variables were taken from 2000 US Census data.  The census data was linked to the individual data by geocoding techniques. Neighborhood was conceptualized at the census tract level (N=36) for these analyses (Sampson et al., 1997).   Seven census items were included in the models.  The seven items are as follows: percent of families in the census tract with annual incomes less than $15,000 (referred to as poverty), percent of families in the tract who are on public assistance, percent of families in the tract who are single-headed and have at least one child under 18 years of age, percent of unemployment in the tract, percent of households detached from the labor force in the tract, percent of vacant housing in the census tract, and percent of households with a head of household who has less than a high school education level.  Higher values on the individual neighborhood disadvantage items denote greater levels of neighborhood disadvantage in the census tract.

**Analytic strategy**

To examine the proposed research hypotheses I conducted a multilevel analysis using HLM 6.0 software (Raudenbush, Bryk & Congdon, 2004), and examined the effect of individual-level risk and promotive factors, and neighborhood-level disadvantage (stressors) on cortisol reactivity.  Multilevel modeling accounts for both the intra-individual (multiple measures over time) and intergroup clustering (neighborhood tract

data) by partitioning variance in cortisol reactivity into within person variance, between person variance, and between group (census tract) variance.

Multilevel modeling not only corrects for correlations within individuals and within groups, but it has several additional benefits. Individuals who do not have complete data for the dependent variable at all time points are retained in the models (Raudenbush & Bryk, 2002). Multilevel models also enable cross-level interactions between neighborhood-level factors and the individual-level time varying covariates, which is not testable in traditional repeated measures or clustered analyses.

Although HLM handles missing data for cortisol at level-1, it does not handle missing data at levels 2 or 3. To maximize the sample size, I imputed missing data at level-2 (individual level) to ensure that cases with missing demographic data were not dropped from the analysis. Expectation maximization (EM) algorithm (West et al., 2006) in EQS (6.1, Multivariate Software Inc.) was used to impute missing data. We conducted an attrition analysis comparing participants with missing (N=46) and complete data (N=133) to ensure that the imputation did not result in a biased analysis. Based on this analysis I found no significant differences in the original or imputed sample.

I performed the data analysis in successive steps. First, a fully unconditional model (FUM) was examined to confirm that there was significant variation in individuals' initial cortisol levels and in cortisol reactivity by neighborhood (census tracts). Next, a variable for time was included in the model to examine the change in cortisol (reactivity) during the interview (level-1 or time varying model). Then, I determined whether adding fixed individual-level factors improved the model fit by explaining some of the random variation in initial cortisol levels and in cortisol reactivity

(level-2 model). All fixed factors that had a meaningful value of zero (e.g., categorical variables and dummy variables) were entered into the models as uncentered variables. All other variables, including the neighborhood disadvantage indicators, were centered around their grand mean.

Finally, I included neighborhood disadvantage indicators in the model (level-3 model) to determine whether the addition of these factors explained additional variation in initial cortisol levels or cortisol reactivity attributed to the neighborhood level. Each neighborhood-level variable was entered and tested in the model separately due to power limitations of estimating random variation of several variables at the neighborhood level. I also examined cross-level interactions between disadvantage and individual risk and promotive factors, as I expected the risk and promotive factors to differentially affect cortisol based on the socio-economic characteristics of individuals' neighborhoods.

To determine whether the relationship between neighborhood disadvantage and cortisol varied between and within neighborhoods, I allowed the error terms at level-3 to vary (opposed to fixing the variance) on the intercept and slope, to account for this within neighborhood variation. However, I fixed the neighborhood variation for all cross-level interactions tested in the models, as my model did not have sufficient power to simultaneously calculate four or more random variances.

## Results

Bivariate correlations of the measures are displayed in Table 3.2. John Henryism was associated with all promotive and risk factors as well as with self-rated health. Individuals who engaged in more high effort coping used fewer substances, reported more social support, and higher self-rated health. Using more substances was associated

with lower social support and sources of social support were all related. Individuals who reported more daily hassles engaged in less high effort coping and more substance use, and reported less social support.

**Cortisol reactivity (level-1)**

Based on a fully unconditional model (FUM), which contains no individual or neighborhood predictors, individuals' initial cortisol levels and average growth rates are positive and associated with cortisol reactivity. Estimation of the neighborhood-level variation indicates significant variability among neighborhoods for initial cortisol levels $[\chi^2=55.76\ (35),\ p<0.05]$, and for change in cortisol $[\chi^2=49.86\ (35),\ p<0.05]$. In this model time is centered on zero, which corresponds to the initial cortisol sample collected at the beginning of the interview. The linear time factor is interpreted as the change in cortisol over each sampling time (slope), and for young adults in the sample, cortisol increased linearly over time ($\beta=0.03$, $SD=0.01$, $p<0.01$). Next, a quadratic time factor was added to the model to determine whether the trajectory of cortisol included a curvilinear component. This term was not significant, however, and was removed from the model. The final level-1 model contained only the intercept (initial cortisol) and the slope (linear change in cortisol) (Table 3, Model 1).

**Individual-level model (level-2)**

Next, I included individual-level fixed effects in the model at level-2 to determine whether initial cortisol levels (intercept) or change in cortisol (slope) were explained by demographic, risk, promotive, or neighborhood perception factors that are unique to the individual. I also controlled for saliva sampling time at level-2. Using a step-down modeling approach I first entered all demographic and control variables into the model,

and only variables that were associated with the dependent variable were retained (Raudenbush & Bryk, 2002). SES was associated to cortisol reactivity (slope), but not baseline levels; thus I retained it for all subsequent analyses. Next, the risk and promotive factors were entered into the model and retained only if significant. The final model is displayed in Table 3.3 (Model 1).

The time at which the first saliva sample was taken was associated with the initial cortisol level for the sample ($\beta$= -0.00, $SD$=0.00, $p$=0.01), but not with cortisol reactivity. This variable was retained in the model as a control for sampling time. None of the demographic factors were associated with baseline cortisol. Family SES for those individual's with families in highest 75th percentile of SES was associated with cortisol reactivity, such that having a high versus average family SES resulted in a reduction in cortisol reactivity ($\beta$=-0.02, $SD$=0.01, $p$<0.05). No additional demographic factors were associated with cortisol reactivity, and were therefore dropped from the model.

Next the intra- and interpersonal risk and promotive factors were entered into the model. In initial exploratory analysis I found that the relationship between the risk and promotive factors and cortisol was curvilinear. Similarly to standard multiple regression, HLM assumes a linear relationship between the predictors and dependent variable, and incorrectly assuming a linear relationship may result in biased results. Thus, I created three group categorical variables for each risk and promotive factors (e.g., low, average, high) and included the low and high predictors in the model and used the average level as the reference category. All predictors were retained if they were significant at a value of $p\leq0.10$, or if the other level of the predictor was significant (e.g., high stress would be retained if p=0.05 for low stress, regardless of p-value for high stress).

Daily hassles, which is a measure of perceived stress, was marginally associated with baseline cortisol such that individuals who reported high versus average levels of hassles had higher baseline cortisol ($\beta$=0.06, $SD$=0.04, $p$=0.09). The variable representing experiencing fewer daily hassles was not significant. High effort coping was also related to baseline cortisol in our sample. Individuals who reported high versus average levels of high effort coping had higher baseline cortisol than their average coping peers ($\beta$=0.0, $SD$=0.03, $p$=0.018). Low (versus average) peer support was also marginally significant in predicting cortisol reactivity, and was therefore retained in the final models. Individuals who had low peer support had higher cortisol reactivity than their counterparts with average support ($\beta$=0.03, $SD$=0.02, $p$=0.076). Finally, daily hassles were significantly related to cortisol reactivity. Participants with fewer daily hassles had less reactivity than those with average daily hassles ($\beta$=-0.03, $SD$=0.01, $p$=0.015), as did participants who reported more daily hassles ($\beta$=-0.03, $SD$=0.01, $p$=0.017).

Although addition of the individual-level predictors improved the overall fit of the model [$\chi^2$=27.34 (12), $p$=0.01], significant variation in initial cortisol and change in cortisol based on differences between neighborhoods still remains [$\chi^2$=66.49 (35), $p$=0.001; $\chi^2$=59.64 (35), $p$=0.006]. Based on the model comparison test, as well as the theoretical and statistical significance of the individual-level predictors, they were retained in all sequential models.

**Neighborhood disadvantage model (level-3)**

Neighborhood indicators of social and economic disadvantage were included in the final (level-3) model to determine whether a portion of the random variation

remaining in cortisol reactivity after including level-1 and level-2 variables is attributed

to between-neighborhood variations. Each indicator of neighborhood disadvantage was

individually entered in the model at level-3 on the intercept and the slope for cortisol to

identify differences in initial cortisol levels or change in cortisol (direct relationships)

based on the level of disadvantage in a neighborhood. Living in a neighborhood with a

higher proportion of individuals detached from the labor force resulted in higher baseline

cortisol for residents, controlling for cortisol sampling time, SES, perceived stress and

high effort coping [$\beta$=0.32, $SD$=0.12, $p$=0.011]. The addition of the neighborhood

indicator explained a significant proportion of the variance in cortisol [$\chi^2$=3.92 (1),

$p$=0.045], although significant variation remains. None of the remaining neighborhood

disadvantage indicators was directly related to baseline cortisol or cortisol reactivity.

**Cross-level interactions between neighborhood disadvantage and fixed effects**

Finally, I tested interactions between the significant risk and promotive factors in

my model and the indicators of neighborhood disadvantage for both baseline cortisol and

cortisol reactivity. My results suggest a moderating relationship between John Henryism

(high effort coping) and baseline cortisol for neighborhood disadvantage. Individuals

living in neighborhoods with a high proportion of vacant houses, and who demonstrated a

higher level of John Henryism compared to average levels in our sample, had

significantly higher baseline cortisol levels than their average high effort coping peers

living in neighborhood with an average number of vacant houses [$\beta$=1.55, $SD$=0.75,

$p$=0.043] (Figure 3.3). I also found a similar relationship based on the neighborhood-

level education. Individuals in our sample who reported high John Henryism, and who

lived in neighborhoods with the largest percentage of residents with less than a high

school education, had the highest baseline cortisol [$\beta$=0.351, $SD$=0.18, $p$=0.054]. Public

assistance usage in a neighborhood also interacted with John Henryism to increase

baseline cortisol, although it was significant at only $p \leq 0.10$. Despite not meeting my a

priori level of statistical significance, this cross-level interaction is qualitatively similar to

that of John Henryism and the other neighborhood disadvantage indicators, suggesting a

consistent pattern.

Neighborhood-level indicators of disadvantage also interacted with an individual-

level factor to influence cortisol reactivity. Neighborhood poverty moderated the

relationship between an individual's report of experiencing high (versus average) daily

hassles and their cortisol reactivity. While an individual's high versus average perceived

stress level, as measured by daily hassles, was associated directly with cortisol reactivity

such that people with the highest level of perceived stress had lower reactivity than their

peers with average levels of perceived stress, the relationship between stress and cortisol

reactivity was further accentuated by neighborhood-level poverty. Individuals who lived

in neighborhoods with a high proportion of households earning less than $15,000 per

year, and who also reported a high number of daily hassles, had significantly reduced

cortisol reactivity over the three sampling times compared to their counterparts with an

average number of hassles living in neighborhoods with an average number of

households earning less than $15,000 per year [$\beta$=-0.122, $SD$=0.06, $p$=0.030]. Similarly,

those individuals living in neighborhoods with a higher proportion of households using

public assistance (Figure 3.4), and who reported high levels of daily hassles, had reduced

cortisol reactivity compared to their counterparts with an average number of daily hassles

and who lived in neighborhoods at the mean for the percentage of families on public

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assistance [$\beta$=-0.317, $SD$=0.15, $p$=0.036]. Finally, the same relationships emerged when neighborhood-level education was tested as a measure of disadvantage. Individuals who lived in neighborhoods with a high proportion of residents with less than a high school education, and who reported above average daily hassles, had less cortisol reactivity compared to individuals who reported an average number of daily hassles and who lived neighborhoods with an average number of individuals with less than a high school education [$\beta$=-0.138, $SD$=0.04, $p$=0.002].

Although I found several significant cross-level relationships between indicators of neighborhood disadvantage, individual-level risk and promotive factors, and cortisol, significant between-neighborhood variation in cortisol remains. The overall fit of the models including these neighborhood-level predictors, however, improved the fit of the individual-level (level-2) model (Model 1) based on model comparison, in all cases except the model that contained the neighborhood education predictor.

**Discussion**

The results of this study support the biopsychosocial model, theories of stress and coping, and neighborhood models as they relate to neighborhood disadvantage and cortisol. Individuals who lived in more stressful neighborhoods, as measured by socio-economic disadvantage, have atypical cortisol patterns compared to individuals living in neighborhoods with fewer stressors (less disadvantage). In addition, the relationships between coping and cortisol, and between perceived stress and cortisol, were moderated by the degree of neighborhood disadvantage. This supports the biopsychosocial model and Transactional Theory of Stress and Coping, which reveal complex relationships between stressor exposure, stress, and health that include psychological, coping and

physiological processes. Although only some of the neighborhood disadvantage indicators tested in my models were associated with cortisol, the results are consistent across these indicators in the direction of their relationship with baseline cortisol and cortisol reactivity. Similarly, the synergistic interaction between neighborhood stressors and individual-level predictors consistently indicate that both high effort coping and perceived stress are dependent on an individual's level of neighborhood disadvantage in their effect on cortisol.

This research contributes to the continuously growing body of literature of neighborhood effects on health, by addressing the way in which neighborhood stressors are associated with cortisol for Black Americans living in disadvantaged, urban areas. Numerous studies support the relationship between neighborhood context and stress (Aneshensel, 2008; Schulz et al., 2000), and research also indicates that an atypical cortisol response is associated with health problems including cardiovascular disease, diabetes, depression and obesity (Miller, et al., 2007). To date, only a handful of researchers have connected neighborhood context with cortisol as a physiological biomarker for stress (Chen & Paterson, 2006; Do, 2011; Dulin-Keita, Casazza, Fernandez, Goran, & Gower, 2012; Kliewer, et al., 2008), and of these studies, one combines neighborhood and individual risk (Kliewer, et al., 2008). My research fills a major gap in the literature because it includes neighborhood- and individual-level risk factors, and multiple cortisol measurement.

Interpreting the relationships between stressor exposure and cortisol, and between cortisol and health is not well understood or straightforward. What is *normal* for cortisol is dependent on the specific stressor; the quantity and duration of the exposure; individual

demographic, health, and behavioral factors; individual vulnerability and assessment (Dowd et al., 2011; Miller, et al., 2007). Despite this uncertainty, researchers believe that elevated cortisol levels in the morning indicate an unhealthy response to stressor exposure (Dowd et al., Almeida, et al., 2009; JenniferB. Dowd, et al., 2009), an increased cortisol awakening response (CAR) is related to chronic stress (Fries, Dettenborn, & Kirschbaum, 2009), and a flatter cortisol response (blunted sensitivity) (whether hyper- or hypo- regulated) also suggests an atypical stress response (Almeida, et al., 2009; Dowd et al., 2009). In this research cortisol was only measured at the beginning of the interview (this represents cortisol levels 20-30 minutes prior to sampling), and two time points following the baseline assessment, thus I cannot interpret waking cortisol or the CAR as they relate to stressor exposure.

Although the baseline cortisol reading in this study was not a measure of waking cortisol, it is intended to capture an individual's cortisol level prior to the interview stressor, and thus the individual's baseline stress level. This approach has been used in prior research (Fernald & Gunnar, 2009). Researchers suspect that individuals who are chronically exposed to stressors have atypical basal cortisol (McEwen & Seeman, 1999), although the direction of the aberration is not consistent across studies (Do, 2011; Dowd et al., 2011; Fernald & Gunnar, 2009; Lupien, King, Meaney, & McEwen, 2001). Controlling for sampling time, I found that individuals who lived in neighborhoods where more people were disconnected with the labor force had higher baseline cortisol. This may therefore suggest that these individuals have chronically elevated cortisol levels compared to their peers living in neighborhoods with more people involved in the labor force. Several researchers have also found that exposure to stressors resulted in higher

cortisol (Fernald & Gunnar, 2009; Lupien, et al., 2001; van Eck, Berkhof, Nicolson, & Sulon, 1996). Van Eck et al. (1996) found, for example, that individuals who experienced more stressful daily events had elevated cortisol levels compared to individuals who experienced fewer stressful events, and Lupien et al. (2001) reported higher salivary cortisol for children ages 6 to 10 from lower SES vs. higher SES families (although this relationship did not hold for children 12-18). Chronically elevated cortisol levels have been connected with cardiovascular disease, diabetes, obesity, psychiatric disorders and overall health (McEwen & Seeman, 1999; Miller, et al., 2007)

The biopsychosocial model relates stressors with psychosocial and behavioral responses, and coping, and physiological stress. Based on this framework I expected to find an association between neighborhood disadvantage and cortisol. Coping mechanisms, and psychological, behavioral and demographic factors, were also expected to mediate and moderate this relationship, as proposed in the framework. Although only neighborhood-level labor force participation was directly associated with baseline cortisol, I found that neighborhood disadvantage and high effort coping interacted to affect cortisol across several indicators of disadvantage. Individuals, who reported high, compared to average John Henryism, and who also lived in neighborhoods with the highest degree of disadvantage, had significantly higher baseline cortisol levels than their peers who lived in neighborhoods with less disadvantage. This relationship held true for the percentage of vacant housing in a neighborhood, the percentage of residents in a neighborhood with less than a high school education, and the percentage of residents in a neighborhood on public assistance. Thus, individuals who constantly strive to overcome a challenge and who persist in their coping, and live in neighborhoods with the highest

disadvantage, have significantly higher cortisol at baseline than their counterparts in less disadvantaged neighborhoods.  These results support Sherman James' hypotheses and research, where he has consistently found that Black and African Americans who have low SES and high levels of John Henryism (James, 1994; James, Keenan, Strogatz, Browning & Garrett, 1992; James, Strogatz, Wing & Ramsey, 1987), have an elevated risk of hypertension and higher diastolic blood pressure than low SES Blacks with low levels of John Henryism (James, 1994; James, Hartnett & Kalsbeek, 1983).  Atypical cortisol levels, which are related to the incidence of hypertension and cardiovascular disease (Miller, et al., 2007), may also be negatively affected by the interaction between risk and John Henryism.

The synergistic effect that emerged between neighborhood stressor exposure and high effort coping lends more support to the biopsychosocial model and Transactional Theory of Stress and Coping, which suggests that coping mediates or moderates the relationship between stressor exposure and the physiological expression of stress (and future disease).  To my knowledge, only one researcher has simultaneously examined stressor exposure, John Henryism and physiological stress.  Schmeelk-Cone et al. (Schmeelk-Cone et al., 2003) examined multiple measures of SES for young adults (using the same data used in the present study) and cortisol, and found that John Henryism moderated the relationship between job category (e.g., blue collar, student, unemployed) and cortisol.  Students who were on the low end of the John Henryism scale, had higher cortisol compared to their peers in blue collar or support staff jobs, although job type did not have an effect on cortisol for youth with higher levels of John Henryism (Schmeelk-Cone et al., 2003).  The authors conclude that John Henryism may

be protective for higher educated African-Americans. The stressor or risk factor used in the current study, however (neighborhood disadvantage), may function differently than job type, such that having high John Henryism and living in the most disadvantaged neighborhoods is harmful to health. Individuals who constantly strive to get ahead in life and work hard to overcome the odds, and who also live in the most disadvantaged neighborhoods where upward mobility is limited (Mayer & Jencks, 1989; Wilson, 1987), may have high baseline cortisol levels due to the dissonance between actions (high effort coping) and results (what the individual actually achieves) (Sellers & Neighbors, 2008). Although my results differ from those of Schmeelk-Cone et al. (2003), the John Henryism hypothesis substantiates the idea that individuals who engage in more high effort coping may have higher cortisol levels (James, 1994).

Although I found that individuals who lived in the most disadvantaged neighborhoods had higher levels of cortisol on average, other researchers have found that exposure to higher neighborhood or individual socio-economic disadvantage resulted in decreased cortisol levels (Chen & Paterson, 2006; Dowd et al., 2011; Dulin-Keita, et al., 2012; Kliewer, et al., 2008). Dulin-Keita et al. (2012) report that while exposure to acute stressors may result in increased cortisol levels, chronic stressor exposure may be associated with lower cortisol secretion. These discrepant results suggest inconsistencies in measurement, methodology and theory, as well as the possibility that a measure of baseline cortisol, or using a single measure of cortisol may not be sufficient as a marker of physiological stress. Several researchers, therefore, assessed cortisol reactivity, using multiple cortisol measurements to characterize the stress response (Buske-Kirschbaum, et al., 2003; Chandola, Heraclides, & Kumari, 2010; Diez-Roux, 1997; Do, 2011; Dowd et

al., 2011; Fernald & Gunnar, 2009; Gustafsson, et al., 2010; Hajat, et al., 2010; Mair, Diez Roux, & Morenoff, 2010).

To better assess an individual's reaction to a stressor (the interview in the case of our research), I also examined the relationship between intra- and interpersonal risk and promotive factors, neighborhood disadvantage, and cortisol reactivity. In response to an acute stressor, cortisol is excreted in conjunction with other hormones as a part of the stress response. Under normal conditions, cortisol peaks in response to stress, and then quickly returns to baseline; researchers hypothesize, however, that cortisol fails to peak and decline normally in individuals who are chronically exposed to stressors (Almeida, et al., 2009; Djuric et al., 2008; Lupien, et al., 2001). In some individuals cortisol reactivity is blunted (the body is not sensitive to the fight and flight response), which may indicate an abnormal response and result in health problems like hypertension, obesity, or cardiovascular disease after prolonged exposure to stressors (Djuric, et al., 2008; McEwen & Seeman, 1999). Due to the limited number and timing of cortisol measurement in this study I was not able to capture the full cortisol cycle, and thus cannot characterize the decline in cortisol post-stressor exposure.

My research does, however, indicate that individuals exposed to more stressors or those who report more perceived stress, had a blunted cortisol response during the interview. This is suggestive of the physiological desensitization of individuals who are chronically exposed to stressors. Ganzel et al. (2010) refer to the long term wear on the body due to stressor exposure (expressed as a blunted cortisol response in this research) as "the cost of physiological accommodation to environmental demand (p. 134)." When the body is forced to constantly fight against demands (i.e., neighborhood disadvantage),

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and the physiological stress response is chronically activated, the natural stress defense system begins to wear down, resulting in a desensitized or blunted response, and eventual harm the body.  My results are consistent with much of the reactivity literature, which finds that individuals who experience chronic stressors including low SES (Hajat, et al., 2010), neighborhood violence (Do, 2011), workplace stressors (Chandola, et al., 2010), cumulative stress (Suglia et al., 2010), family history of alcoholism (Sorocco, Lovallo, Vincent, & Collins, 2006), and laboratory or interview stressors (Buske-Kirschbaum, et al., 2003; Hankin, Badanes, Abela, & Watamura, 2010) experience a blunted or reduced cortisol response.

I also found, however that individuals who reported low levels of perceived stress, had a blunted cortisol response.  Although this may seem counterintuitive, it is possible that these individuals report low psychological stress because they are desensitized to stressors in their life.  Despite reporting little stress, these individuals may still experience physiological responses to living in disadvantaged neighborhoods, which influences cortisol reactivity and results in a blunted cortisol response to acute stressor exposure.  I did not find significant interactions between low levels of perceived stress and neighborhood disadvantage.  The curvilinear relationships I identified between perceived stress and cortisol reactivity, based on the level of stress, indicate that future research could include more thorough investigation of the relationship between varying degrees of psychological stress and cortisol reactivity.  It is also possible that different measures of psychological stress may yield different results, as the daily hassles measure used in this analysis most likely assesses current feelings of stress.

Perceived stress was directly associated with a reduced or flattened cortisol response, and individuals who reported higher psychological stress (versus average levels), and lived in disadvantaged neighborhoods, consistently had a diminished cortisol response to the interview stressor compared to their counterparts in less disadvantaged neighborhoods. These results further substantiate the biopsychosocial model, in which psychological factors like perceived stress are associated with the physiological stress process (e.g., cortisol). Although a few researchers have also examined the association between psychological stress and physiological stress, most do not find support for this relationship (Kudielka, Hellhammer, & Wüst, 2009). This may be a result of the timing of assessment of psychological and physiological stress, as the cortisol reaction is delayed approximately 20 minutes from stressor exposure, unlike a measure of perceived stress, which is immediate (Kudielka, et al., 2009). The measure of daily hassles used in this study might capture more chronic or regular stress perceptions, which may relate more strongly to cortisol. The significant interaction between an individual's experience of psychological stress and their neighborhood context supported by my research also suggests that living in a disadvantaged neighborhood increases the risk of physiological harm to the body beyond psychological factors alone.

I did not find evidence to support an association between social support and cortisol, substance use and cortisol, and neighborhood fear and cortisol. Although stress and coping theories identify social support as a critical intermediary in the stress process, it may not relate to physiological stress in the same way. It is also possible that our measures of social support and neighborhood fear assess more immediate support and perceptions, and that cortisol reflects accumulation of stressor exposure during childhood

and adolescence. Future research could consider various risk and promotive factors over time, and their changing effect on cortisol. Despite research indicating the relationships between substance use and blunted cortisol reactivity (Kudielka, et al., 2009), I also did not find support for this relationship. Most of the physiological stress research that includes substance use as a covariate also includes lifetime or abuse measures of substance use, and it is therefore possible that I was not able to detect a relationship between substance use and cortisol using past 30 day substance use measures.

Although this research suggests a critical link between neighborhood context and physiological stress, several limitations should be noted and addressed in future research. Most notably, the measurement of cortisol used in this study, while preferable to a single measure, is still not ideal for assessing the diurnal rhythm of cortisol in its entirety. Cortisol is best assessed over multiple time points each day, beginning at wakeup and extending to bedtime, over several days (Dowd et al., 2009). By examining cortisol reactivity over three closely-spaced sampling times, I was unable to assess the cortisol awakening response, multiple reactions to stress throughout the day, or the general decline of stress during the day. My measure of baseline cortisol, for example, may not actually represent the baseline level of an individual. This sample was not taken at waking so it is impossible to assume that individuals did not experience acute stressors prior to the interview, or that participants in more disadvantaged neighborhoods did not differentially experience acute stressors. Researchers could consider more longitudinal measurement of both cortisol and stressor exposure.

Another limitation of this research is the measurement of neighborhood. First, I defined neighborhoods based on census tract designations, which does not best

characterize natural neighborhoods for residents. Census tracts are rather large, and contain an average of around 4,000 people (U.S. Census Bureau, 2001), whereas residents would most likely define their neighborhood as an area within a one mile radius from their home, school or work. In addition, characterizing neighborhood stressor exposure with a measure of neighborhood socio-economic disadvantage may fail to capture more important structural, social and physical aspects of the neighborhood, which residents may identify with better than census-based indicators of disadvantage. Research that includes measures of crime and violence; physical neighborhood conditions and disorder; and neighborhood social capital and cohesion, has the potential to recognize additional stressors to which residents are regularly exposed and may relate more closely to cortisol. Despite this limitation, using a measure of socio-economic disadvantage is more likely to underestimate the effect of neighborhood context on cortisol rather than produce an inflated result. Therefore, researchers who more thoroughly assess neighborhood effects on cortisol may find stronger effects than those found in this study.

In this study I test theories that connect neighborhood stressor exposures to cortisol and health via social, psychological and behavioral pathways. The theoretical basis that informed my research helped me identify important risk and promotive factors, and also interactions between individual-level factors and neighborhood disadvantage that influence cortisol. Only two other studies include important covariates (Chen & Paterson, 2006; Do, 2011) related to neighborhood exposures and cortisol, however, despite theoretical connections between neighborhood exposures, individual behavior, coping, psychological factors and stress. This research fills a critical gap in the literature, and my results suggest that risk and promotive factors and cross-level interactions, should

be examined as factors that may intervene in the relationship between neighborhoods and physiological stress.

Another strength of this study is using three cortisol measurements to assess reactivity. Although researchers could ideally examine cortisol throughout the day for several days, by using three measures of cortisol this research extends beyond a single measurement, which enabled me to examine the change in cortisol during an interview stressor. Researchers have suggested that even two cortisol measurements result in a valid assessment of cortisol reactivity (Fernald & Gunnar, 2009), and thus the results from this study substantiate theories that individuals who are chronically exposed to stressors may have blunted cortisol reactivity when they experience acute stress.

Results from this research indicate that neighborhood socio-economic disadvantage affects baseline cortisol and cortisol reactivity to a stressor, beyond the effects of individual-level occupation, education, sex and health status. This suggests neighborhoods have an independent effect on cortisol, and possibly health, via a stress mechanism. In addition to directly influencing cortisol, neighborhood disadvantage also interacted with John Henryism and perceived stress to more strongly affect the stress response, which supports the biopsychosocial model and theories that posit that social, psychological and genetic/physiological interactions affect health. Understanding the effect of stressor exposure during adolescence and early adulthood is critical, as this period entails significant social and physiological changes, and sets the foundation for health later in life (Evans, et al., 2007; Goodman, 2005; Murali & Chen, 2005). In addition, non-clinical symptoms for cardiovascular disease, which is associated with atypical cortisol patterns (Do, 2011; Goodman, 2005), begin during adolescence

(Goodman, 2005).  My results, therefore, support early intervention and neighborhood improvements, as exposure to disadvantaged neighborhoods during childhood and adolescence may result in physiological stress and poor health in adulthood.

Programs aimed at improving neighborhood conditions including reducing poverty, unemployment; and improving physical conditions may help reduce stressor exposure and improve health. These programs might include job training, education, and crime reduction, as well as promoting positive social interactions within a neighborhood including crime watch groups and community social events.  By reducing residents' exposure to chronic stressors in their neighborhood, and helping them cope more effectively, it may be possible to reduce the constant activation of the stress response system and minimize physiological wear on the body.  Although previous research does not consistently support the relationship between neighborhood context and psychological stress (Brenner, Bauermeister, & Zimmerman, 2011), exposure to neighborhood disadvantage may have greater physiological versus psychological effects on health.  My results suggest that continued examination of the effects of neighborhood exposures on cortisol is warranted and may help better understand the relationships between context and health

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**Table 3.1: Sample description**

|  | N | Mean (SD)[a] | Range[b] |
|---|---|---|---|
| **Cortisol mean** | 165 | 0.43 (0.51) | 0-2.9 |
| **Cortisol reactivity** | 165 | | |
| Normal (decreasing) | | 98 (48.8%) | |
| Reactive | | 53 (26.4%) | |
| Non-reactive | | 50 (24.9%) | |
| **John Henryism** | 165 | 4.29 (0.51) | 2.67-5 |
| **Substance use** | 164 | 0.05 (0.81) | -0.62-2.92 |
| **Mother support** | 155 | 3.96 (1.02) | 1-5 |
| **Peer support** | 164 | 3.06 (1.05) | 1-5 |
| **Relationship support** | 162 | 4.44 (0.57) | 2.17-5 |
| **Health (self-rated)** | 165 | | |
| Healthier than others | | 143 (21.1%) | |
| Same as others | | 464 (68.5%) | |
| Less healthy than others | | 70 (10.3%) | |
| **Neighborhood attitudes** | 164 | 2.51 (0.81) | 1-4 |
| **Fear of neighborhood violence** | 165 | | |
| Positive | | 406 (79.1%) | |
| Negative | | 107 (20.9%) | |

[a] Categorical variables are reported as frequency(%)
[b] The range is reported only for continuous variables

**Table 3.2: Correlations between individual-level study variables**

| | Mean Cortisol | Perceived stress | John Henryism | Substance use | Mom support | Peer support | Relation-ship support | Health | Neighbor-hood attitudes | Neighbor-hood violence |
|---|---|---|---|---|---|---|---|---|---|---|
| **Mean Cortisol** | 1.o | | | | | | | | | |
| **Perceived stress** | -0.035 | 1.o | | | | | | | | |
| **John Henryism** | -0.017 | **-0.386** | 1.o | | | | | | | |
| **Substance use** | -0.112 | **0.170** | **-0.120** | 1.o | | | | | | |
| **Mom support** | -0.046 | **-0.204** | **0.268** | **-0.108** | 1.o | | | | | |
| **Peer support** | -0.043 | **-0.130** | **0.180** | **-0.111** | **0.141** | 1.o | | | | |
| **Relation-ship support** | **0.146** | **-0.334** | **0.364** | **-0.159** | **0.445** | **0.218** | 1.o | | | |
| **Health** | -0.048 | 0.045 | **-0.124** | 0.036 | -0.087 | 0.032 | **-0.108** | 1.o | | |
| **Neighborhood attitudes** | -0.081 | **-0.200** | **0.223** | -0.006 | **0.168** | **0.259** | **0.192** | -0.056 | 1.o | |
| **Neighborhood violence** | 0.006 | **0.179** | **-0.147** | -0.018 | -0.068 | -0.009 | **-0.115** | 0.068 | **-0.157** | 1.o |

Bolded values denote statistically significant correlations (p<0.05)

**Table 3.3: Individual and interpersonal (fixed) influences on baseline cortisol and cortisol reactivity**

| | B (SE) |
|---|---|
| **FIXED EFFECTS** | |
| **Mean cortisol at baseline ($\beta_0$)** | |
| Intercept | 0.155 (0.03)*** |
| Start time | -0.00 (0.00)*** |
| SES (continuous) | NS |
| Perceived stress (low) | NS |
| Perceived stress (high) | 0.062 (0.04)* |
| High effort coping (low) | 0.056 (0.03)* |
| High effort coping (high) | 0.07 (0.03)** |
| **Cortisol reactivity ($\pi_1$)** | |
| Intercept | 0.043 (0.01)*** |
| SES (low) | NS |
| SES (high) | -0.02 (0.01)** |
| Peer support (low) | 0.028 (0.02)* |
| Peer support (high) | NS |
| Perceived stress (low) | -0.026 (0.01)** |
| Perceived stress (high) | -0.034 (0.01)** |

*$p \leq 0.1$, **$p \leq 0.05$, ***$p \leq 0.01$

**Table 3.4: Individual, neighborhood, and cross-level effects on baseline cortisol and cortisol reactivity ($\beta$ (SE))**

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| **Mean cortisol at baseline ($\beta_0$)** | | | | | |
| Intercept | 0.156 (0.03)*** | 0.165 (0.03)*** | 0.161 (0.03)*** | 0.158 (0.03)*** | 0.151 (0.03)*** |
| % households in tract not in labor force | | | | | 0.324 (0.12)*** |
| Start time | -0.00 (0.00)*** | -0.00 (0.00)*** | -0.00 (0.00)*** | -0.00 (0.00)*** | -0.00 (0.00)*** |
| SES (continuous) | NS | NS | NS | NS | NS |
| Perceived stress (low) | NS | NS | NS | NS | NS |
| Perceived stress (high) | 0.048 (0.03)* | 0.048 (0.03)* | 0.046 (0.03)* | 0.058 (0.04)* | 0.067 (0.04)* |
| High effort coping (low) | 0.056 (0.03)* | 0.056 (0.03)* | 0.060 (0.03)* | 0.055 (0.03)* | 0.062 (0.03)* |
| High effort coping (high) | 0.070 (0.03)** | 0.038 (0.02)* | 0.070 (0.03)** | 0.066 (0.03)** | 0.066 (0.03)** |
| % in tract on public assistance | | 1.166 (0.65)* | | | |
| % vacant houses in tract | | | 1.55 (0.75)** | | |
| % families with < HS Edu | | | | 0.351 (0.18)** | |

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**Table 3.4 Continued**

**Cortisol reactivity ($\pi_1$)**

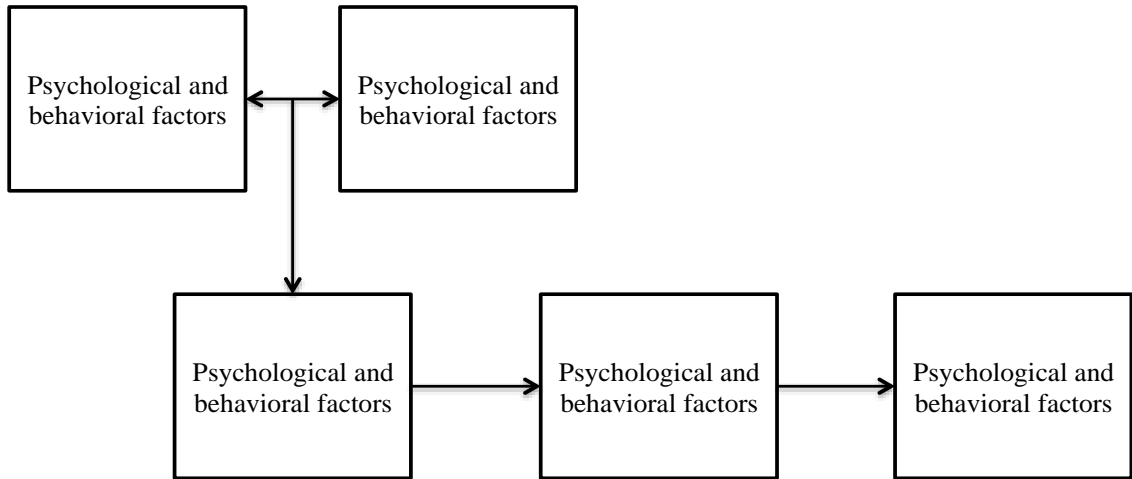| | | | | | |
|---|---|---|---|---|---|
| Intercept | 0.042 (0.01)*** | 0.043 (0.01)*** | 0.045 (0.01)*** | 0.043 (0.01)*** | 0.043 (0.01)*** |
| SES (low) | NS | NS | NS | NS | NS |
| SES (high) | -0.020 (0.01)** | -0.022 (0.01)** | -0.024 (0.01)** | -0.023 (0.01)** | -0.02 (0.01)** |
| Peer support (low) | 0.031 (0.02)** | 0.030 (0.02)** | 0.030 (0.02)** | 0.030 (0.02)** | 0.029 (0.02)* |
| Peer support (high) | NS | NS | NS | NS | NS |
| Perceived stress (low) | -0.025 (0.01)** | -0.025 (0.01)** | -0.022 (0.01)* | -0.026 (0.01)** | -0.025 (0.01)** |
| Perceived stress (high) | -0.034 (0.01)** | -0.031 (0.01)** | -0.037 (0.01)*** | -0.035 (0.01)*** | -0.033 (0.01)** |
| % in tract earning <$15,000/year | -0.122 (0.060)** | | | | |
| % in tract on public assistance | | -0.317 (0.15)** | | | |
| % vacant houses in tract | | | -0.312 (0.17)* | | |
| % families with < HS Edu | | | | -0.138 (0.04)*** | |

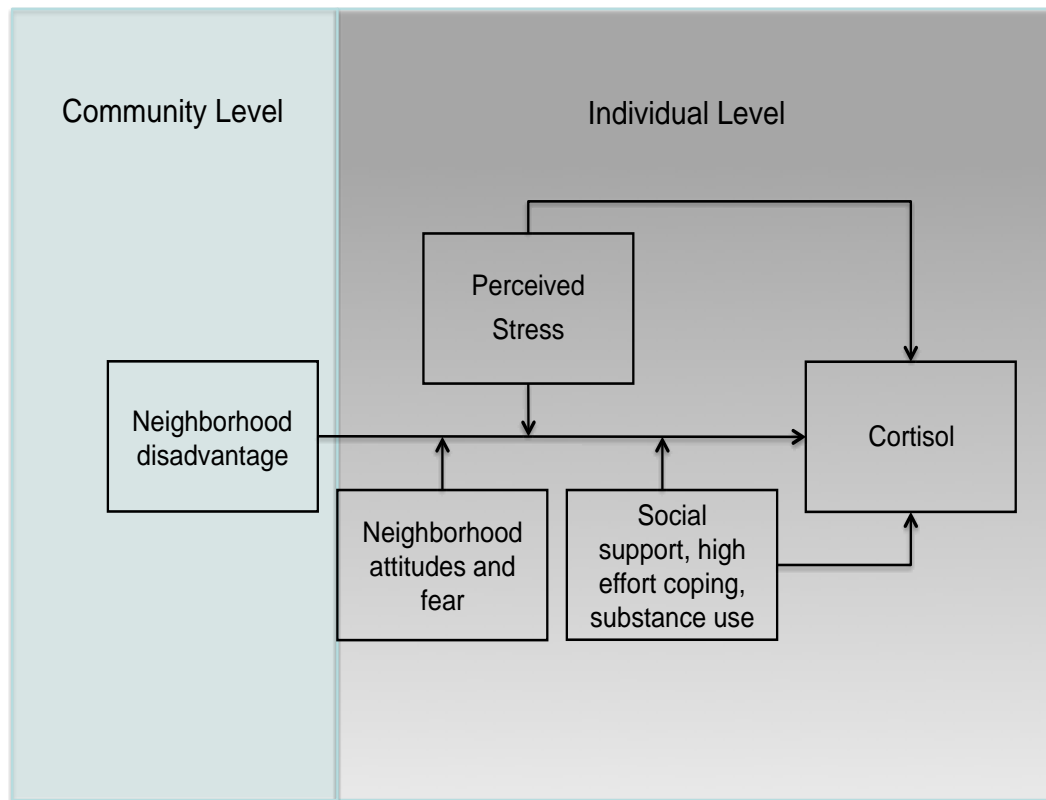*p≤0.1, **p≤0.05, ***p≤0.01

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**Figure 3.3 - Biopsychosocial Model**

**Figure 3.2: Conceptual model for neighborhood disadvantage and cortisol**

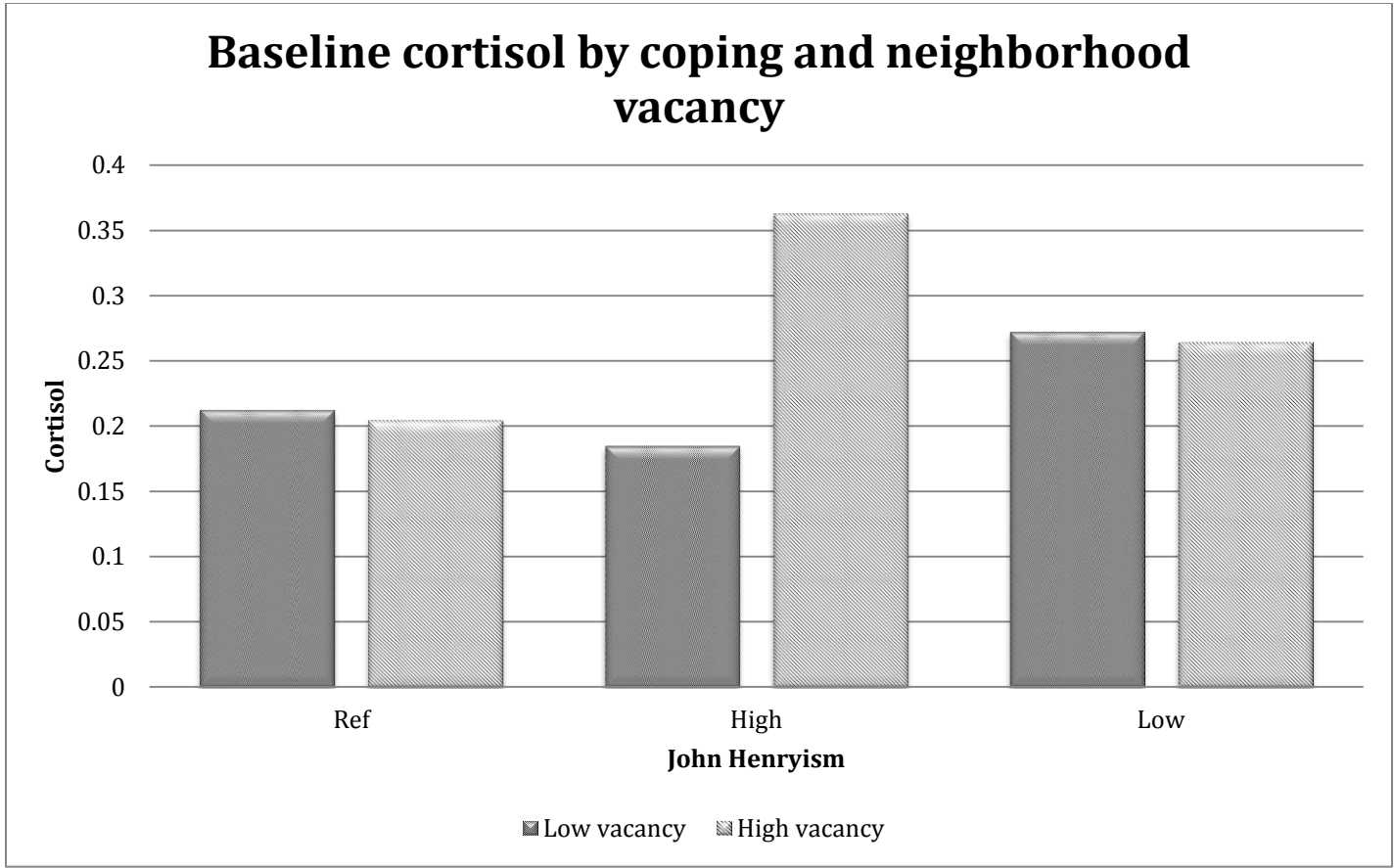**Baseline cortisol by coping and neighborhood vacancy**

Figure 3.3 - Baseline cortisol by coping and neighborhood vacancy
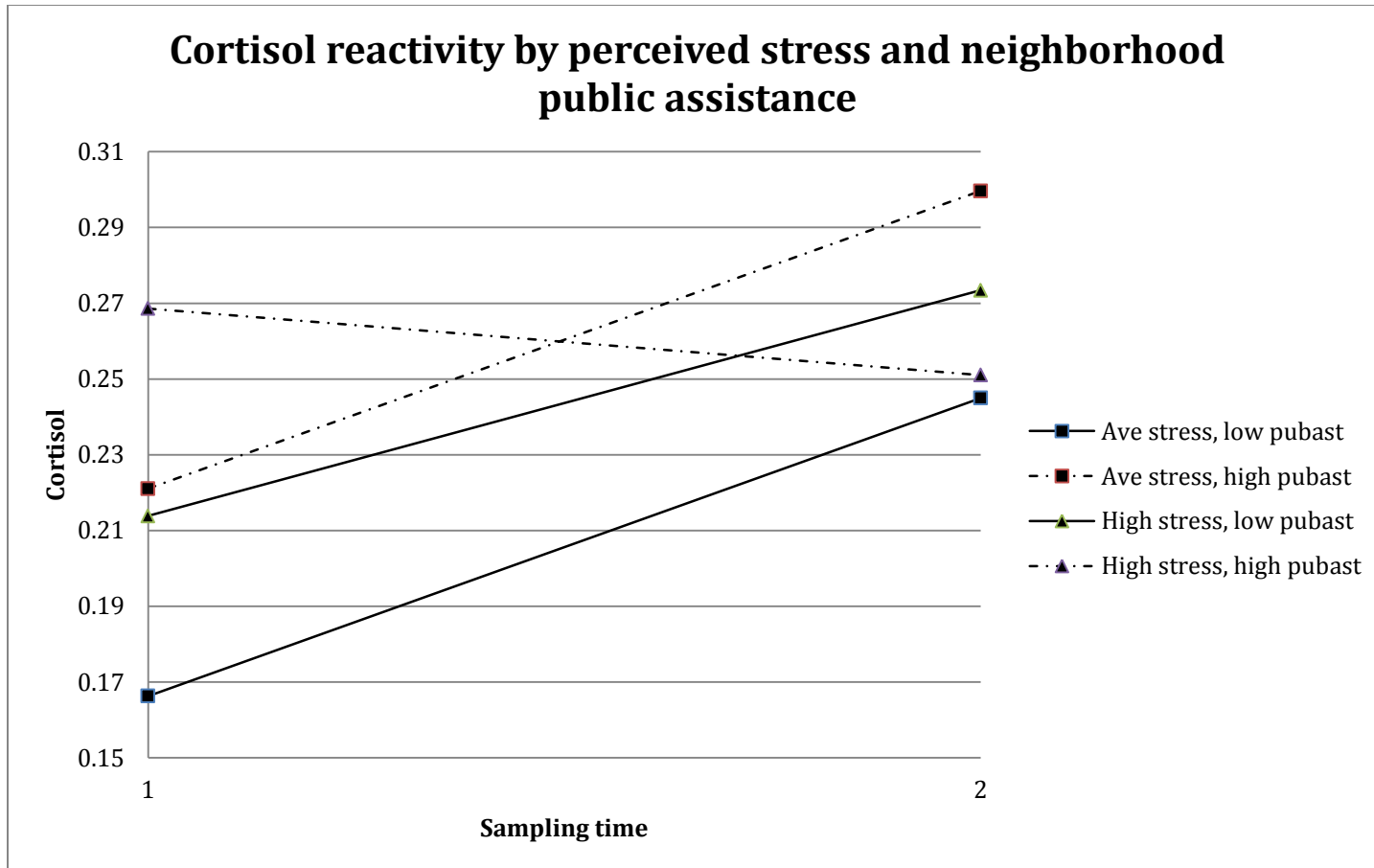
**Figure 3.4 – Cortisol reactivity by perceived stress and public assistance**

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**CHAPTER 4**

**WHAT IS IT ABOUT DISADVANTAGED NEIGHBORHOODS? UNRAVELING THE MECHANISM THROUGH WHICH NEIGHBORHOOD DISADVANTAGE AFFECTS HEALTH**

**Introduction**

Researchers have found that neighborhood context affects health beyond the influence of individual factors (Aneshensel, 2008; Diez Roux, 2001; Diez Roux & Mair, 2010). Individuals who live in neighborhoods with higher degrees of socio-economic disadvantage and segregation; more violence and crime; and lower levels of neighborhood social support and cohesion; experience worse health outcomes across a variety of health indicators (Diez Roux, 2001; Diez Roux, 2003; Diez Roux & Mair, 2010; Diez Roux, 1997; Pickett & Pearl, 2001; Yen & Kaplan, 1999). Particularly concerning, are the disparities in exposure to harmful neighborhood conditions patterned by race and socio-economic status (SES) (Diez Roux & Mair, 2010; Ellen, Mijanovich, & Dillman, 2001; Morenoff et al., 2007). Non-white, and lower SES individuals are more likely to live in communities with more economic, social and physical disadvantage (Massey & Denton, 1993), which may be a fundamental cause of mental and physical health inequalities (Diez Roux & Mair, 2010). Despite the growing number of studies that address neighborhood effects on health, the means through which neighborhood context affects health (and health inequalities) are still unclear (Ellen, et al., 2001). Thus, developing a more comprehensive understanding of the causal pathway, and the

individual- and neighborhood-level factors that mediate the pathway between neighborhood context and health, is critical in improving health and reducing health inequalities.

A variety of theories and conceptual frameworks connect neighborhood exposures and health. These include structural, social, and environmental stress models, social disorganization theory, and General Strain Theory (Agnew, 2001; Aneshensel, 2010; Sampson, Raudenbush & Earls, 1997; Shaw & McKay, 1969; Wandersman & Nation, 1998). Although these theories and neighborhood models are often examined separately, the complexities of the neighborhood context render it impossible to adequately explain the relationship between neighborhood exposures and health using just a single perspective (Aneshensel, 2010). Neighborhood factors influence and interact with inter- and intra-personal factors to affect psychological, behavioral, and physical health (Browning, Cagney & Wen, 2003; Cagney & Browning, 2004; Diez Roux & Mair, 2010), which makes the effect of neighborhoods even more complex. These intricacies, however, underscore the importance of working towards a deeper understanding of the means through which neighborhoods influence health.

**Neighborhood structure**

Structural neighborhood models typically connect socio-economic aspects of the neighborhood (e.g., poverty, unemployment, households headed by single parents) to mental health through social breakdown in the neighborhood (Aneshensel, 2010; Wandersman & Nation, 1998; Wheaton & Clarke, 2003). Wheaton and Clarke (2003) found, for example, that neighborhood socio-economic disadvantage during childhood exerted a direct effect on adult mental health. The relationship between neighborhood

disadvantage and health is often eliminated once individual socio-demographic factors are considered (Aneshensel, 2010), which suggests the relationship between neighborhood disadvantage and health may operate through other important mediating neighborhood and individual factors like neighborhood disorder, perceptions, social processes, individual behavior, stress and coping (Diez Roux & Mair, 2010; Ellen, et al., 2001; Yen & Kaplan, 1999).  Although disadvantaged neighborhoods are not always stress provoking, and may not lack social resources and support, they often result in social breakdown and disorder, which can be harmful to health (Aneshensel, 2010).

**Neighborhood stressors**

Neighborhood social and stress models, therefore, highlight the importance of the social factors that mediate the relationship between neighborhood disadvantage and health (Aneshensel, 2010; Wandersman & Nation, 1998).  Physical and social deterioration, or neighborhood disorder (e.g., crime, public drunkenness, abandoned buildings, vandalism), is hypothesized to result in social and psychological withdrawal, perceptions of fear, stress and poor mental health (Aneshensel, 2010; Ross & Jang, 2000; Ross & Mirowsky, 2001; Ross, Mirowsky, & Pribesh, 2001; Wandersman & Nation, 1998).  Researchers hypothesize that exposure to crime, and perceptions of fear and safety, may be a particularly important neighborhood stressor that is associated with health (Morenoff, 2003; Wandersman & Nation, 1998).

Exposure to neighborhood violent crime, and feelings of fear and safety in the neighborhood have notable effects on health (Gorman-Smith, 1998; Morenoff, 2003; Murali & Chen, 2005; Sundquist et al., 2006; Wright, 2006).  Sundquist et al. (2006) reported a 75% increase in the risk of cardiovascular disease for women living in

neighborhoods with the highest rates of violent crime, and a 39% increase in cardiovascular disease for men. Fear of crime also has significant effects on mental health (Perkins & Taylor, 1996; Wandersman & Nation, 1998); researchers have noted increased anxiety, alienation, withdrawal, and mistrust, which may result in a cycle of social withdrawal and breakdown, and increased crime in the community (Liska, Sanchirico, & Reed, 1988).

**Neighborhood social resources**

Social resources in the neighborhood may help combat the detrimental effects of socio-economic disadvantage and social disorder (Sampson et al., 1997; Wandersman & Nation, 1998). Researchers have found evidence for the benefits of social capital, informal and formal social control, social cohesion and ties to neighbors, and sense of community on mental and physical health (Cagney & Browning, 2004; Kruger, Reischl, & Gee, 2007; Mair, Diez Roux, & Morenoff, 2010; Morenoff, 2003; Ross, Reynolds, & Geis, 2000), although the effect may be moderated by aspects of neighborhood context including disadvantage and disorder (Caughy, O'Campo, & Muntaner, 2003; Morenoff, Sampson, & Raudenbush, 2001). High levels of neighborhood disadvantage and disorder may inhibit the formation or effectiveness of social resources in neighborhoods (Ross & Jang, 2000; Sampson et al., 1997), and neighborhood disorder may increase feelings of isolation, hopelessness, mistrust and alienation in the community (Ross & Jang, 2000; Ross & Mirowsky, 2009; Ross, et al., 2001), which are related to mental health problems.

Several researchers, however, report the beneficial effects of neighborhood social resources. Morenoff (2003) found, for example, that social exchange and volunteerism in a neighborhood mediated the effect of residential stability and violent crime on low birth

weight. Residents of neighborhoods with more neighboring (e.g., ask advice or receive help from neighbors; visiting neighbors) and interaction with neighbors, were buffered from the effects of socio-economic disadvantage on birth outcomes (Morenoff, 2003). Cagney and Browning (2004) reported a 6% reduction in the probability of asthma and breathing problems for residents of disadvantaged neighborhoods with high versus low levels of collective efficacy.  The authors hypothesize that higher levels of social control and cohesion in the neighborhood is influential in creating and advocating for a healthier neighborhood environment (Cagney & Browning, 2004).  Finally, Kruger et al. (2007) examined the effects of residential deterioration on individuals' mental health, and found that neighborhood social capital, assessed by measures of trust and helping neighbors, resulted in increased neighborhood satisfaction, and less fear of crime, stress and depressive symptoms.  Taken together, the evidence on social resources within a neighborhood indicates that positive social factors may protect residents from physical and mental harm, or reduce the deleterious effects of living in a disadvantaged and disordered neighborhood.

Although the number of published studies including neighborhood context and health has increased over the past decade, (Diez Roux, 2001) several limitations constrain our understanding of the mechanisms through which neighborhoods affect health.  First, many researchers consider only objective (Brenner, Bauermeister, & Zimmerman, 2011; Browning et al., 2003; Mason, 2010; Morenoff, et al., 2007; Schempf, Strobino, & O'Campo, 2009; Wodtke, Harding, & Elwert, 2011), or subjective (Dupéré, 2007; Hill & Angel, 2005; Latkin & Curry, 2003; Ross & Jang, 2000; Ross & Mirowsky, 2009) neighborhood factors as causes of poor health, whether due to theoretical decisions or

methodological constraints.  Of those researchers who consider both types of neighborhood measures in their analysis (Browning & Cagney, 2003; Browning, Burrington, Leventhal & Brooks-Gunn, 2008; Burgard & Lee-Rife, 2009; Curry, Latkin, & Davey-Rothwell, 2008; Dupéré, 2007; Ross & Mirowsky, 2001; Ross, et al., 2000; Stockdale et al., 2007; Weden, Carpiano, & Robert, 2008), few extend the investigation of the pathways through which they affect health beyond simple mediation models (Dupéré, 2007; Seidman et al., 1998; Weden, et al., 2008).  Despite the common theoretical perspective utilized by researchers relating neighborhood socio-economic disadvantage to health via social processes in the neighborhood, or aspects of neighborhood social disorder, limited research includes a thorough analysis of these relationships.  Understanding the full (complex) pathway through which neighborhood disadvantage affects individual-level psychosocial, behavioral, and health factors is a necessary step towards developing intervention and policy to improve neighborhoods.

Connecting neighborhood exposures to distal health outcomes is another challenge we face in addressing neighborhood context and health.  Many researchers propose that neighborhood exposures are partially responsible for, or are associated with certain diseases or health outcomes through complex mechanisms.  Most of these studies do not, however, include an assessment of the full mechanism, and thus the rationale for the results is based purely on hypotheses and untested theory.  Commonly examined outcomes include cardiovascular disease (CVD), coronary heart disease (CHD), birth outcomes and even mortality (Diez Roux, Augustin, Glass, James, & Schwartz, 2008; 1997; Masi, Hawkley, Harry Piotrowski, & Pickett, 2007; Messer, Kaufman, Dole, Savitz, & Laraia, 2006; Morenoff, 2003; Sundquist, et al., 2006; Yen & Kaplan, 1999).

Messer et al. (2006) propose fear of crime as the critical psychological construct that connects neighborhood environment and birth outcomes, although they do not include a measure of fear in their analysis. Similarly, Sundquist et al. (2006) assert that their research is based on social disorganization and disintegration theory, but they examine only direct pathways between neighborhood crime and CHD; and neighborhood unemployment and CHD (estimated in separate models). Despite their discussion of the theoretical implications of neighborhood exposures on CHD, the authors did not test the theory. Thus, their support for the association between crime and CHD, and unemployment and CHD could be attributed to any number of neighborhood (or untested individual) factors. Although research assessing the effects of neighborhood on distal health outcomes is critical in determining whether neighborhood context is associated with, or influences health (controlling for individual factors), it does not elucidate the neighborhood and individual-level social, psychological, or behavioral mechanisms at play. By examining more proximal health outcomes or intermediaries to disease (e.g., depression, stress, diet, physical activity, self-rated health), it may be possible to identify the causal pathway through which neighborhoods influence specific health outcomes.

Of the researchers who do examine more proximal health outcomes, many omit an analysis of important intermediary pathways between neighborhoods and health (Mair, et al., 2010; Morenoff, et al., 2007; Mulia, Ye, Zemore, & Greenfield, 2008; Ross, 2000; Weden, et al., 2008; Wodtke, et al., 2011). Some of these intermediaries function at the neighborhood-level, and others at the individual-level, and researchers rarely test models that include both types of intermediary factors. Weden et al., (2008) for example, examine the influence of neighborhood affluence, disadvantage, and perceived

neighborhood conditions on self-rated health using a structural equation model. Although their study is one of few that include relationships between objective and subjective neighborhood factors and health using a causal analytic technique (SEM), Weden et al. (2008) test the effect of objective and subjective factors on health simultaneously, and they do not include additional neighborhood or individual factors that might connect their neighborhood exposures to self-rated health. The authors conclude that neighborhood disadvantage has a weaker relationship with health than neighborhood affluence or perceived conditions, because it has the smallest path coefficient. Weden et al. (2008) are not, however, able to tease apart the relationships between the neighborhood factors (aside from correlations), or explore the pathways through which certain neighborhood exposures influence other neighborhood factors to affect health.

Stockdale et al. (2007) conducted a more thorough analysis of the neighborhood exposure process and examined the effect of neighborhood economic disadvantage, neighborhood stressors, and neighborhood stress buffering mechanisms on alcohol, drug and mental health (ADM) disorders. They found that disadvantage was not related to ADM, but that violent crime arrests and promotive neighborhood factors; including the number of churches, neighborhood social interaction and social support; were related to ADM (Stockdale, et al., 2007). Authors concluded that the relationship between violent crime arrests and ADM was likely a result of residents perceiving their neighborhood to be dangerous, threatening and stressful. Although very plausible given neighborhood theories, the researchers did not include measures of neighborhood perceptions or stress in their analysis. Research that more fully captures all critical aspects of the mechanism

connecting neighborhood factors to health or well-being is necessary to more definitively understand the way neighborhood exposures influence individual health.

**The current study**

Few researchers thoroughly examine the relationship between neighborhood factors and health at both the neighborhood and individual level (Kruger, et al., 2007; Ross & Mirowsky, 2001, 2009; Ross, et al., 2000), and only Kruger et al. (2007) fully examines the multiple pathways connecting neighborhood context to health. To gain a clearer understanding of the processes through which neighborhood context influences health, my conceptual model includes both neighborhood- and individual-level mediating factors, as neighborhood exposures affect health through neighborhood processes and risks, and individual psychosocial and behavioral factors (Browning et al., 2003; Cagney & Browning, 2004; Diez Roux & Mair, 2010). Additionally, I conceptualize exposure to neighborhood stressors as a part of a larger stress process, as is commonly applied to the individual level (Pearlin, Menaghan, Lieberman, & Mullan, 1981). This may clarify the connections between neighborhood exposures and individual health, and aid in our understanding of how neighborhood factors influence health outcomes. By including a more proximal psychological construct in the model (stress), I avoid linking distal neighborhood exposures to health directly; stress provides a means through which neighborhood context and social processes may influence physical health. I also utilize neighborhood structural, social and stress models, and social disorganization, and stress and coping theories to examine the relationships between neighborhood disadvantage and health. To better connect neighborhood and individual factors that contribute to health,

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my research also addresses neighborhood crime and safety, and neighborhood social capital (or neighboring) as important mediating factors (Figure 4.1).

Based on social disorganization theory (Sampson & Groves, 1989) I expect a direct relationship between neighborhood disadvantage and perceived stress, as well as an indirect relationship through crime and safety, and neighborhood social capital or neighboring (Figure 4.1). I hypothesize that neighborhoods with more socio-economic disadvantage will have higher crime rates and more perceived fear (and less safety) as reported by residents. I also expect neighborhood disadvantaged to inhibit social capital within the neighborhood. These relationships are depicted in Figure 4.1 by direct paths from neighborhood disadvantage to crime and safety (hypothesized positive relationship), and social capital (hypothesized negative relationship). As outlined by stress and coping theories (Aneshensel, 2010; Pearlin, et al., 1981), residents of neighborhoods with more disadvantage and crime, and less safety and social capital, will report greater psychological stress. This is depicted by a direct positive relationship between disadvantage and psychological stress, as well as indirect relationships through crime and safety, and social capital (Figure 4.1). Finally, I expect residents of neighborhoods with more perceived stress, on average, to report worse self-reported health, which is indicated by the pathway between stress and health.

**Methods**

**Data**

Data from four different sources were used for this study. Individual level data on psychological stress and health were aggregated to the census block group level due to methodological and power constraints. Individual-level data were obtained from the Flint

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Adolescent Study (FAS), which is a longitudinal study of adolescent health and development. At the start of the study, 850 youth entering high school who were at risk for substance use and school dropout were sampled from several public high schools in Flint, Michigan. Eligible youth had an eighth grade grade point average (GPA) of 3.0 or below upon entering high school, and were free of emotional or developmental impairments, as determined by school counselors. The initial sample consisted of youth self-identified as African American (80%), White (17%) or Bi-racial (3%), and males and females were equally represented.

The FAS consists of four waves of data collected during the high school years (Waves 1-4; 1994-1997), four waves of data collected after high school (Waves 5-8; 1999-2002), and three waves of data collected in early adulthood (Waves 9-11; 2008-2011). Retention rates were generally high (90% from Waves 1 to 4, 75% from Waves 4 to 8, and about 60% from Wave 8 to 9). FAS data were collected through structured face-to-face interviews and a self-administered questionnaire, which contained the more sensitive items, and was distributed at the conclusion of each interview to facilitate confidentiality. This study was approved by the University of Michigan Institutional Review Board (UMIRB# H03-0001309).

In the current research, only the ninth wave of data collection (2009/2010) is used, as these data correspond to the 2009 neighborhood crime and community survey data. The FAS data (N=254), which contained a geographically coded address for each participant, were aggregated to the census block group level, resulting in 138 observations. Approximately 63% of the initial sample (non-aggregated) was female, 80% were African American, and they were 29.3 years old on average. Just under 14%

of FAS participants did not complete high school, and over 70% reported either just having enough money left at the end of the month to make ends meet, or not having enough money.

Neighborhood data were obtained from three sources: the 2000 U.S. Census, the Flint police department and the *Speak to Your Health!* Community Survey.  The *Speak to Your Health!* Community Survey is a survey of residents from Genesee County, Michigan that examines social determinants of health and addresses community member concerns to promote community change.  The survey was first conducted in 2003, and then repeated in 2005, 2007 and 2009.  *Speak to your Health!* is a collaborative effort between the Community Based Organization Partners (CBOP), the Genesee County Health Department, the Greater Flint Health Coalition, the University of Michigan-Flint and the University of Michigan-Ann Arbor, School of Public Health.

The survey collects a variety of information related to behaviors, attitudes, health and perceptions about the residential context from participants in Flint and Genesee County Michigan.  Individuals were randomly sampled by census tract (10-20 individuals per tract), which resulted in a total of 1862 Genesee County residents.  Approximately 19% of the sample identified as African American, 75% as White, and 6% as mixed race. A little over half of the sample was female (53%).  The Community Survey data were weighted to account for varying population size and response rates across census tracts, and differences between the survey sample and Genesee County population distributions by age and sex.  In this study I used data collected in 2009, which included data from 1609 residents from 380 different census block groups in Genesee County.  Participants were 48 years (SD=0.2) on average, 73% of the sample was female, 66% identified as

White, 27% as African American, and 47% of the sample earned less than $20,000 in 2009.

Data on all crime incidents that occurred in Flint, Michigan were obtained from the Flint police department for 2004-2009. These data were geocoded using ArcGIS based on address information collected by the police at each crime incident location, and crime counts were created based on geographical units. For the current study I limited the data to only violent crime (assault, homicide, robbery and forced sexual offenses) committed between 2004 and 2008, and aggregated the data to the census block group level. This resulted in violent crime data from 208 census block groups in Flint. On average in each block group, 128 assaults, 1 homicide, 16 robberies and 6 forced sexual offenses were committed between 2004 and 2008, although averages across block groups range greatly. Finally, 2000 U.S. Census data were obtained for the block group level, and linked by geocode to the FAS, Community Survey and Crime data. The final dataset consists of 136 census block groups in Flint, Michigan.

**Measures**

Bivariate correlations for the variables included in the models are presented in Table 4.1, and descriptions of the measures are presented below.

Neighborhood Disadvantage

Neighborhood socioeconomic disadvantage was measured using eight items from the 2000 U.S. Census including the percent of vacant houses in each census block group; the percentage of households in the census block group on public assistance, the percent with less than a high school education, with an unemployed head of household, with a head of household outside of the labor force, headed by a Black or African American,

headed by a single mother with children under 18, and the percent below 1.5 times the Federal Poverty Level (FPL).  Three parcels were created from the eight items, and items were distributed such that the one with the highest factor loading was assigned to parcel 1, the item with the second highest loading to parcel 2, the item with the next highest loading to parcel 3, and so forth.  Items in each parcel were then standardized around their means (mean=0, SD=1.0) and summed to create three indicators of neighborhood disadvantage (Parcel 1: $\alpha$=0.77; Parcel 2: $\alpha$=0.86; Parcel 3: $\alpha$=0.76).

<u>Crime and safety</u>

The crime and safety factor was assessed using three items: violent crime incidence, perceptions of crime, and safety while walking in the neighborhood.  A count of the number of violent crime incidents in each census block group was used to assess crime exposure.  Crime data were obtained from the Flint Police Department, and data on the incident date and location (address) of each assault, homicide, robbery, and forced sexual offense (violent crimes as defined by the FBI) were used in this research.  Incident address data were then geocoded to the census block group level using Geographical Information Systems (GIS), in ArcGIS software.  Due to the variation in crime rates in Flint between 2004 and 2008, a total count of each violent crime over the five-year period was used to obtain a more stable crime exposure variable.  To reduce the skewness and kurtosis of the non-normal violent crime data, a total count of all four violent crimes was created, and this value was transformed using the square of the violent crime count ($\alpha$=0.89).

Perceived crime was measured using two survey items from the *Speak to Your Health!* Community Survey, which were standardized (mean=0, SD=1) and then

averaged. The first item asked respondents how the crime in their neighborhood compared to that in other neighborhoods, and responses included: very high, high, about the same, low, and very low. The second perceived crime survey question asked participants how fearful they were of crime in their neighborhood, and responses included: very fearful, somewhat fearful, not very fearful, and not fearful. Higher values denote greater perceived crime and fear ($\alpha=0.82$).

Neighborhood safety was measured using two survey items from the Community Survey, which were also standardized and averaged. The first item asked participants how safe it was to walk alone in their neighborhood during the daytime, and the second item asked participants how safe it was to walk alone during the nighttime. Possible responses for both items included: very dangerous, somewhat dangerous, fairly safe, and completely safe. Higher values denote greater feelings of safety ($\alpha=0.87$).

Neighboring/neighborhood social capital

Three items constitute the neighboring factor included in the analysis, and each item was included in the Community Survey. The first item asked how often people in the neighborhood visit each other either inside or outside the homes, and responses include: never, rarely, sometimes, and often. The next two items asked participants to what degree they agreed with the following statements: "People in this neighborhood are willing to help their neighbors" and "People in this neighborhood can be trusted." Possible response range on a 4-point Likert Scale from 4=Strongly Agree to 1=Strongly Disagree. Each of the three items was transformed using the square of the value to reduce skewness and kurtosis. Higher values on each item and on the factor correspond to more neighboring and greater social capital in the neighborhood.

Perceived stress

Perceived or psychological stress is the primary pathway of interest, through which neighborhood context influences health. A shortened version of Cohen and colleagues' Perceived Stress Scale (PSS) (Cohen, Kamark & Mermelstein, 1983), which was designed to measure subjective (psychological) stress, was used in this analysis and aggregated to the census block group level. The scale assesses the degree to which people believe their lives to be "unpredictable, uncontrollable, and overloading (p. 387)" (Cohen et al., 1983). Although the original PSS included 14 items, the version included in the FAS only includes 13 of these items with the best psychometric properties. Sample items include, "In the past month, how often have you…": been upset because of something that happened that you didn't expect, felt that you had so many problems that you could not deal with them, and felt able to handle your personal problems (positive items were reverse coded). Possible responses ranged from 1=Never to 5=Very Often on a 5-point Likert Scale, where higher values correspond to a greater experience of psychological stress. To reduce the number of individual variables include in the models, four parcels were created from the thirteen original items based on random assignment of items with the highest and lowest factor loadings into each of the four parcels. Three of the resulting parcels include three items, and one parcel includes four items. Means of the parcel items were computed and used in the analysis, and values for two parcels were transformed using the square transformation to reduce the skewness and kurtosis of the measure. All parcels retained adequate reliabilities ($\alpha$ =0.54-0.84).

Health

The primary outcome of interest in this study is self-rated health, which was measured using two indicators included in the FAS, and aggregated to the census block group level. First, the survey asked, "on a scale from 1 to 7, 1 being not at all concerned and 7 being very much concerned, during the last month, how much has your health worried or concerned you?" Responses to this item were treated as continuous and range from 1 to 7, with higher numbers corresponding to more health concern. Standardized values were computed and used in the analysis. Health was also assessed by a survey item, which asked participants to compare their health in relation to others. Possible responses include: healthier than others, about the same and less healthy than others, where higher numbers correspond to rating health as less healthy. Since the data were relatively normally distributed, the item was standardized and used as a continuous variable in the analysis.

**Analytical Strategy**

Structural equation modeling (SEM) was employed to test the study hypotheses for several reasons. First SEM uses adjusted reliability estimates for the latent factors, which allows for a more accurate estimation of model path coefficients. Given the numerous potential indicators of neighborhood constructs in this research (e.g., disadvantage, social cohesion), SEM helps correct for the omission of important predictors when determining the path coefficients between latent factors. Second, SEM can also account for high degrees of multicollinearity between factors, which is often the case with measures of neighborhood context (Warner & Coomer, Gardner & Brooks-Gunn, 2009; 2003; Wen & Christakis, 2005). Finally, SEM is particularly useful for hypothesis testing and exploratory work, which is the focus of this research (Kline,

2005).  SEM allows for evaluation of an entire conceptual model instead of focusing only on the significance of coefficient estimates (Kline, 2005).  Holistic evaluation of the conceptual model through which neighborhoods influence stress and health is theoretically more relevant to understanding these largely unknown relationships.

The analysis was conducted in several stages, beginning with exploration of the data, in which I examined bivariate correlations between variables of interest.  The correlations suggest that multicollinearity will not present problems in the analysis, as correlations between indicators of the same factor were not greater than $r$=0.80, and correlations between indicators of different factors were generally low.  I also examined the normality of the distribution of each variable, and transformed variables as indicated to reduce skewness and kurtosis, as SEM assumes normal distributions of the data (Kline, 2005).

Next, I examined measurement models for the neighborhood crime and safety, neighborhood social capital/neighboring, neighborhood disadvantage, and stress latent factors, individually and with appropriate correlations with other factors to ensure that the variables adequately described the corresponding latent factor.  Analyses were conducted using EQS version 6.1 (Bentler, 2005) and the covariance matrices were analyzed.  Based on the results from the measurement models, as well as principal components analysis (PCA), existing literature, and theory, I determined the most parsimonious, theoretically relevant and best-fitting specification of each latent construct.  Reliabilities for each latent construct were examined to confirm that the indicators accurately reflected each latent factor.  Cronbach's alphas for most of the indicators are above 0.70, which suggests that they reliably reflect the factor presented in the theoretical model (Kline,

2005), although the scale reliability for health was low (~0.30).  Given that the two items

that reflect self-rated health are theoretically related, I include these in the analysis

despite the low alpha.  Maximum likelihood estimation was used to estimate all models

in this research, as several variables violated normality assumptions (despite

transformations), and robust goodness-of-fit indices and parameter estimates were used to

determine model fit.  Lastly, I examined a full structural model to examine causal

relationships between the neighborhood context, psychological stress and health.  The

structural model was informed by theory and by the measurement models.  Similarly to

the analysis of the measurement models, evaluation of model fit and parameter estimates

were based on robust statistics.

　　　　Model fit for both the measurement models and the SEM was assessed by

multiple goodness-of-fit statistics including the comparative fit index (CFI), the root

mean-square error of approximation (RMSEA) and the ratio of the Satorra-Bentler scaled

chi-square statistic to the model degrees of freedom (Raykov, Tomer, & Nesselroade,

1991).  The CFI is a measure of incremental fit that compares the independence (null)

and hypothesized models based on a chi-square distribution.  Although several measures

of incremental fit are presented in SEM results, some researchers prefer the CFI because

it does not assume that the hypothesized model has perfect population fit (Kline, 2005).

CFI values greater than 0.90 represent adequate model fit, and values greater than 0.95

suggest excellent fit.  The RMSEA is a measure of model misfit, which adjusts for model

complexity and does not assume perfect population fit of the hypothesized model.

RMSEA values <=0.05 suggest good fit, and values between 0.05 and 0.08 suggest a

reasonable error of approximation (Kline, 2005).  Finally, the ratio of the Satorra-Bentler

scaled chi-square statistic to the model degrees of freedom was used as a rough estimate of model fit, with a ratio of less than 2 suggesting an excellent model fit, and a ratio of less than 5 suggesting an adequate fit (Kline, 2005). Individual paths between latent factors were assessed using robust standardized parameter estimates.

**Missing data**

Complete data were used for the 2000 Census, although some corresponding data were missing from the FAS (N=1), Community Survey (N=10) and Crime (N=35) data. Missing data were imputed to retain adequate sample size and power, and the expectation maximization (EM) algorithm was used to impute missing data for violent crime (N=35), relative health (N=1), concern about health (N=1), neighborhood social capital (N=9), perceived crime (N=9), safety walking at night (N=10) (West et al., 2006). I conducted an attrition analysis comparing census block groups with missing (N=40) and complete data (N=98). The means and standard deviations of variables in the imputed data set and data set containing missing data were very similar, which indicates that data imputation did not result in large discrepancies.

Block groups with missing violent crime data consisted of younger residents (29.4 vs. 29.1, $t$=2.7, $df$=136), and residents reported having more money left at the end of the month (2.99 vs. 2.43, $t$=3.6, $df$=136). All non-violent crime data is assumed to be missing at random, however the census block groups with missing violent crime data for Genesee County may be different than the Flint block groups with non-missing data. I do not anticipate significant differences, however, as Flint is representative of Genesee County, and the missing data were not a result of sampling or attrition.

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<center>**Results**</center>

**Measurement model**

Results for the measurement models are presented in Figure 4.2 and Table 4.2. Figure 4.2 presents factor loadings for indicators of the corresponding neighborhood factor, and the correlations between neighborhood constructs. The neighborhood measurement model suggests that all of the variables adequately load onto their respective latent construct. All factor loadings are significant ($\alpha<0.05$), and except for crime and safety, are greater than 0.40, which implies that they accurately reflect the proposed construct. Although the coefficient for violent crime is slightly less than 0.4, it is significant and was retained in the model.

All of the factor loadings are in the hypothesized direction. The number of violent crime incidents within a census block group, and perceived crime in the neighborhood, are positively associated with the crime and safety factor, and reports of feeling safe walking in the neighborhood are negatively associated with crime and safety. The three parcels of socio-economic census indicators were positively associated with neighborhood disadvantage, and reports of trusting neighbors, the frequency neighbors visit each other, and helping neighbors were all positively associated with neighborhood social capital/neighboring. The factor correlations are also in the expected direction, and although they are moderately high, they are not so highly correlated as to suggest that the factors are not distinct constructs. Overall the measurement model fits the data well. The Satorra-Bentler scaled chi-square value is 30.12 ($df$=24, $p>0.05$), which suggests an adequate fit. Additionally, the model CFA is above 0.95 (CFA=0.99), and the RMSEA is less than 0.05 (RMSEA=0.044 (0.00, 0.086)), both of which also indicate a good fit for

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the data.  Taken together, the fit statistics and parameter estimates suggest that the model could be specified in a fully structural form.

Table 4.2 displays standardized factor loadings, as well as the error terms and R-squared coefficients for all variables included in the final model, and like the neighborhood measurement model, all coefficients are significant and most are greater than 0.40.  The standardized loadings for self-rated health are slightly less than 0.40, but given theoretical considerations they were retained in the model.  Each of the parceled indicators of daily hassles are positively associated with perceived stress, and the direction of the relationship between concern for one's health and relative health with the self-rated health factor was also positive as expected.

**Structural model**

Next, I proceeded to test my full structural model, which addresses the study objectives.  The model is displayed in Figure 4.3.  I expected that neighborhood disadvantage and violent crime and safety in the neighborhood caused more perceived stress for residents, and that living in neighborhoods with more social capital decreased stress.  The model also proposes an indirect effect of neighborhood disadvantage on stress via crime and safety and social capital.  I also hypothesized that greater perceived stress results in worse self-rated health.  Overall, the model represents the data well, as indicated by excellent goodness-of fit-statistics.  The Satorra-Bentler scaled chi-square for the model was 118.85 (*df*=83, *p*=0.01).  Although the significant p-value suggests that the model does not fit the data well, the ratio of the chi-square test statistic to the degrees of value is less than two (1.4), which is consistent with a good fit.  The CFI was 0.961

and the RMSEA was 0.06 (0.03, 0.08), both of which are either excellent or good fit

statistics supporting the model as an adequate fit for the data.

Figure 4.3 includes the standardized path coefficients for the structural portion of

the model. The measurement portion of the model, which is not presented, did not

change, and the factor loadings for perceived stress and self-rated health are included in

Table 4.2. A dashed arrow in the figure displays hypothesized path coefficients that were

not significant. Increased socio-economic disadvantage in the neighborhood resulted in

more crime and less safety ($\beta$=0.67), and less neighboring/neighborhood social capital

($\beta$=-0.45), as hypothesized. Despite significant relationships between disadvantage and

the other neighborhood constructs, neither crime and safety, or neighboring were sources

of perceived stress, as predicted. Neighborhood disadvantage did, however, directly

result in increased stress ($\beta$=0.31), although the path coefficient is relatively low and does

not explain a large proportion of the variance in the stress factor ($R^2$=~5%). Greater

levels of perceived stress for residents resulted in worse self-rated health ($\beta$=0.85), as

expected.

Although the results from this model are suggestive of the causal pathway

described in Figure 4.2, the results do not rule out reverse causation as a possible source

of the resulting path coefficients and relationships between factors. To determine

whether a model where poor health causes increases in stress, which then results in

harmful neighborhood processes and exposures, I ran the reverse model. The reverse

causation model did not fit the data as well as the hypothesized model (Satorra-Bentler

scaled chi-square = 210.70 (*df*=112*, p*=0.00); CFI=0.90; RMSEA=0.08 (0.06, 0.10)).

More convincing is the fact that the path coefficients between health and perceived stress;

and perceived stress and disadvantage; were not significant.  These results suggest that the causal mechanism proposed in the hypotheses is reasonable and supported by the model.

**Post-hoc analyses**

Despite the acceptable goodness-of-fit statistics, and significant path coefficients for the proposed model, the results indicate that only a very small proportion in the variance of the perceived stress construct was explained by the proposed factors (~5%). Additionally, the paths between neighborhood crime and safety and neighborhood social capital/neighboring, were not significant.  This suggests that the hypothesized model may be missing a factor that is critical in explaining residents' psychological stress. Research indicates that people's attitudes and perceptions of their neighborhood are important, and positive neighborhood experiences can help insulate residents from socio-economic disadvantage, daily experiences in the neighborhood, and possible subsequent perceptions of stress (Wandersman & Nation, 1998).  A neighborhood attitudes factor was therefore included in the re-specified model as a mediating variable (Figure 4.4).

Based on the results of the previous model, along with theoretical considerations of the processes through which neighborhood context affects psychological stress, I added neighborhood attitudes to the model to connect neighborhood context and stress (Figure 4.4) and re-specified the model.  The attitudes factor is composed of items from the Flint Adolescent Study survey, asking participants how they felt about living in their current neighborhood, and about various aspects of the neighborhood.  Participants were instructed to determine their level of agreement on a 4-point Likert Scale to five items. Sample items include: "I like living in my neighborhood" and "The friendships I have

with other people in my neighborhood mean a lot to me." Responses ranged from Strongly Disagree to Strongly Agree. Higher values on the items denote more favorable attitudes ($\alpha$=0.78).

Next, I tested the revised model based on re-consideration of the initial results and theory. It is important that I note that these results were obtained from a model that was not hypothesized a-priori. Yet, analysis of the post-hoc model suggests an adequate fit to the data. The Satorra-Bentler scaled chi-square for the model was 227.59 (df=161, p<0.01). Like the previous model, although the p-value is significant, the ratio of the test statistic to the degrees of freedom is around 2, suggesting a good model fit. The CFI (0.94) and RMSEA (0.06, CI (0.04,0.07)) also fall within the guidelines for a very good fitting model, as the CFI is just under 0.95 and the RMSEA is close to 0.05. In addition to comparing goodness-of-fit statistics for the original and revised model, I also examined Akaike Information Criterion (AIC) and the Consistent Akaike Information Criterion (CAIC) for both models as a means to compare non-nested models. Both AIC and CAIC are used for model comparison, and smaller values represent a more accurate fit of the data, while accounting for (penalizing) parsimony and sample size (CAIC). Both the AIC and CAIC for the model specified post-hoc, that include the neighborhood processes factor, were smaller that those of the hypothesized model ($AIC_{hypoth}$=-49.41, $CAIC_{hypoth}$=-374.16; $AIC_{pos-hoc}$=-94.41, $CAIC_{post-hoc}$=-721.96). Model comparison results suggest that the re-specified model that accounts for neighborhood processes is a better fit for the data.

The structural path coefficients also support that the model fits the data. As in the previous model, the current results indicate that neighborhoods with higher levels of

socio-economic disadvantage result in more crime and less perceived safety ($\beta$=0.68) as well as lower levels of social capital/neighboring ($\beta$=-0.45). Both pathways were statistically significant and in the expected direction. The direct pathway between neighborhood disadvantage and neighborhood attitudes was also significant and was negative ($\beta$=-0.34), which suggests that individuals living in more disadvantaged neighborhoods have more negative attitudes towards their neighborhoods. Socio-economic disadvantage explained about 8% of the variation in the neighborhood social processes factor, which is slightly higher than the explained variance in the hypothesized model. Crime and safety and social capital were not, however, directly related to neighborhood attitudes, despite their relationship with neighborhood disadvantage. These non-significant pathways are represented in Figure 4.4 by a dashed line. When the model was re-run without these pathways, the fit statistics and parameter estimates were unchanged, and the non-significant paths were retained, as they represent my original theory.

As hypothesized upon model respecification, individuals living in neighborhoods where residents reported more negative attitudes about their neighborhoods reported more perceived stress ($\beta$=-0.28), and as expected, these individuals reported worse health than their peers with lower stress levels ($\beta$=0.85). The addition of neighborhood attitudes did not have a large effect on overall model fit, but it did increase the proportion of variation in perceived stress explained by the model (~3% more). Finally, I examined the reverse causation model to rule out the possibility that poor self-rated health causes more psychological stress, which then affects neighborhood processes and resulting levels of socio-economic disadvantage. I did not find evidence to support reverse causation.

## Discussion

This research addresses the need to more fully explore the complex mechanisms through which neighborhood socio-economic disadvantage affects health. By examining a more thorough causal pathway connecting neighborhood context to global self-rated health, I was able to more comprehensively investigate the distal influence of neighborhood disadvantage on health, via neighborhood and individual mediating factors and processes. Many researchers have documented associations between neighborhood context and health (Culhane & Elo, 2005; Diez Roux et al., 2001; Diez Roux, 2003; Diez Roux, 1997; Ross & Mirowsky, 2001; Schempf, et al., 2009; Yen & Kaplan, 1999). While my results are consistent with this large body of literature, I applied neighborhood models and stress and coping theories to better understand the means through which this relationship occurs.

I began with a model where I hypothesized that neighborhood disadvantage would exert both direct, and indirect effects on psychological stress. Consistent with neighborhood structural models (Aneshensel, 2010; Wandersman & Nation, 1998), I found that socio-economic disadvantage was associated with more social breakdown in the neighborhood, as assessed by violent crime, perceived crime and safety. Additionally, the presence of neighborhood disadvantage resulted in residents reporting less neighboring and social capital in their communities, which is also consistent with the idea that socio-economic disadvantage may limit social interactions within a neighborhood (Aneshensel, 2010). In support of neighborhood structural models, I found a direct relationship between neighborhood socio-economic disadvantage and perceived

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stress, such that higher degrees of disadvantage resulted in more perceived stress. This is consistent with literature citing increased psychological stress for residents from disadvantaged neighborhoods (Aneshensel, 2010; Boardman, Finch, Ellison, Williams, & Jackson, 2001; Schulz et al., 2008; Steptoe & Feldman, 2001).

Although neighborhood disadvantage resulted in more crime and less safety, and less social capital in the neighborhood, these factors did not mediate the relationship between neighborhood disadvantage and perceived stress as predicted. In fact, crime and safety, and social capital or neighboring, were not directly associated with stress in my sample, which is contradictory to theory. Lack of either direct or mediating effects may be a result of methodological considerations in measuring the two constructs, or it may indicate the omission of important and more proximal factors in the model. It is also possible that in our sample there is no effect of crime and safety or neighborhood social capital on stress.

First, I included both violent crime and perceptions of crime and safety in the same factor, although objective and subjective reports of crime and safety may be theoretically very different concepts (Lewis & Maxfield, 1980). The violent crime indicator, while retained in the models, did not load as strongly onto the factor as measures of perceived crime and safety. It is possible that this weaker loading is indicative that neighborhood violent crime is distinct from perceptions of crime and safety, and that including it in the same construct may be obscuring important relationships. To test this hypothesis the objective crime measure was removed from the larger construct, leaving a factor that represented perceived crime and safety. Running the model with this new crime and safety factor did not change any of the results,

however, which suggests that the non-significant relationship between neighborhood crime/safety and stress may be due to theoretical concerns.

Although neighborhood crime and safety are expected to influence perceived stress, it is possible that this relationship is moderated by more proximal neighborhood or individual factors. Family context and social support may, for example, mediate or moderate the relationship between neighborhood crime and psychological stress. Gorman-Smith et al. (2004) found, for example, that family structure mediated the relationship between exposure to community violence and mental health. Youth from families with more social support and less tolerance for deviant behavior had more favorable mental health outcomes when exposed to violence than youth from less supportive families (Gorman-Smith, et al., 2004). Future research could include various psychological and behavioral mediating/moderating pathways, as they are important factors in the stress process (Pearlin, et al., 1981; Turner & Turner, 2005; Wenzel, Glanz, & Lerman, 2002).

I also expected social capital to be associated with stress, such that living in neighborhoods with more social capital and neighboring would help buffer residents from psychological stress. Kruger et al. (2007) found that neighborhood social capital reduced perceived stress in their sample, although the relationship was not very strong. My model did not support this hypothesis despite the negative relationship between disadvantage and social capital. One explanation for these unexpected results may be that high social capital and neighboring is not sufficient to overcome the effects of living in a neighborhood with a high degree of socio-economic disadvantage. Researchers suggest that the effect of various neighborhood and individual resources on health and behavioral

outcomes may be dependent on characteristics of the neighborhood, like the degree of disadvantage (Aneshensel, 2010; Caughy, et al., 2003; Dupéré, 2007; Kingston, Huizinga & Elliott, 2009; Schulz, et al., 2008; Warner & Wilcox-Rountree, 1997).

It is also possible that the social capital/neighboring construct I included in my model did not reflect the specific type of neighborhood social resources that are most beneficial in helping residents avoid stress. When I re-specified the model to include neighborhood attitudes as a more proximal measure of neighborhood resources, I found that it mediated the effect of socio-economic disadvantage on stress; neighborhoods where residents reported more positive perceptions of their neighborhood, also had lower levels of perceived stress on average. This is consistent with theories of stress and coping, which posit that psychological appraisals mediate the relationship between stressor exposure and psychological stress (Lazarus & Folkman, 1984). Although researchers have examined various neighborhood resources that may be beneficial for residents, the benefit of these resources is likely dependent on the type of neighborhood, characteristics of the individuals living in the neighborhood, and the specific health outcomes considered (Dupéré, 2007). Researchers have found, for instance, that high levels of social cohesion in neighborhoods with non-normative deviant beliefs may actually undermine social control and result in more crime and violence (Warner & Wilcox-Rountree, Dupéré, 2007; 1997).

While the model I initially hypothesized fit the data based on global goodness-of-fit statistics and significant path coefficients, neighborhood socio-economic disadvantage did not explain a large proportion of the variation in perceived stress. Additionally, crime and safety and social capital and neighboring, were not associated with stress as expected

based on theory. This may suggest that my model did not include all of the relevant pathways between disadvantage and stress. To address this concern, I re-specified my model based on theoretical considerations of the importance of neighborhood attitudes and perceptions as a mediating factor between neighborhood context and well-being. The re-specified model had approximately the same fit to the data, but explained slightly more of the variation in perceived stress. This is not surprising given the more proximal relationship between neighborhood attitudes and perceived stress, versus that between neighborhood disadvantage and stress. Researchers have found, for example, that individuals with more positive attitudes about their neighborhood, or those who report a greater sense of community, engage in more prosocial behaviors, are more involved in community social control, and report greater happiness and overall well-being (Kingston, Mitchell, Florin & Stevenson, Chavis & Wandersman, 1990; Davidson & Cotter, 1991; S. Kingston, Mitchell, Florin, & Stevenson, 1999). These factors may translate into greater community- and individual-level control and psychological empowerment, and less perceived stress (Chavis & Wandersman, 1990; Davidson & Cotter, 1991). Conversely, negative feelings towards the neighborhood, and a low sense of community may not adequately insulate individuals from socio-economic disadvantage in their neighborhoods, or may even be stress provoking.

Overall, the results of the adjusted model support stress and coping theory, which underscores the importance of considering psychological and social coping resources as mediators between stressor exposure (disadvantage) and psychological stress (Lazarus & Folkman, 1984; Pearlin, et al., 1981). Research that fails to support the relationship between disadvantage and health (or other intermediary outcomes) without examining the

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full structural model, may simply be unable to detect such distant effects (Browning et al., 2003; Cagney & Browning, 2004; Morenoff, et al., 2007). In some neighborhoods, socio-economic disadvantage may be so pervasive that residents become psychologically immune to exposure to these stressors, but it may affect stress and mental health through neighborhood processes that are more palpable to residents. Social isolation, neighborhood fear, and lack of social control and cohesion may all have a more immediate influence on residents' perceptions of stress (Aneshensel, 2010). Although neighborhood disadvantage did have an indirect effect on stress, and on health via the other factors in the model, the influence of neighborhood attitudes on stress and on health was much stronger, suggesting that the more proximal construct is more closely and directly related to health.

Despite the addition of a proximal mediating factor connecting neighborhood disadvantage to stress, neighborhood crime and safety, and neighborhood social capital were not related to neighborhood attitudes or perceived stress in the second model. As noted previously, future research could more thoroughly examine additional mediating and moderating factors – at both the neighborhood and individual level – that may help determine the means through which neighborhood crime and safety, and neighborhood social capital affect individuals' perceived stress. In addition to social support, which was mentioned previously as one such factor, interactions between neighborhood crime/safety and social capital, and individual vulnerabilities (e.g., acute stressors and life events), coping (e.g., active vs. active coping style), and psychological factors (e.g., self-efficacy) could be examined. Researchers have documented synergistic effects of neighborhood- and individual-level factors on health (Aneshensel, 2010; Diez Roux,

2001; Diez Roux & Mair, 2010), and thus the effect of neighborhood crime and social capital may be dependent on a variety of individual-level factors.

Additionally, different measures of stress could be considered, as perceived stress may not most accurately reflect exposure to chronic stressors (Worthman & Costello, 2009). This may be particularly problematic for residents of highly disadvantaged neighborhoods who are constantly exposed to economic, social, environmental and psychological stressors in their communities. For these individuals, the perceived stress measure used in this study may reflect current perceptions of stress during the interview, and not chronic stress due to exposure to neighborhood crime and violence, or other stressors. Future research could include more neighborhood-specific stress measures, or focus questions of perceived stress on the neighborhood experience (e.g., "In the past month how often have you felt like things were happening in your neighborhood that were out of your control?"). Researchers could also include assessments of physiological stress biomarkers, which may help connect exposure to neighborhood stressors to physiological health.

In the final portion of the hypothesized model I examined the relationship between psychological stress and health, to relate neighborhood exposures to a physical health indicator through a stress process. Residents of neighborhoods with more perceived stress had worse self-reported health. Researchers have documented consistent relationships between self-rated health and mortality (Idler & Benyamini, 1997), even after accounting for demographics and other physical health covariates. My results, therefore, suggest that psychological factors like perceptions of stress may be the, "initial link in a chain of events that leads to a biological stress (p. 413)" (Cicchetti & Walker,

2001).  Researchers believe that psychological stress exerts real effects on health, whether through physiological mechanisms (e.g., allostatic load or immunity) (Cohen & Herbert, 1996; McEwen & Seeman, 1999), or coping (e.g., John Henryism) (James, 1994; James, Strogatz, Wing & Ramsey, 1987).  Despite the well-documented association between stress and health, few researchers have included measures of physical health, whether self-rated health or specific health outcomes, in their neighborhood models.  Thus, research that examines the relationship between neighborhood context, stress, and health is critical in refining our understanding of connections between psychological and physical health.

**Limitations**

Several limitations of this research should be noted.  First, the individual-level data were aggregated to the census block group unit for methodological concerns.  Thus, the results of an ecological analysis should be considered as such, and must be cautiously generalized to individual-level effects, as relationships at the ecological level cannot necessarily be attributed to each individual.  Yet, Schwartz (Schwartz, 1994) argues that conflicting results from ecological versus non-ecological studies may have more to do with internal and construct validity of the measures, than the analysis itself.  By using SEM I am able to partially control for problems with internal validity, as SEM accounts for unmeasured variance between the estimated regression paths (Kline, 2005).

Another limitation of this study is the small sample size.  Although I had sufficient power to examine my hypothesized model, the small sample size limited my ability to consider additional mediating factors, as well as to test for moderation.  The small number of observations also resulted in the need to impute data for the violent

crime measures outside of Flint.  Additionally, this research was cross-sectional, which further limits our ability to determine causality.  Bradley and Schaefer (1998) note that "a claim of causality must not only demonstrate correlation, but must also provide an explanation of a *mechanism* by which the cause produces the effect (p. 161-162)." Although this study did not use longitudinal data, structural equation modeling increases our confidence determining causality, because by nature this analytic technique tests a mechanism by which neighborhood disadvantage affects health.

Finally, because of methodological limitations and the availability of several data sets that were mapped to census units, I conceptualized *neighborhood* as a census block group in this analysis, and used U.S. Census data, crime data, and neighborhood perceptions aggregated to the block group.  Administrative definitions of neighborhood are useful because the data are readily available, but administrative boundaries may differ significantly from historical, environmental, and social neighborhood boundaries (Aneshensel, 2010; Diez Roux, 2001).  This limitation may be particularly problematic when assessing neighborhood processes like social capital and neighboring, as these constructs are likely to cross census boundaries.  By using data aggregated to the block group level, I may be underestimating or omitting important neighborhood-level factors that affect perceived stress and health.

**Conclusions**

Despite the limitations noted, this research extends our understanding of the ways through which neighborhood disadvantage affects health.  By using structural equation models I was able to more thoroughly examine mediating pathways connecting neighborhood context to health.  Few researchers have examined neighborhood models

that include theoretically important neighborhood and individual intermediary constructs connecting neighborhood disadvantage to health. By using structural equation modeling, and data from several sources, I was able to include neighborhood social processes, perceptions, and psychological stress in my model. Although theories that neighborhood factors influence health are not new, I identified neighborhood perceptions and perceived stress as critical mediators of this relationship. Understanding the full process through which neighborhood disadvantage influences health is critical in developing policies and interventions to improve the health of residents from socio-economically disadvantaged communities. By identifying these mediating factors, public health practitioners can focus valuable resources on aspects of the neighborhood or individual psychological or coping processes that are both changeable and relevant.

While most of the research on neighborhood context and health focuses on the effects of neighborhood context on mental health, results from this study suggest that neighborhoods may also affect physical health through a stress mechanism. Interest in the connections between mental and physical well-being is emerging rapidly, including the relationship between psychological and physiological stress, and mental and physical health (Cicchetti & Walker, 2001; Dowd, Simanek, & Aiello, 2009; Jackson et al., 1996). Overall, the results from my study support the connection between psychological and physical health. They suggest that neighborhoods may play a role in shaping residents' physical health, through a stress and coping mechanism that includes both neighborhood- and individual-level psychological factors.

Where people live matters for their emotional, psychological and physical health. Research that clarifies the processes through which neighborhood factors like socio-

economic disadvantage influence health can help inform policy and intervention to improve health and reduce socio-economic and racial health disparities. While reducing poverty and unemployment in disadvantaged neighborhoods will take time, my results suggest that it may be possible to intervene by improving resources and social connections in neighborhoods to improve residents' attitudes about where they live. Building a sense of community and organization through community gardens, for example, has the potential to improve perceptions of the neighborhood (Armstrong, 2000), which may reduce stress and improve health in disadvantaged communities. Future research could continue to explore the processes through which neighborhood and individual resources may protect individuals from stress, to improve health and reduce health inequalities

**Table 4.1: Correlations among study variables**

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1.NH dis. 1 | 1.00 | | | | | | | | | | | | | | |
| 2. NH dis 2 | 0.75* | 1.00 | | | | | | | | | | | | | |
| 3. NH dis 3 | 0.72* | 0.74* | 1.00 | | | | | | | | | | | | |
| 4. Violent crime | 0.32* | 0.37* | 0.36* | 1.00 | | | | | | | | | | | |
| 5. Perceived crime | 0.50* | 0.52* | 0.47* | 0.28* | 1.00 | | | | | | | | | | |
| 6. Safety | -0.55* | -0.57* | -0.54* | -0.29* | -0.84* | 1.00 | | | | | | | | | |
| 7. Trust | -0.33* | -0.41* | -0.32* | -0.22* | -0.61* | 0.66* | 1.00 | | | | | | | | |
| 8. Visit | -0.22* | -0.25* | -0.24* | -0.14 | -0.33* | 0.36* | 0.44* | 1.00 | | | | | | | |
| 9. Help | -0.27* | -0.24* | -0.17* | -0.17* | -0.46* | 0.50* | 0.64* | 0.52* | 1.00 | | | | | | |
| 10. Stress 1 | 0.14 | 0.07 | 0.18* | 0.15 | 0.06 | -0.14 | -0.04 | -0.22* | -0.07 | 1.00 | | | | | |
| 11. Stress 2 | 0.16 | 0.18* | 0.21* | 0.09 | -0.01 | -0.04 | -0.01 | -0.14 | -0.10 | 0.60* | 1.00 | | | | |
| 12. Stress 3 | 0.18* | 0.17* | 0.17* | 0.14 | 0.05 | -0.07 | -0.05 | -0.11 | -0.09 | 0.63* | 0.63* | 1.00 | | | |
| 13. Stress 4 | 0.08 | 0.05 | 0.09 | 0.12 | -0.04 | 0.07 | 0.08 | -0.10 | 0.02 | 0.75* | 0.65* | 0.71* | 1.00 | | |
| 14. Health | 0.13 | 0.18* | 0.09 | 0.06 | -0.09 | 0.00 | 0.04* | 0.04 | -0.05* | 0.20 | 0.28 | 0.29 | 0.21 | 1.00 | |

Table 4.1 Continued

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 15. Health concern | 0.08 | 0.17* | 0.14 | 0.06 | 0.07 | -0.09 | -0.04* | -0.11 | -0.08* | 0.24 | 0.27 | 0.35 | 0.19 | 0.12 | 1.00 |

*$p \leq 0.05$

**Table 4.2: Standardized coefficients and error terms for measurement portion of final structural model**

| | Standardized coefficient | Error | $R^2$ |
|---|---|---|---|
| **Crime and safety** | | | |
| Violent crime | 0.071 | 0.980 | 0.04 |
| Perceived crime | 0.878 | 0.479 | 0.77 |
| Safety walking in hood | -0.957 | 0.291 | 0.92 |
| **Disadvantage** | | | |
| Parcel 1 | 0.858 | 0.513 | 0.74 |
| Parcel 2 | 0.880 | 0.475 | 0.77 |
| Parcel 3 | 0.842 | 0.539 | 0.71 |
| **Social Cohesion** | | | |
| Help neighbors | 0.698 | 0.716 | 0.49 |
| Visit neighbors | 0.484 | 0.875 | 0.23 |
| Trust neighbors | 0.913 | 0.407 | 0.83 |
| **Neighborhood attitudes [a]** | **0.851** | **0.524** | **0.73** |
| **Perceived stress** | | | |
| Parcel 1 | 0.737 | 0.676 | 0.54 |
| Parcel 2 | 0.755 | 0.655 | 0.57 |
| Parcel 3 | 0.868 | 0.497 | 0.75 |
| Parcel 4 | 0.813 | 0.582 | 0.66 |
| **Self-rated health** | | | |
| Concern about health | 0.355 | 0.935 | 0.13 |
| Relative health | 0.339 | 0.941 | 0.12 |

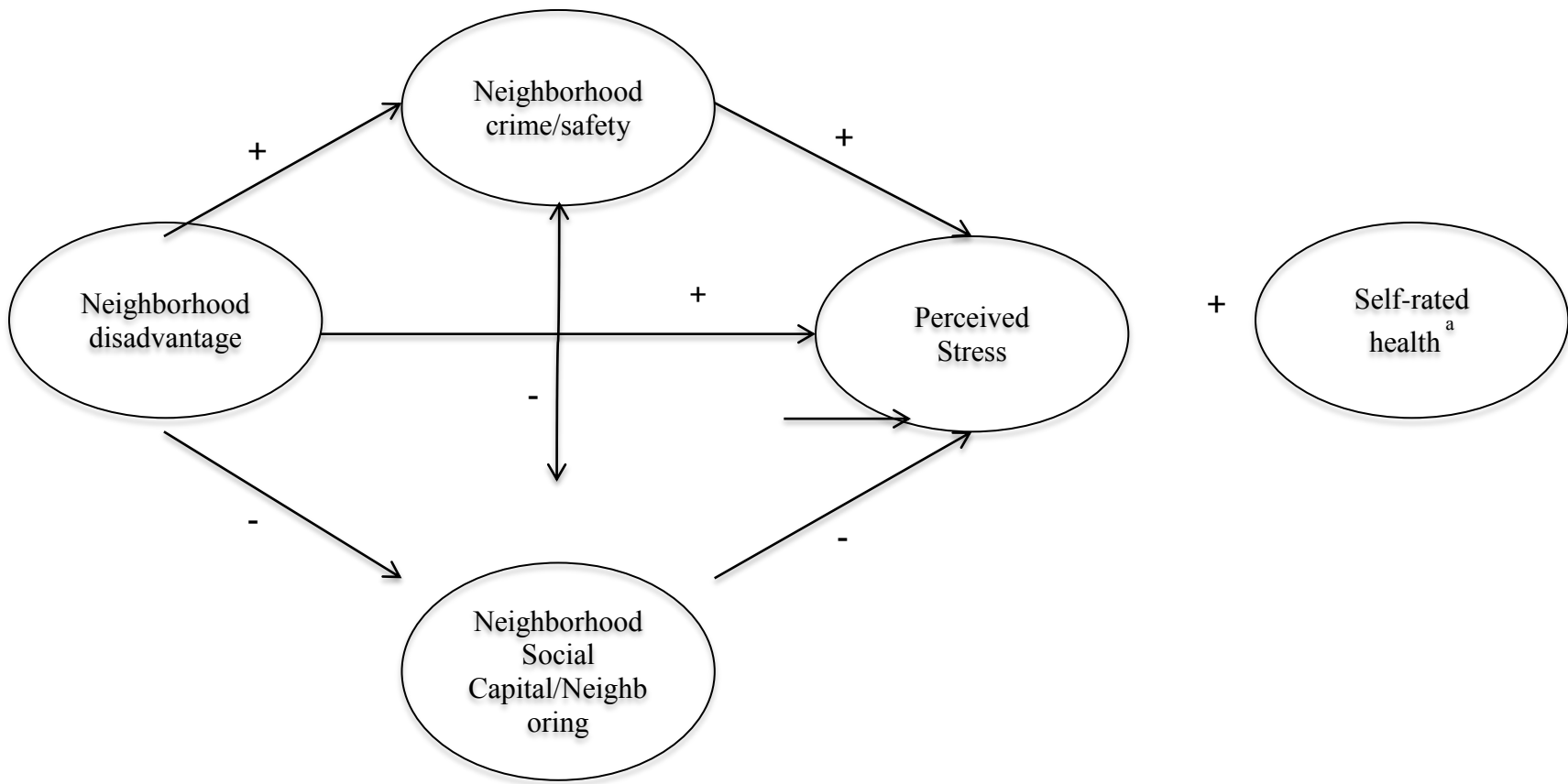[a] Included in the second model only (described in model respecification section)

**Figure 4.4 - Hypothesized structural model for neighborhood context, stress and health**

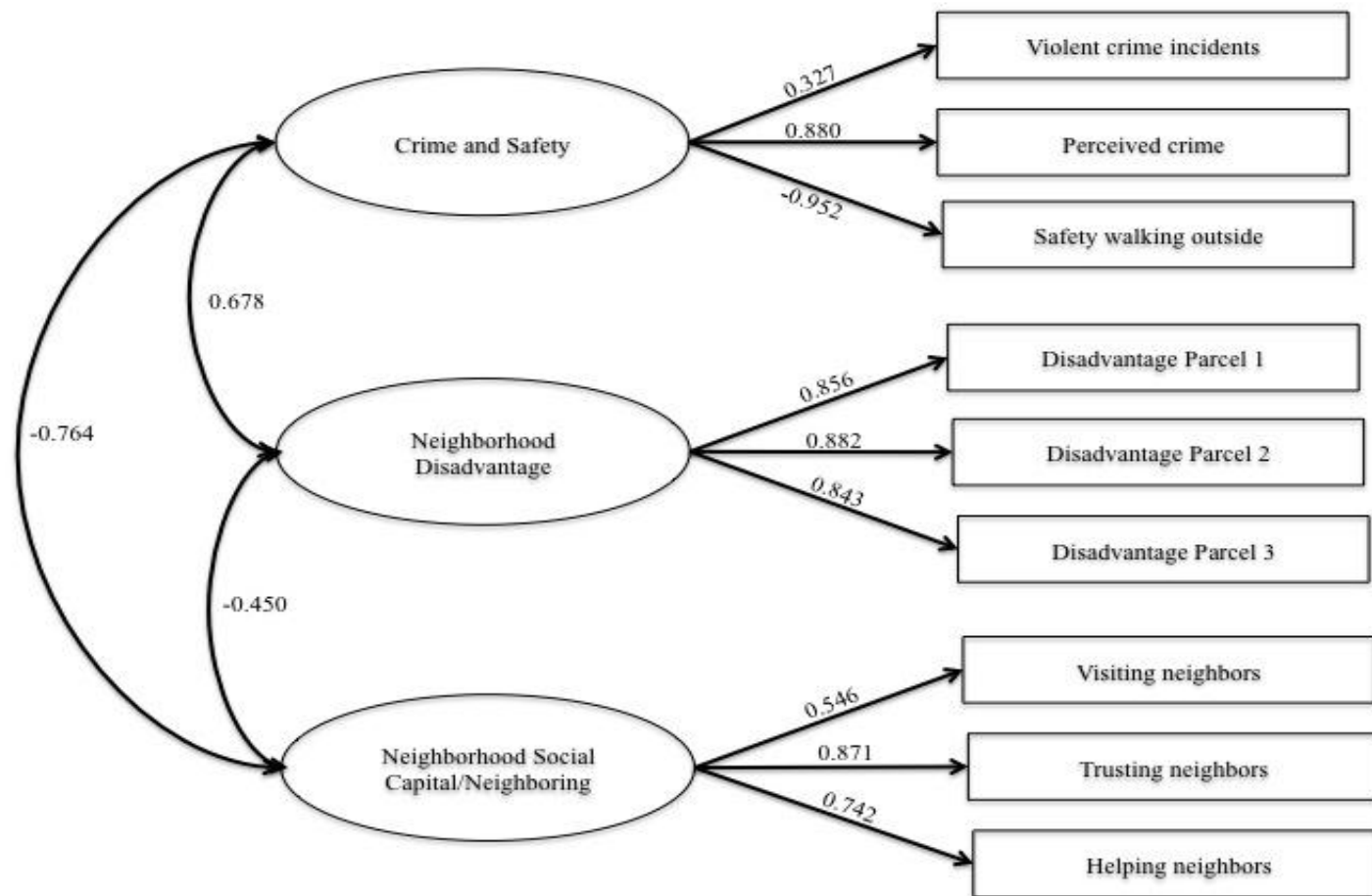[a] **Higher values denote worse health**

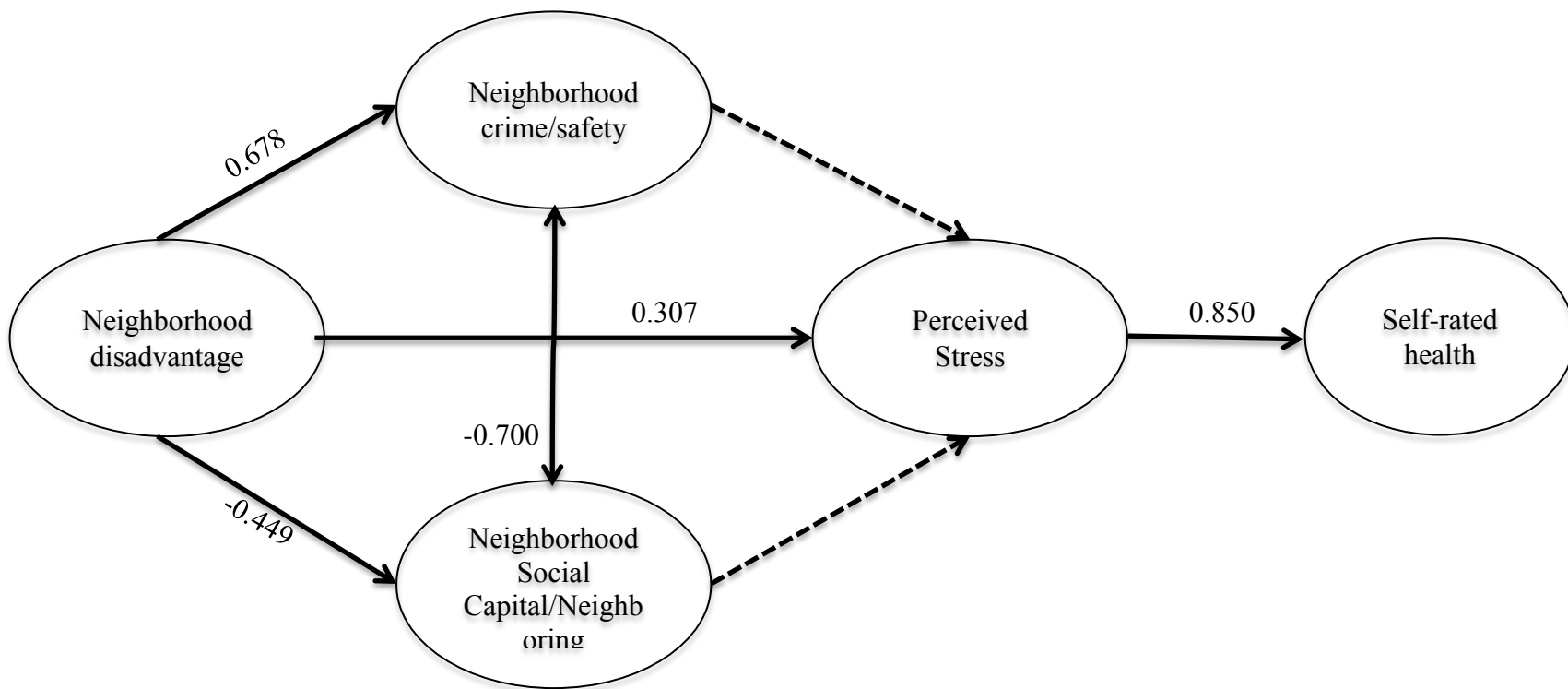**Figure 4.5 – Neighborhood measurement model**

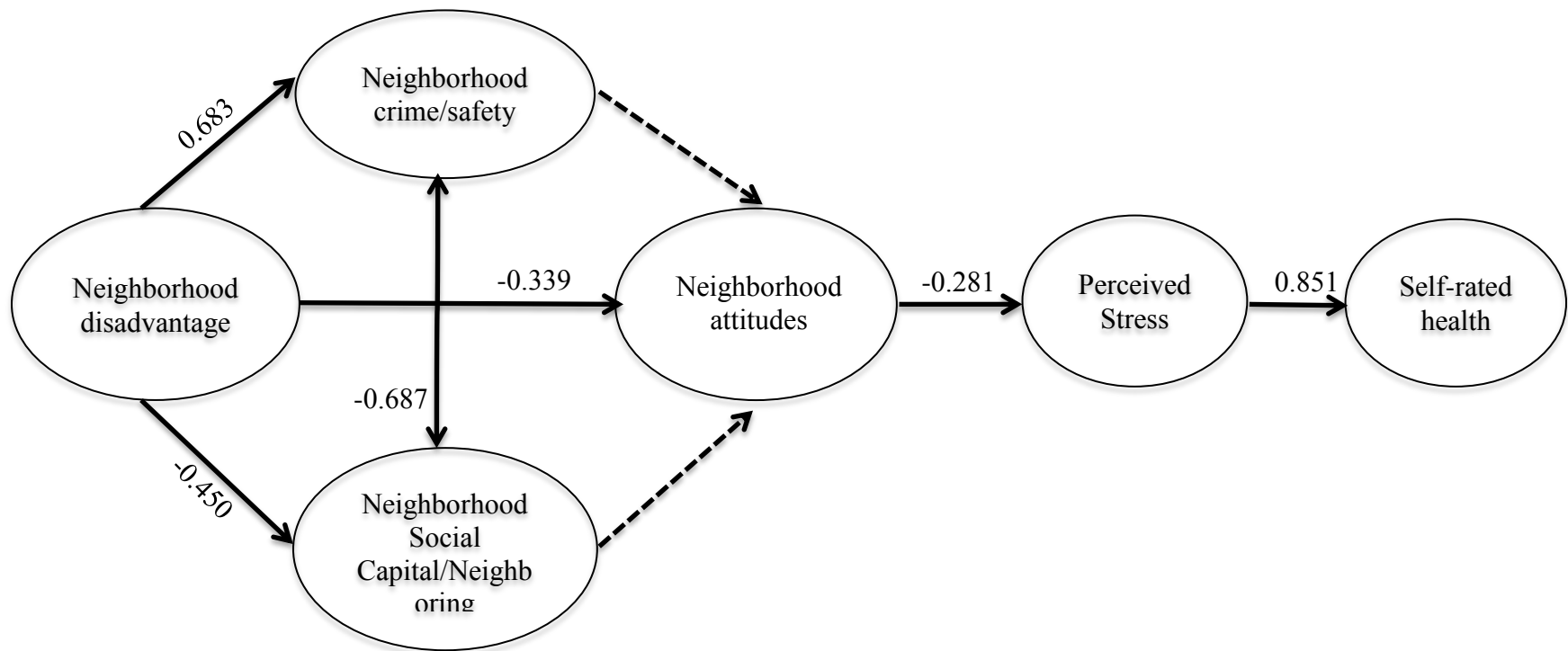**Figure 4.6 - Structural model with path coefficients (dashed lines denote non-significant paths)**

**Figure 4.4 – Post-hoc re-specified structural model with path coefficients**

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# CHAPTER 5

## DISCUSSION AND IMPLICATIONS

Unequal exposure to neighborhood stressors has been proposed as a fundamental cause of Black-White health inequalities, yet few researchers have thoroughly examined the relationship between neighborhood exposures and psychological stress through a stress and coping framework. The goal of this dissertation was to better understand the means through which the neighborhood social and economic environment influences individual's behavior, perceptions, social support, physiological stress (cortisol), and overall well-being. More broadly, by elucidating the mechanisms through which residents of disadvantaged neighborhoods are exposed to stress, we may begin to understand how stress contributes to physical and mental health.

Overall, the results from my dissertation suggest that neighborhood context influences stress beyond individual socio-economic factors, although the relationship between neighborhood context and stress may be dependent on mediating neighborhood social factors and processes, and individual attitudes and behavior. While the idea that neighborhood effects are complex and interact with factors at the individual and interpersonal-level is not new (Aneshensel, 2010; Cagney & Cornwell, 2010; Diez Roux, 2001; Diez Roux & Mair, 2010), this dissertation advances research by including both psychological and physiological measures of stress, and focusing on stress as an important health outcome.

**Summary of findings**

In Chapter 2, I applied the socio-ecological framework, the Transactional Model of Stress and Coping, and neighborhood structural and stress models to examine the perceived stress trajectory as youth transitioned into young adulthood, which is a notoriously stress-provoking developmental period (Arnett, 1999). Perceived stress decreased for youth during middle and late adolescence, and then began to increase during the transition into adulthood. Although living in a more socio-economically disadvantaged neighborhood was initially associated with the stress trajectory, the effect of neighborhood disadvantage on perceived stress disappeared once I accounted for inter- and intrapersonal factors. Specifically, individuals who used more substances reported more stress, but social support from mothers and important adult relationships, and more high effort coping (e.g., John Henryism), protected youth and young adults from stressor exposure.

Overall, this study is consistent with stress and coping theories, which identify social support, behavior, and coping, as critical intervening factors of the stress process (Pearlin, Menaghan, Lieberman & Mullan, 1981; Turner & Turner, 2005). My results supported the socio-ecological framework and neighborhood models less robustly. Although neighborhood socio-economic disadvantage did not exert an influence on perceived stress, or interact with intra- and interpersonal risk and promotive factors as hypothesized, residents' perceptions of their neighborhood did affect their stress. Individuals who reported more negative perceptions of their neighborhood also reported more perceived stress. This suggests that more proximal measures of neighborhoods, which shape the ways youth and young adults perceive their communities, may be more

meaningful measures of neighborhood context, and may be more strongly associated with psychological stress than more distal assessments of the context using archival data. In addition, results from this study indicate that intra- and interpersonal promotive factors like coping and social support can help residents living in socio-economically disadvantaged neighborhoods manage stress.

In Chapter 3, I extended my conceptualization of *stress* beyond the psychological process, by including cortisol as an important stress biomarker, which may reflect the physiological expression of chronic exposure to neighborhood stressors. Similarly to the previous study, I also examined the influence of neighborhood, inter- and intrapersonal factors within the socio-ecological framework, on physiological stress. In support of theories of stress and coping and the Biopsychosocial model, John Henryism and perceptions of stress were associated with cortisol. Individuals, who reported more John Henryism, or high effort coping, had higher baseline cortisol levels than their peers with average John Henryism. Consistent with previous research on allostatic load, weathering, and John Henryism (Geronimus, Hicken, Keene, & Bound, 2006; Jackson, Knight, & Rafferty, 2009; James, Keenan, Strogatz, Browning, & Garrett, 1992; James, Strogatz, Wing & Ramsey, 1987), these results suggest that while high effort coping may confer psychological benefits (less perceived stress), as documented in the previous study (Chapter 2), it may also increase physical wear on the body. I also found that individuals who reported more perceived stress had a blunted cortisol response, which alludes to the possibility that psychological aspects of stress influence the physiological expression of stress.

In support of a socio-ecological framework, and neighborhood structure and stress models, results from this research also indicate that neighborhoods matter. Neighborhood socio-economic disadvantage influenced cortisol directly for individuals living in neighborhoods with low labor force participation, such that these individuals had elevated cortisol levels at baseline. This suggests that neighborhoods where more people are completely detached from the labor force may increase chronic stress for residents, which is expressed at a physiological level. I also found that neighborhood socio-economic disadvantage interacted with intrapersonal factors related to the stress process, to indirectly affect cortisol. Thus, living in disadvantaged neighborhoods may take a psychological and physiological toll on residents, and may also exert synergistic effects through individual coping and vulnerabilities.

Finally, in Chapter 4, I examined the stress mechanism through which socio-economic disadvantage in a neighborhood influenced global self-rated health. Although many researchers have examined neighborhood influences on mental health, few have studied a thorough causal pathway including both neighborhood and individual-level mediating processes, and their affect on overall health (Kruger, Reischl & Gee, 2007; Ross & Mirowsky, 2001, 2009; Ross, Reynolds, & Geis, 2000). The results from this study support neighborhood models and stress and coping theories by suggesting that the more distal effects of socio-economic neighborhood disadvantage influence individuals' perceptions of stress and general health via a pathway that includes attitudes about the neighborhood.

Results of my research emphasize the importance of individuals' perceptions of their neighborhood, as well as their perceptions of stress, in the causal pathway between

neighborhood disadvantage and health. While my models supported the effect of both disadvantage and neighborhood attitudes on perceived stress and resulting self-rated health, I did not find evidence that elements of neighborhood disorder (e.g., crime, safety), or social capital mediate the relationship between socio-economic disadvantage and stress. It is possible that the social breakdown in neighborhoods caused by socio-economic disadvantage influences psychological stress through additional (unexamined) pathways that include more proximal intra- and inter-personal factors identified in stress and coping theories (Cagney & Cornwell, 2010; Ellen, Mijanovich, & Dillman, 2001; Elliott, 2000). Despite the distal nature of socio-economic neighborhood exposures, and the difficulties presented in addressing inequalities in neighborhood context, my results suggest that focusing on more proximal and changeable factors in neighborhoods may help improve health.

**Themes and public health implications**

Although the relationship between neighborhood socio-economic disadvantage and health was not consistent across all studies in this dissertation, several overall conclusions and themes unite the three papers. First, and perhaps most consequential, my results indicate that neighborhood context does matter for the health of older adolescents and young adults. The research from this dissertation supports the broader literature that cites neighborhoods as having a critical influence on health and well-being beyond family and individual socio-economic and demographic factors (cite).

While many researchers have suggested the critical role neighborhoods play in shaping individual behavior, psychological and physical health, and well-being (Diez Roux & Mair, 2010; Ellen, et al., 2001; Yen & Syme, 1999), my results indicate that

socio-economic disadvantage may directly affect health through a stress mechanism, and indirectly through neighborhood processes and interactions with individual-level factors. While neighborhood disadvantage did not consistently influence stress, my results reinforce the value in considering neighborhood exposures through a socio-ecological framework, and as critical social stressors in the stress process. Despite substantial support for the stress process at the individual-level (Pearlin et al., Cicchetti & Walker, 2001; Compas, Connor-Smith, Saltzman, Harding Thomsen, & Wadsworth, 2001; 1981), research on neighborhood exposures and health has yet to thoroughly examine neighborhood, intrapersonal, interpersonal, and cross-level interactive effects on stress. Many researchers have proposed that neighborhood context affects health through a stress process (Augustin, Glass, James, & Schwartz, 2008; Latkin & Curry, 2003; Ross, 2000; Steptoe & Feldman, 2001), but few have specifically tested this theory, which requires measuring neighborhood exposures, mediating and moderating factors in the stress process (e.g., coping, social support, appraisals of stress), and stress.

In this dissertation I attempted to more concretely connect neighborhood stressor exposure to health via a stress mechanism by including these important variables in my models. I found that, in each of my studies, neighborhood perceptions or neighborhood socio-economic disadvantage was associated with psychological or physiological stress. This implies that neighborhood stressors may relate to mental and physical health through a stress process, and that intervening in this process may be an important point of prevention for future health problems and health inequalities

Understanding the complex processes through which neighborhoods affect mental and physical health is necessary for intervention and prevention, as well as establishing

the connections between social and psychological exposures and physical health outcomes. The studies from this dissertation contribute to the initial stages of this understanding, as I found that neighborhood context – both socio-economic disadvantage and perceptions of the neighborhood – are related to stress. Although several limitations in this dissertation temper my conclusions, this research supports the idea that neighborhood exposures influence residents through a physiological and psychological stress process, and provides a rationale for continued research on neighborhood stressors as a part of a larger biopsychosocial process.

Reducing inequalities in neighborhood socio-economic conditions may be central to eliminating health disparities. Despite recent appeals to move beyond measures of socio-economic disadvantage in neighborhood research, and to consider more proximal neighborhood factors like social and physical disorder, social capital, and collective efficacy (Aneshensel, 2010; Diez Roux, 2001; Diez Roux & Mair, 2010; Ellen, et al., 2001; MacIntyre, 1993), we should not neglect the influence of neighborhood SES and social disadvantage. The results from this dissertation indicate that neighborhood disadvantage may exert direct and indirect effects on stress. It is important, however, to remember that the psychological stress measure used in these analyses may assess more immediate feelings of stress or daily hassles despite my claim that neighborhoods expose residents to chronic stressors. Researchers could consider additional measures of psychological stress that are specific to neighborhood stressor exposure, or different measures that better assess chronic perceptions of stress. Despite this limitation, my research suggests that future work on neighborhood disadvantage as a potential source of stress is warranted. As Wodtke et al. (2011) concluded in their study of the effects of

disadvantage on adolescent educational outcomes, socio-economic factors may exert significant influences on residents over time and during critical developmental periods. Thus, researchers could account for the dose and exposure to disadvantage, and capture neighborhood context longitudinally to more fully capture these neighborhood exposures.

Understanding early and chronic exposure to socio-economic disadvantage may be particularly relevant for Black adolescents and young adults living in segregated neighborhoods such as those in Flint and Genesee County, Michigan. Researchers suggest that mobility is quite limited in these communities (Browning & Cagney, 2003), and thus the individuals in my sample were likely exposed to neighborhood disadvantage from a very young age. Although interventions and policies that address more proximal aspects of the neighborhood context like access to resources, social cohesion, and safety may be more feasible than decreasing neighborhood poverty and unemployment, unequal exposure to neighborhood disadvantage may be a fundamental cause of health disparities. Aspects of disadvantage like unemployment could be addressed through interventions in the neighborhood by increasing opportunities for childcare, or by offering job training. Thus, research that identifies socio-economic disadvantage as a contributor to poor health may help inform future policy and planning to reduce residential segregation.

Like other researchers (Aneshensel, 2010; Haney, 2007; Kruger et al., 2007; Ross & Mirowsky, 2001), I also found that subjective indicators of the neighborhood context partially mediated the effect of socio-economic disadvantage on mental health. Specifically, attitudes about and perceptions of the neighborhood mediated the relationship between disadvantage and perceived stress. The results from each of my studies suggest that interventions that address these attitudes and perceptions may help

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reduce residents' psychological stress, and mitigate the effect of socio-disadvantage on stress and health. These proximal neighborhood factors might be more tangible causes of psychological stress for residents; implementing changes to the neighborhood that could improve how residents perceive their communities, may also reduce stress and improve health. Although researchers and policy makers should not ignore neighborhood disadvantage as a potential fundamental cause of health inequalities, in the interim public health practitioners may be able to mitigate the effect of neighborhood disadvantage by focusing on neighborhood-level mediators in the stress process.

Public health researchers and practitioners could collaborate with government, urban planning, social workers, and public safety to address neighborhood stressors. Specific targets might include cultivating a sense of community through regular social events, crime watch groups, public meetings and functions for children; improving the physical landscape and decreasing physical disorder by increasing access to parks and recreation, or cleaning up the streets and public buildings; or increasing commercial resources such as grocery stores and banks. Overall, the results from this dissertation suggest a multifaceted approach to addressing exposure to neighborhood stressors, with a focus on the underlying cause related to poverty and social disadvantage, as well as stressors and social structures that result from disadvantage.

Another overarching conclusion from the studies in this dissertation is that neighborhood exposures are a part of a larger socio-ecological framework that includes multiple and cross-level influences on individual well-being. Researchers have called for a more thorough analysis of the processes through which behavioral, psychological, and social factors at both the individual and neighborhood level interact to shape health

across different neighborhood contexts (Diez Roux & Mair, Aneshensel, 2010; 2010).

As a response to this appeal, my dissertation included an analysis of several important mediating and moderating processes through which neighborhood socio-economic disadvantage affects stress and health.  Each of these studies generally supported the socio-ecological framework, which emphasizes the critical influence of intra- and interpersonal risk and promotive factors on stress, as well as their role as mediating or moderating factors in the relationship between neighborhood context and stress.  My research indicates that future work on neighborhood influences on health should consider these multiple and interacting factors, as neighborhood factors do not affect residents in isolation.

Consistent with the literature on stress and coping (Elliott, 2000; Pearlin et al., 1981; Pearlin & Schooler, 1978), resources like social support and coping helped youth and young adults avoid psychological stress, while risk factors including substance use, daily hassles, and worry about neighborhood safety increased stress.  The significance of these factors to the larger stress process is well documented (Pearlin et al., 1981), as exposure to neighborhood stressors does not uniformly translate into stress for all individuals; stress is dependent on individual exposures, vulnerabilities, psychology, coping, and behavior (Aneshensel, 2010; Diez Roux, 2001; Diez Roux & Mair, 2010).  These factors may be critical points of intervention, and in particular, early intervention.  To combat the persistent and sustained effect of neighborhood disadvantage, interventions directed at young children to increase coping effectiveness, improve family and mentor support, and help youth avoid substance use to cope with stressors, may be necessary.

A final conclusion derived from this dissertation is the importance of extending the study of stress beyond psychological stress, as exposure to neighborhood stressors may exert influences on health via physiological body systems. Researchers have proposed alternative vulnerability hypotheses, arguing that residents of highly disadvantaged and stressful neighborhoods may become desensitized to stressors and less vulnerable to psychological results of exposures (Elliott, 2000; Kessler, 1979; Wheaton, 1982). Kessler (1979) notes that "life situations are not inherently stressful…they must be interpreted. These interpretations…are partly dependent on the context of one's life, the relevancies and circumstances to which one selectively attends (p. 260)." Thus, individuals who are exposed to neighborhood stressors may not interpret their context as stressful, despite deleterious chronic exposures in the community. Psychological immunity does not necessarily translate into physiological immunity, as research indicates that chronic stressor exposure may take a physiological toll on the body (McEwen & Seeman, 1999).

The results from my research indicate that perhaps not all stressors work through the same mechanism. More distal and chronic stressors like socio-economic disadvantage may affect cortisol as I found in my research, while essentially bypassing more accessible psychological pathways. Researchers should not, therefore, ignore potential physiological effects of neighborhood disadvantage. I documented important distinctions and relationships between the psychological and physiological expression of stress as a result of exposure to neighborhood stressors.

While most researchers focus on the effects of neighborhood stressor exposure and psychological health outcomes (Aneshensel, 2010), exposure to chronic

neighborhood stressors like poverty and unemployment may operate through a physiological stress mechanism, as suggested in the Biopsychosocial Model (Engel, 1980), and supported by my results. This model connects stressor exposure and psychological stress processes (e.g., coping, behavioral responses) to physical health through physiological stress, and thus helps bridge psychological factors and physical health outcomes. Although the physiological stress mechanism operates differently than psychological pathways, I found that individual risk and promotive factors interacted with neighborhood risk to synergistically affect cortisol, similarly to psychological stress processes. This suggests that focusing on these inter- and intrapersonal mediators and moderators of the stress process may be essential in reducing both psychological and physiological stress.

**Limitations and future directions**

A number of limitations inherent in this research should be noted, as they affect the interpretation of my results and relate to future directions of research on neighborhoods and stress. One of the most significant limitations of this research relates to measurement of neighborhood context using only cross-sectional assessments of socio-economic disadvantage. More recent research indicates that the effects of neighborhood disadvantage may exert stronger influences on health during childhood (Wodtke, et al., 2011), thus by using only current disadvantage measures I may be overlooking important neighborhood influences on stress. Additionally, in my conceptual models I propose that neighborhood disadvantage functions as a chronic stressor to increase psychological and physiological stress, but I assessed disadvantage at only one timepoint. Cross-sectional analysis of neighborhood effects also ignores residential mobility, although the

population in the FAS is fairly stable, and those who move tend to move to neighborhoods with similar levels of disadvantage.

Despite this limitation in measuring neighborhood context, using more sophisticated and longitudinal assessments would likely only increase the strength of the associations between neighborhood exposures and stress, making my results more robust. Nevertheless, researchers could continue to explore more nuanced and complex ways to assess the effect of neighborhood stressor exposure, including assessing neighborhood factors during multiple developmental stages beginning in infancy, and including objective and subjective neighborhood measures in research. As I found in my structural equation models, neighborhood disadvantage may both directly and indirectly affect stress and health, and thus research that includes multiple neighborhood measures can account for indirect effects and interactions between disadvantage, perceptions, and social processes in the neighborhood.

Another limitation of this dissertation is the definition of and measurement of neighborhood effects. While most researchers use administratively defined boundaries (i.e., census units) to define neighborhoods because of methodological convenience, this assignment of neighborhood boundaries may not align with the way residents perceive their neighborhood (Aneshensel, 2010; Diez Roux, 2001; Diez Roux & Mair, 2010; Kruger, 2008). Census boundaries often create unnatural divisions in true socially, environmentally, and historically defined neighborhoods (Aneshensel, 2010; Diez Roux & Mair, 2010). When data are then aggregated based on these unnatural administrative distinctions, researchers may either under- or over-estimate neighborhood effects. Due to methodological concerns, however, and the need to combine multiple sources of data

using a common field for this dissertation, this limitation may affect my results and interpretations.  Future research that uses more logical definitions of neighborhoods is necessary to account for actual neighborhood exposures to which residents are exposed. Kruger et al. (2007) operationalized neighborhoods by creating buffer zones around residents' homes, and used GIS to map neighborhood factors based on whether they fell within the pre-determined radius.  Researchers could continue to define neighborhoods in similar ways, or by considering other important factors such as physical characteristics of the neighborhood, social characteristics or school districts.

Although data from the Flint Adolescent Study are ideal for examining risk and promotive factors in youth and young adults longitudinally, and during important developmental time periods, these data present some limitations.  Most of the FAS participants were African American, and while this group of individuals typically lives in more disadvantaged neighborhoods, and have worse health – thus supporting my overall conceptual model – I was not able to examine racial comparisons.  This is particularly important to note when the rationale for my research relates to inequalities in stressor exposure that may contribute to racial health inequalities.  My research focused almost exclusively on within-group comparisons, and future research should address differences between groups to investigate the relationship between stressor exposure and racial inequalities in stress.

While using a nationally representative dataset might allow me to conduct subgroup analyses and would be more generalizable to larger populations, few longitudinal datasets contain such rich data as were collected in the FAS.  Additionally, I was able to combine assessments of neighborhood crime, safety, and social processes

with FAS data on stress and health to avoid same source bias and expand my neighborhood variables. Future researcher could continue to include multiple data sources, as few datasets contain both detailed neighborhood information and individual psychosocial, behavioral and health data.

Lastly, while I was able to link FAS and U.S. census data to study neighborhood effects on health, the FAS was not initially designed for this purpose. Participants were sampled by school, and not by neighborhood unit, thus some neighborhoods only include one individual, while other include ten or more. Ideally, relatively equal numbers of participants would be sampled from each census unit to ensure adequate variation both within and between neighborhoods. Despite this limitation, I did find significant neighborhood effects, which suggests that my results would have been stronger if the FAS had been designed to test the influence of neighborhood context on health.

My results suggest that efforts to continue to design research specifically to test neighborhood effects on individual health outcomes is warranted and necessary to address several unanswered questions. Ideally these data will be sampled based on a pre-defined neighborhood sampling scheme, and will include a variety of data including neighborhood ratings (e.g., neutral observer ratings of physical and social neighborhood disorder), residents' perceptions of their neighborhood, police crime data, information on neighborhood resources (e.g., retailers and commercial business, recreation, community resources), and measures of collective efficacy and social capital. Based on the results from this dissertation, researchers could also include intra-, interpersonal, and organizational factors based on the socio-ecological framework. These factors are likely to mediate and interact with neighborhood stressors to influence individual health. By

considering neighborhood and individual-level theories, researchers will be better able to test complex models and multiple influences on health.

**Conclusion**

Taken together, results from this dissertation make an important contribution to the literature on neighborhood context and health, and lend credence to the idea that unequal exposure to neighborhood stressors may result in inequalities in psychological and physiological stress. Consequently, these inequalities may translate into and contribute to future health inequalities through a stress mechanism. Despite the recent escalation in research on neighborhood effects and health, we are only beginning to understand the complex ways through which neighborhood exposures affect health.

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