NEIGHBORHOOD CONTEXT AND WELL-BEING: HOW MUCH DOES NEIGHBORHOOD REALLY MATTER?

by

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(Under the Direction of RONALD L. SIMONS)

ABSTRACT

Although the link between neighborhood contexts and individual well-being is robust, little is known about how individual characteristics affect the way in which people respond to neighborhood conditions. In addressing this issue, this dissertation assessed whether prior neighborhood studies can be replicated using a sample of adult African American females and the possible gendered and temporal nature of street code adoption. Additionally, I addressed why there is so much heterogeneity in the behavior of individuals residing in the same neighborhood. Concerning non-criminogenic outcomes, I analyzed how neighborhood effects influence physical health?

Using a sample of adult females, I found that the impact of neighborhood disadvantage on antisocial behavior was mediated by social ties. Further, the effects of neighborhood disadvantage and social ties on antisocial behavior were moderated by genes. Examination of these moderating effects provided support for the differential susceptibility model of G×E. Finally, the effect of G×neighborhood disadvantage on antisocial behavior was explained by the effect of G×social ties. These findings provide strong support for an expanded view of social disorganization theory.

Beyond criminogenic outcomes, health outcomes are essential for understanding the holistic impact of neighborhood contexts on individuals. The findings of this dissertation showed that women who live in disorderly neighborhoods were more likely to report poor health status and to have elevated inflammatory responses. Furthermore, the relationship between neighborhood disorder and self-reported health was mediated by the inflammatory burden as a signal of physiological distress. More importantly, effects of neighborhood disorder on the inflammatory responses and health were not uniform but were most pronounced among unmarried women carrying the minor allele of the IL-6r gene.

Finally, while a handful of existing studies support Anderson's street code thesis, few studies have considered the dynamic nature of street code. I found that adopting the street code is a dynamic process rather than a fixed and stable trait. Moreover, my results demonstrated that the change in adoption of street code over time can be explained by change in socially demoralizing environments. The results support that adherence to code values can be switched over time in accordance with social environmental change.

INDEX WORDS: Neighborhood disorganization, Antisocial behavior, Physical health, Street code adoption, Violence, Gene by environment interaction

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DEDICATION

To god and my mom.

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CHAPTER 1

INTRODUCTION

"No man is an island entire of itself; every man is a piece of the continent, a part of the main…."
(John Donne, Meditation XVII, Devotions upon Emergent Occasions)

Because people spend a considerable amount of their lives in their local residential neighborhoods, sociologists and criminologists have had a long-standing interest in the role of neighborhood context. In his presidential address to the American Society of Criminology, Robert Sampson, a highly respected sociological criminologist, stated that "[neighborhood] is a fundamental context that has widespread effects on crime, perceptions of order and disorder, well-being, and much more" (Sampson, 2013, p. 1). During the past two decades, numerous studies have provided evidence for the link between neighborhood context and different aspects of well-being, including crime (Sampson, Rausdenbush, & Earls, 1997; Simons et al., 2005), delinquency (Beaver et al., 2012; Brody et al., 2001; Foster & Brooks-Gunn, 2013; Martin et al., 2011), violence (Berg et al., 2012; Lei et al., 2014; McNulty, Bellair, & Watts, 2013), subculture (Anderson, 1999; Simons et al., 2012; Stewart & Simons, 2006), depression (Hill, Ross, & Angel, 2005; Ross & Mirowsky, 2009), biomarkers (Browning, Cagney, & Iveniuk, 2012; Holmes & Marcelli, 2012), and self-reported health status (Aneshensel & Sucoff, 1996; Browning & Cagney, 2003; Cohen et al., 2003; Franzini et al., 2005). In addition, the link between neighborhood context and well-being has been observed across countries, populations,

sampling strategies, study designs, and with controls for a wide range of individual characteristics (Leventhal & Brooks-Gunn, 2000; Sampson, 2012; Wikström & Sampson, 2003).

For over a half century, two main findings from neighborhood studies dominated the literature. First of all, in association with differing socioeconomic composition, individual well-being is disproportionately distributed across neighborhoods. At the macro level, studies have revealed that neighborhoods characterized by high levels of poverty have high rates of crime and mortality as well (Macintyre & Ellaway, 2003; Shaw & McKay, [1942] 1969). In macro/micro models, empirical research has supported that individuals who live in disadvantaged neighborhoods are more likely to engage in crime/deviance and to have poor health status than those who live in advantaged neighborhoods (Diez-Roux & Mair, 2010; Leventhal & Brooks-Gunn, 2000; Sampson, Morenoff, Gannon-Rowley, 2002). As a result, the concentration of disadvantage in neighborhoods has gained a great deal of attention in sociology (Wilson, 1987).

The second finding of previous neighborhood studies is that the relationship between race and well-being is confounded by neighborhood context (Peterson & Krivo, 2010). For example, many studies have reported that African-Americans are more likely than other racial groups to reside in extremely disadvantaged neighborhoods, which in turn relates to heightened engagement in criminal behavior (McNulty, 2001; McNulty & Bellair, 2003; Sampson & Wilson, 1995). Similar to criminal behavior, poor health status is also disproportionately concentrated in dominated African-American neighborhoods because chronic stress, racial discrimination and poverty are common in these areas (Massey, 2004).

These two general findings are rooted in the assumption that the places where people live are important determinants of human behavior and well-being, and people living in a same neighborhood are more similar to each other than to those from different neighborhoods with

respect to individual outcomes. Nevertheless, just as Fischer (2013, p. 7) noted, "urban sociologists' and the public's assumption that neighborhood matter may be wrong and may be right only in small ways." For instance, many researchers (Barnes & Jacobs, 2013; Brody et al., 2001; Elliott et al., 2006; Hart & Marmorstein, 2009; Meier et al., 2008; Simons et al., 2005) have argued that traditional neighborhood studies cannot explain why some people who live in disadvantaged neighborhoods do not lead to crime/deviance or why people living in the same neighborhood present so much heterogeneity. Furthermore, several scholars (Bowling & Stafford, 2007; Browning & Cagney, 2003; Gallo et al., 2012; Petersen et al., 2008; Robert, 1998) have posed the classical questions: Do neighborhood effects apply to health or any well-being outcomes? Does neighborhood context really matter? However, the results are mixed. Several studies found a strong association between neighborhood contexts and health or well-being outcomes (Browning, Cagney, & Iveniuk, 2012; Ross & Mirowsky, 2001), whereas other studies reported no such association after controlling for individual SES and health-related lifestyles (Bowling & Stafford, 2007; Petersen et al., 2008; Reijneveld & Schene, 1998).

In summary, while the basic association between neighborhood context and well-being is fairly well established, it is still unclear why there is much heterogeneity among individuals residing in the same neighborhood, does neighborhood context explain not only crime/deviance but also health outcomes, and what mechanisms are involved in neighborhood context on different aspects of well-being? The purpose of the dissertation is to answer such questions. Given that African-Americans are more likely than other racial groups to reside in disadvantage neighborhoods, to engage in crime/deviance, and to have health problems (Gabbidon, 2010; Peterson & Krivo, 2010; Thoits, 2010), this dissertation uses data from the Family and Community Health Study (FACHS) that was designed to assess the linkages between

neighborhood context and African Americans' well-being. The research in this dissertation will propose three studies to examine the conceptual models linking neighborhood context and well-being.

In the following, the first section demonstrates why focus on African Americans. The second section outlines the arguments and limitations of social disorganization theory and proposes research questions in Study 1. The third section introduces the broken window theory and indicates which neighborhood characteristics and processes seem to have the most impact upon health status. In addition, I extend neighborhood research on the stress process model and identify the main research questions for Study 2. Finally, the fourth section introduces Elijah Anderson's code of the street thesis, elaborates models of the impact of changes in socially demoralizing conditions in local neighborhoods on changes in street code adoption, and describes the purpose of Study 3.

WHY ARE NEIGHBORHOOD EFFECTS ESPECIALLY IMPORTANT FOR AFRICAN AMERICANS?

One of the more consistent and robust findings in criminology and public health is that African Americans commit much more crime/deviance and have worse health outcomes than other ethnic groups. For example, the official data show African Americans are imprisoned five times as often as Whites (Wakefield & Uggen, 2010) and are thirty percent more likely than Whites to die of cardiovascular disease (The National Center for Health Statistics, 2011).

In response to these findings, Shaw and McKay observed ([1942] 1969) that juvenile delinquency rates remained high in certain structurally disadvantaged areas regardless of which racial/ethnic group predominately occupied those neighborhoods. Contemporary neighborhood

scholars (McNulty, 2001; Peterson & Krivo, 2010; Steffensmeier et al., 2010; Wilson, 1987) have expanded their ideas and proposed the racial invariance hypothesis. This states that neighborhood structural conditions equally affect human behavior of all racial/ethnic groups. As a result, racial inequalities in well-being are rooted in structural spatial inequalities. An abundance of studies have provided evidence that African Americans have the highest rates of crime/deviance and experience more stressful events and chronic strains, such as housing discrimination, economic hardship, social isolation, racism, marital difficulties, and constrained choices (Beach et al., 2012; Conger et al., 2002; Massey, 1993; Sampson, 2012; Simons et al., 2002, 2005; Turner & Lloyd, 1995) because they are disproportionately represented in underprivileged neighborhoods (McNulty, 2001; McNulty, Bellair, & Watts, 2013; Peterson & Krivo, 2010; Sampson, Morenoff, & Raudenbush, 2005; Steffensmeier et al., 2010; Wilson, 1987). There are two main approaches to explain why African Americans are unevenly distributed geographically.

In *The Truly Disadvantaged*, William Julius Wilson (1987) proposed the spatial mismatch approach to understand the link between neighborhood context and race. He argued that neighborhood structures are changed by economic structure. As a result of this, when factories and companies migrated from inner cities to suburbia, job opportunities disappeared in the urban areas which resulted in concentrated disadvantage in these neighborhoods. Given that African Americans are less likely to relocate to affluent neighborhoods, African Americans living in disadvantaged neighborhoods have minimal access to economic resources and find it more difficult to obtain jobs, which in turn lead to poor well-being and high social isolation.

In addition to economic structural factors, Massey (1993) indicated that the American apartheid system still exists in current U.S. society, and he has claimed that racial segregation

combined with class segregation to create underclass neighborhoods. Most Whites live in the suburbs while African Americans remain in the inner city because of discriminatory housing practices. When poor underclass Black people are concentrated together in certain neighborhoods, they experience a loss of economic and social resources, which may, in turn, influence individual well-being. Based on his observations, neighborhood context has a strong impact on African-Americans' life experiences and life chances.

Taken together, racial stratification and spatial inequalities mean that African Americans are at high-risk for both criminal behavior and poor health outcomes. Neighborhood characteristics provide an important context to understand African Americans' well-being and behaviors. I address this important area of research by focusing on an African American sample and their local neighborhoods in the current dissertation.

SOCIAL DISORGANIZATION THEORY

Neighborhood studies can be traced back to the early twentieth century. Chicago School scholars observed that rates of crime and poverty are unevenly distributed in geographical areas. Drawing on ideas from natural ecology, Park and Burgess (1924), pioneers in the field, proposed the concept of social ecology. They indicated that cities develop in a systematic way that is composed of a series of circular zones from a central core. According to their observation, there are five different zones in the city of Chicago, including a central business district, transitional zone, working class zone, residential zone, and commuter zone. More importantly, Park and Burgess identified the transitional zone as located between the central business and working class zones and is characterized by deteriorated housing, factories, residential mobility,

abandoned buildings, and heterogeneous population. Additionally, crime is most prevalent in this zone.

Building on the Park and Burgess concentric zone model, social disorganization theory was developed by Shaw and Mckay's ([1942] 1969) research, *Juvenile Delinquency and Urban Areas*. Using juvenile court records in the Chicago, they reexamined Park and Burgess's (1924) human ecology model and observed that crime and delinquency rates were disproportionately distributed throughout the Chicago. In particular, rates of crime and delinquency were highest in neighborhoods within the transitional zone where many lower-class and African American residents lived. This spurred the intriguing question of how neighborhood contexts influence crime rates.

To understand why crime/deviance rates are unevenly distributed across neighborhoods, Shaw and Mckay claimed that neighborhoods characterized by economically concentrated disadvantage, racial heterogeneity, and residential instability are more likely to encounter social disorganization. This disorganization then disrupts the social control system, which in turn leads to higher rates of crime and deviance. Thus their approach characterizes social disorganization theory as a social control model (Kornhauser, 1978). Given that the highest crime rates in disadvantaged neighborhoods, the theory focuses primarily on those neighborhoods that block neighborhood residents' ability to control neighborhood crime/deviance.

Although Shaw and Mckay provide an insight to understand the neighborhood-crime relationship, classical social disorganization theory still has some limitations. Contemporary neighborhood studies build upon and go beyond Shaw and McKay's social disorganization theory (Bursik, 1988). Unlike Shaw and McKay who focused on official crime rates that may have had biases, contemporary studies examined how neighborhood characteristics influenced a

variety of macro- and micro- level outcomes including victimization rates (Velez, 2001), recidivism (Kubrin & Stewart, 2006), self-report delinquency (Wikström & Loeber, 2000), violence (Silver, 2000), domestic violence (Benson et al., 2004), and health outcomes (Ross & Mirowsky, 2001).

Most importantly, Shaw and McKay's concept of social disorganization referred to an area in which the combined effects of an absence of economic resources, a highly mobile population, and cultural uncertainty produced unstable social institutions. The main problem with this conceptualization of social disorganization is that it confounds neighborhood structure and control systems. Kornhauser (1978) has argued that their concept is unclear and ambiguous. In fact, while neighborhood social process is a one important component of social disorganization theory, an operational definition of neighborhood processes are seldom addressed in Shaw and McKay's classical model. Fortunately, a loose concept of neighborhood processes has been redefined by several prominent current scholars (e.g. Bursik, 1988; Sampson, Morenoff, & Gannon-Rowley, 2002; Wilson, 2009).

Many of the most prominent theoretical works published in the latter 1980s and 1990s (e.g. Bursik, 1988; Sampson & Groves, 1989; Sampson, Rausdenbush, & Earls, 1997; Wilson, 1987) proposed that the concept of neighborhood structure is related to socioeconomic status and demographic composition, whereas the concept of social disorganization is an inability of neighborhood residents to achieve shared values and to effectively maintain informal social controls. As a consequence, contemporary social disorganization models consist of two parts: neighborhood structure and social process. The model assumes that social processes are mediators of neighborhood structure and outcomes.

In current studies, many researchers incorporate some socio-economic indicators from census data to determine the concept of neighborhood structure that generally includes subscales for concentrated disadvantage, residential stability, racial composition, immigrant concentration, family disruption, and urbanization (Morenoff, Sampson, & Raudenbush, 2001; Sampson et al., 1989, 1997). In a meta-analysis of 214 quantitative neighborhood studies from 1960 to 1999, Pratt and Cullen (2005) revealed that racial heterogeneity, residential instability, and concentrated disadvantage were important causes of social disorganization. In particular, concentrated disadvantage is measured by the following census index: average per-capita income, the percentage of unemployment, the percentage of residents below the poverty threshold, the percentage of residents without high school degrees, the percentage of female-headed households, and the percentage of those receiving public assistance.

Almost all recent current neighborhood studies focus on how concentrated disadvantage across neighborhoods influences crime/antisocial behavior. The studies indicate that the measure of concentrated disadvantage is the most reliable neighborhood characteristic to inhibit residents' ability to establish effective informal social control or social ties (e.g. Mazerolle, Wickes, & McBroom, 2010; Sampson, Rausdenbush, & Earls, 1997; Simons et al., 2005). For example, Bursik and Grasmick (1993) argued that the greatest shortcoming of Shaw and McKay's theory is the failure to consider relational networks that pertain to public spheres of control. They proposed the systemic model and indicated that neighborhood social ties are a mediator of the effect of neighborhood characteristics on crime/deviance. Another example, Sampson and colleagues (1997, 2002) included informal social control and social ties to identify specific causal mechanisms and to propose a new concept of collective efficacy that involved the extent to which informal social cohesion and trust occurred within neighborhoods and the degree to

which neighborhoods monitored the behavior of their residents. It has been well documented that collective efficacy mediates the association between concentrated disadvantage and crime/deviance (Browning et al., 2008; Maimon & Browing, 2010; Odgers et al., 2009).

In summary, social disorganization theory emphasizes the roles of structural characteristics and social processes in neighborhoods. The empirical model is developed by testing social ties/collective efficacy as a mediator of concentrated disadvantage on human behavior.

LIMITATIONS OF SOCIAL DISORGANIZATION THEORY: DO GENDER AND AGE MATTER?

Classical social disorganization theory assumes that people living in disadvantaged neighborhoods are more likely to engage in crime or deviance than those living in advantaged neighborhoods. A number of feminist scholars (e.g. Brooks-Gunn et al., 1993; Chesney-Lind & Bloom, 1997; Fagan & Wright, 2012; Zahn & Browne, 2009; Zimmerman & Messner, 2010) have charged that traditional neighborhood studies were presumed to be gender neutral, focused disproportionately on males, ignored women's experience, or simply used gender as a control variable. They believed that neighborhood context influences how people behave in gendered ways (Belknap, 2007; Chesney-Lind, 1989; Cobbina, Miller, & Brunson, 2008). In particular, feminist scholars stress that the conceptualization of gender is embedded in social context, and women and men have unique experiences in their neighborhoods (Cobbina, Miller, & Brunson, 2008; Miller & White, 2006).

Recently, neighborhood studies have found evidence that females and males tend to have different experiences in their neighborhoods (Leventhal & Brooks-Gunn, 2004). Girls are less

likely to be involved in neighborhood gangs (Esbensen et al., 1999) and are more likely to use risk-avoidance strategies in poor communities to protect themselves against crime or sexual assaults (Cobbina, Miller, & Brunson, 2008). Zimmerman and Messner (2010) found that males had higher levels of violence than females within advantaged neighborhoods. Therefore, important questions remain regarding neighborhood effects on well-being among women.

On the other hand, although neighborhoods consist of individuals of diverse ages, most researchers utilize adolescent samples (Bellair & McNulty, 2005; Odgers et al., 2009; Simons et al., 2005), largely omitting adult behavior from their analysis. During the past decade, increasing evidence suggests that neighborhood social ties mediate the association between concentrated disadvantage and adult health status (Valerie, Beggs, & Hurlbert, 2011; Vartanian & Houser, 2010). However, little is known about whether and how neighborhood effects influence adult crime/antisocial behavior. In particular, very few studies have considered the effect of concentrated disadvantage on crime/antisocial behavior among adult African American women. Using a sample of adult African American women, the dissertation attempts to replicate findings of prior studies that have found support for the social disorganization model.

LIMITATIONS OF SOCIAL DISORGANIZATION THEORY: BIOSOCIAL MODELS

In addition to neglecting social disorganization's effect on adult women samples, previous researchers have focused on a macro-level model which limits the investigation of individual variation in neighborhood contexts (Elliott et al., 2006; Farrington, 1993; Meier et al., 2008). Thus, while several studies have supported that neighborhood structures and process are associated with well-being, they do not take into account the question of why so much

heterogeneity is present among individuals residing in the same neighborhood (Barnes & Jacobs, 2013; Brody et al., 2001; Elliott et al., 2006; Simons et al., 2005).

In contrast to purely structural models, recent studies find that neighborhood effects on crime/antisocial behavior are shown to vary depending upon individual characteristics and/or experiences (Brody et al., 2001). For example, a number of studies find that neighborhood effects on adolescent delinquency are based upon adolescents' perceptions of parenting practices (Simons et al., 2005). Other studies provide evidence that the relationship between neighborhood factors and antisocial behavior is moderated by temperament or impulsivity; impulsive individuals are more sensitive than others to the influences of neighborhood contexts (Bush et al., 2010; Lynam et al., 2000).

During the past decade, increasing evidence suggests that genetic variation often interacts with the environmental context to influence the probability of particular human behaviors (Beach et al., 2012; Caspi et al., 2003; Freese & Shostak, 2009; Guo, Roettger, & Cai, 2008; Shanahan et al., 2008). Moreover, Jay Belsky and his colleagues (Belsky, Bakermans-Kranenburg, & von IJzendoorn, 2007; Belsky & Pluess, 2009) reviewed over 20 studies and found two major perspectives relating to the interaction pattern of genes and the environment. One is the diathesis-stress perspective, and another is the differential susceptibility perspective.

The diathesis-stress perspective assumes a fan-shaped interaction, whereby particularly vulnerable genotypes amplify the probability that exposure to some adverse social conditions (e.g., inept parenting, stressful life events) will lead to problem behaviors (Belsky & Pluess, 2009). In contrast, the differential susceptibility perspective emphasizes that some individuals are more sensitive to social environmental context than others. This perspective posits that the individuals most vulnerable to adverse social environments may also reap the greatest benefits

from more favorable environments (Belsky & Pluess, 2009). Using a GLM model, support for the differential susceptibility perspective is evident when the slopes for the gene by environmental interaction show a cross-over pattern with the susceptibility group showing worse outcomes than the comparison group when the environment is negative but demonstrating better outcomes than the comparison group when the environment is positive (Simons & Lei, 2013). Despite the vast literature on social disorganization theory, no studies have looked at the interactive and intervening effects of neighborhood processes and genotype.

Given these limitations, Study 1, as shown in Figure 1.1, fills a theoretical gap by examining whether the neighborhood-level concentrated disadvantage and social ties for adult women antisocial behavior differ by individuals who carry different variants of genes. In addition, I test whether these gene-neighborhood interactions are more consistent with a diathesis-stress or differential- susceptibility perspective. Building on the social disorganization model, the mediating effect of neighborhood social ties is also expected. I examine whether the effect of Gene × concentrated disadvantage on antisocial behavior is explained by the effect of Gene × neighborhood social ties.

BROKEN WINDOW THEORY

In addition to neighborhood social ties or collective efficacy, the concept of neighborhood disorder is another important neighborhood process that links neighborhood structural characteristics to a risk for crime/deviance and poor health status (Cohen et al., 2003; Leventhal & Brooks-Gunn, 2000; Sampson, 2012).The broken windows theory was developed by James Q. Wilson and George Kelling in 1982. They observed that signs of physical deterioration (e.g. graffiti, abandoned buildings, and broken glass) or signs of social deterioration

(e.g. the presence of prostitutes or homeless people) are especially important for understanding crime and violence in neighborhoods because visible signs of disorder convey the message collective apathy and lead to more disorder. Basically, signs of neighborhood order are implicitly about a lack of informal social control in the neighborhood.

Similar to neighborhood social ties, neighborhood disorder also mediates the effect of neighborhood disadvantage and crime/deviance (Sampson, 2012). The literature has provided strong evidence that the effect of neighborhood disorder is related to crime, delinquency, intimate partner violence, and a fear of crime (Cunradi, 2009; Steenbeek & Hipp, 2011; Wyant, 2008) and is a mediator of concentrated disadvantage on these outcomes (Jones, Pebley, & Sastry, 2011; Mrug & Windle, 2009).

THE STRESS PROCESS MODEL WITH THE BROKEN WINDOW THEORY

Although neighborhood contexts fundamentally influence crime/deviance, the extent to which neighborhood characteristics affect physical health is unclear. The reason is that social ties or a lack of informal social control cannot be assumed as a mediator of the effect of concentrated disadvantage on health outcomes. Recently, medical sociologists have suggested incorporating classical neighborhood theories into the stress process model (Hill, Ross, & Angel, 2005; Latkin & Curry, 2003; Pearlin et al., 2005). The stress process model posits that health inequalities are caused by differential exposure to social stress environments that impair the immune system's ability (Pollitt et al., 2008; Turner, 2010). For example, studies have found that when individuals are confronted with a stressful environment, the body releases adrenaline, noradrenaline and cortisol hormones which cause suppression of immune function (Miller, Chen, & Parker, 2011; Taylor, Way, & Seeman, 2011). In particular, their findings indicated that neighborhood context

is a common source of chronic stressors because both social and economic stressors tend to cluster in disordered neighborhoods where unequal socioeconomic opportunity structures exist (Aneshenel, 1992; Hill, Ross, & Angel, 2005; Pearlin, 1989, 2005). Thus, people living in disordered neighborhoods are more likely to trigger the proliferation of stressors, which in turn are associated with poor physical health.

Over the last two decades, a great deal of evidence has been obtained in support of the stress process model. The results suggest that the magnitude of neighborhood disorder on health outcomes remains statistically significant, even after controlling for concentrated disadvantaged, social ties, and individual socioeconomic characteristics (Aneshensel & Sucoff, 1996; Cohen et al., 2003; Franzini et al., 2005; Holmes & Marcelli, 2012; Lee & Cubbin, 2003; Morenoff, 2003; Nazmi et al., 2010;), and the impact of neighborhood disorder on self-reported health status is mediated by psychological and physiological distress (Hill, Ross, & Angel, 2005; Ross, 2000).

While the relationships among neighborhood disorder, distress, and physical health problems are found, the physiological distress mechanisms underlying this relationship remain unclear. First of all, much research to date has used self-reported instruments to measure physiological distress (Hill, Ross, & Angel, 2005). Nevertheless, self-reported data are vulnerable to the inflation of the relationships among variables by "common method variance" biases (Podsakoff et al., 2003). In the past decade, social and natural scientists have used biomarkers to predict physical and mental health status. Unlike self-reported measures, biomarkers are objective measurable biological parameters that relate to specific behavioral states, physical attributes, and cognitive traits. Molecular biologists have provided evidence that inflammation as one of biomarkers represent dysregulated immune systems that predict the likelihood of certain cancers, cardiovascular diseases, neurological disorders atherosclerosis,

diabetes, and chronic illness (Browning, Cagney, & Iveniuk, 2012; Gouin, Hantsoo, & Kiecolt-Glaser, 2011; Gouin et al., 2012; Hurst et al., 2001; Jones et al., 2001; Jones & Rose-John, 2002; Shariat et al., 2001). To address this issue, this dissertation focuses not only on subjective self-report health but also on objective biomarkers. Second, recent studies have reported that genetic variation often interacts with the environmental context to influence health outcomes (Beach et al., 2012; Brody et al., 2013; Hartley et al., 2012; Romanowicz et al., 2012), but the processes by which genetic makeup might moderate the relationship between neighborhood disorder and health have not been examined. Finally, some neighborhood research has demonstrated that social support buffers the effects of neighborhood disorder on health (Diez-Roux & Mair, 2010; Foster & Brooks-Gunn, 2013). In particular, studies have suggested that marital status is one of the important sources of social and emotional support because marriage provides a source of emotional support, material sources, and social connectedness (Turner & Roszell, 1994; Soons & Liefbroer, 2008). For example, Kaplan and Kronick (2006) reported that death rates were lower in married people than in those who were single or divorced.

Given these considerations, the main purpose of Study 2 is to examine the impact of neighborhood disorder on physiological indicators of health in adulthood. As shown in Figure 1.1, I examine whether adult African American women who live in disordered neighborhoods are more likely to report poor health and to have elevated inflammatory responses. In addition, I expect that inflammatory burden is a mediator of neighborhood disorder on self-reported health. I also hypothesize that both marital status and genetic makeup have a moderating effect on the relationship between neighborhood disorder and physical health.

CODE OF THE STREET THESIS: BEYOND NEIGHBORHOOD STRUCTURE

Over the past two decades, "bringing culture back in" is an important topic in neighborhood research because people learn the cultural code through their personal experiences within their own neighborhood context. Furthermore, cultural adaptations as informal rules guide how people interact with one another and interpret their environments (Wilson, 2009). Studies of neighborhood cultures can be traced back to Shaw and McKay's ([1942] 1969) cultural transmission theory. They observed that crime rates in neighborhoods remain relatively stable over time despite changing demographic compositions and claimed that deviant norms and values are culturally transmitted from one generation to the next in the same neighborhood context.

During the 1950s and 1960s, Cohen (1965) and Cloward and Ohlin (1960) extended cultural transmission theory. They indicated that adaptations to strain are influenced by individuals' associations with their local communities and also dependent upon their reference groups. They emphasized that the strain-crime relationship is explained by subculture processes. Unfortunately, the classic theories were criticized for being too simplistic and lacking a complete definition of the concept of subculture and the link between neighborhood structure and culture (Kornhauser, 1978).

In the early 90s, Elijah Anderson's studies expanded on the classical sub-cultural theories to link sub-cultural and structural factors (Anderson, 1994). Based on ethnographic research in Philadelphia inner-cities, Anderson (1999) provided insight into how neighborhood structures are related to cultural adaptation. He observed that residents of concentrated disadvantaged, racially, and socially isolated neighborhoods do not trust the police or other formal control systems and are more like to adopt oppositional culture. Violence, tough identity, and retribution

are viewed as functional mechanisms for maintaining and gaining respect, when people live in areas of structural disadvantage and deprivation.

According to his observations, Anderson proposed the concept of street code and defined it as a set of informal rules governing interpersonal public behavior and a cultural frame in which violence and toughness are viewed as a means of gaining respect and obtaining status. The concept of respect is at the root of the street code. In addition, the code is a cultural adaptation to disadvantaged neighborhood contexts where racial discrimination, alienation, poor family relationships, unemployment, and violence are common. Individuals embracing the street code have a profound lack of faith in the criminal justice system and are likely to endorse violence as a means to gain respect. Therefore, adopting the street code is a critical link between neighborhood structure and violence because it provides a guide for people regarding how to handle interpersonal conflict in disadvantaged neighborhood settings. Basically, Anderson's theoretical model stresses that adopting the street code is a mediator of the effects of neighborhood/social characteristics on violence. In other word, violent behavior is shaped by the street code, and this code thrives in places lacking neighborhood structural resources and demoralizing environments, where respect is an indispensable ideal.

Anderson's code of the street thesis is widely documented. Numerous empirical studies have found that African-American youths who resided in disadvantaged neighborhoods, who had been raised in street-oriented families, and who felt racial discrimination were likely to adopt street code, which in turn increased the likelihood of engaging in violence (Baumer et al., 2003; Brezina et al., 2004; Matsuda et al., 2013; Nowacki, 2012; Oliver, 2006; Parker & Reckdenwald, 2008; Sampson, Morenoff, & Raudenbush, 2005; Stewart, Simons, & Conger, 2002; Stewart and Simons, 2006). Simons and his colleagues (2012) indicated that adopting the street code as a

type of cognitive schema can be used to understand how individuals employ violence to resolve conflict while others do not. Several researches also indicated that the code of the street thesis is true not only for males but also for females. (Brunson & Stewart, 2006; Jones, 2010; Ness, 2004).

Furthermore, while the street code is an adaptation to living in disadvantaged neighborhoods, Anderson claimed that there is no single cultural value in these areas. In fact, social behaviors in disadvantaged neighborhoods are characterized by two codes: the street and descent codes (Anderson, 1999, pp. 37-53). Many residents living in disadvantaged neighborhoods follow the descent code, which reflect middle-class values and the activities of mainstream society. In contrast to the descent code, street residents placed less emphasis on work and education because of their deep distrust of the formal social structure.

More importantly, while traditional sub-cultural (e.g. Cloward & Ohlin, 1960; Cohen, 1965) and cognitive psychology scholars (Beck, Freeman, & Davis, 2004; Young, Klosko, & Weishaar, 2003) tend to assume that core values and/or beliefs are relatively stable cognitive structures that are stored in individual memory, Anderson (1999, p .36) argues that street code adoption is dynamic and that "most people code switch from time to time, depending on how they read a particular [social] situation" (p. 189). He used "code-switching" to describe the alteration of behavior between "decent" and "street" code values. However, Anderson does not clearly demonstrate whether street code is changed over time. In fact, neuroscientific research has provided evidence that cognitive schemas can change over time in response to social contexts (Davidson & Begley, 2012).

Despite strong evidence suggesting that demoralizing environments lead to street code adoption and predict violence, no studies take into account the dynamic nature of the street code

over time. Furthermore, prior research has largely overlooked the extent to which change in adoption of the street code over time can be explained by change in the measures of socially demoralizing environments. Thus, the specific mechanisms of the dynamics of street code remain unclear. Unlike many cross-sectional studies, this dissertation utilizes a longitudinal design and a latent growth curve model with time-varying covariates examining the temporal nature of the street code.

In Study 3, I examine whether change in adopting the street code occurs over time and whether change in demoralizing environments in local neighborhoods is associated with change in street code adherence. Finally, I test the mediating effect of change in street code adherence in the relationship between change in demoralizing social environments and change in violence.

SUMMARY: CONCEPTUAL MODEL

Although a substantial body of literature has documented the association between structural characteristics of neighborhoods and well-being, the detailed mechanisms are unclear. In particular, individual well-being as an umbrella construct includes aspects of behavioral and personal adjustment outcomes that are related to different neighborhood processes. To better understand the linkages among neighborhood structure, neighborhood processes, and different well-being outcomes, this dissertation consists of three studies as shown in Figure 1.1.

Using an Adult African American women sample, Study 1 examines the effect of neighborhood social ties as a mediator of concentrated disadvantage and anti-social behavior and whether such a relationship is moderated by genetic makeup. Study 2 tests whether neighborhood disorder is an important neighborhood process in explaining physical health. In addition, I examine the stress process mechanisms through an objective inflammation biomarker

and assume that the relationship between neighborhood disorders and self-reported health is mediated by systemic inflammation. These relationships are also likely moderated by both genetic makeup and marital status. Finally, using an African American youth sample from late childhood to young adulthood, Study 3 examines the dynamics of the street code over time and assumes that changes in street code adherence occur over time and are likely explained by fluctuations in the socially demoralizing nature of local environments and individual exposure to violence.

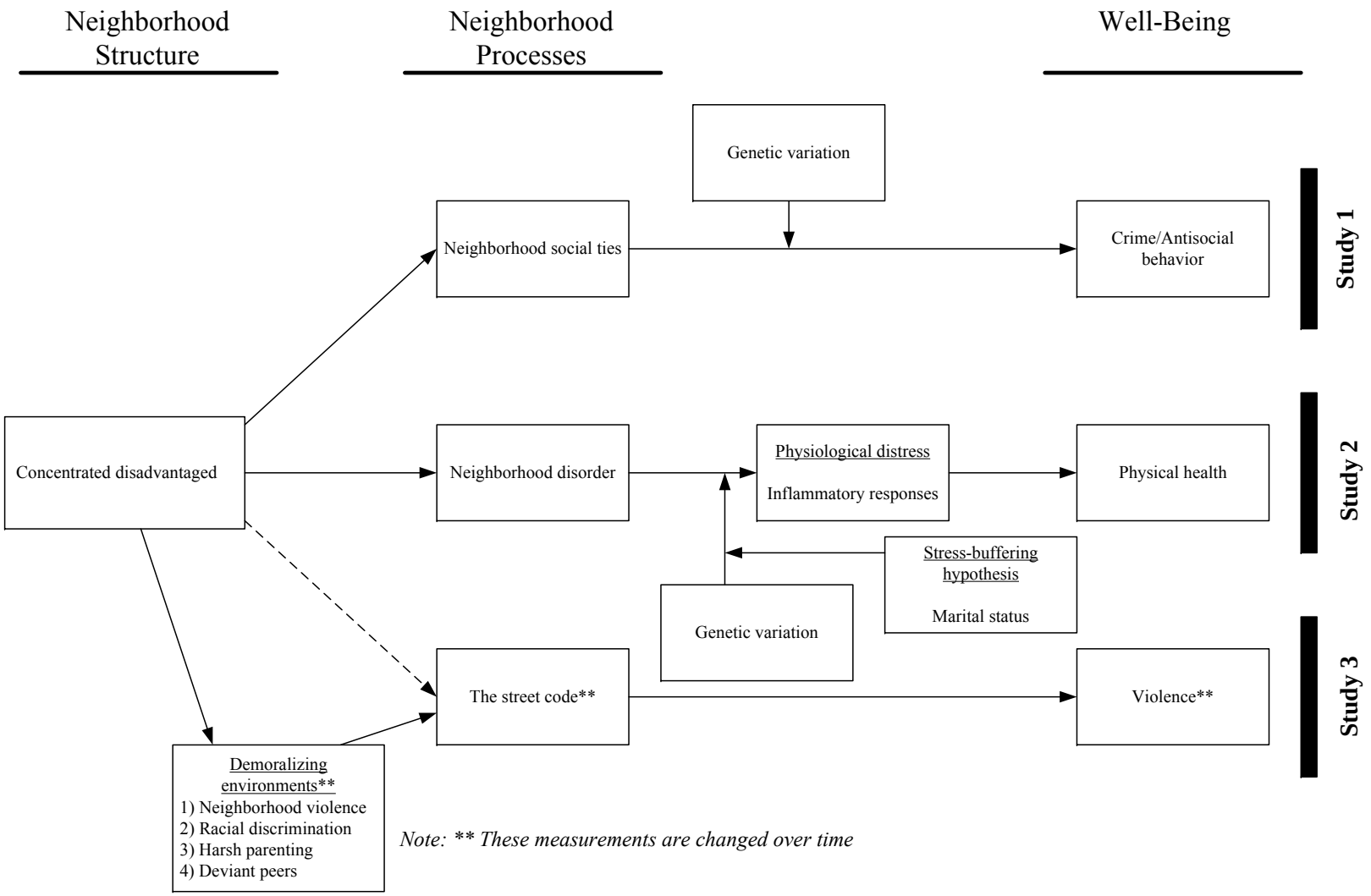

Figure 1.1.
Conceptual Model of the Dissertation

CHAPTER 2

STUDY 1: NEIGHBORHOOD EFFECTS, GENETIC VARIATIONS, AND ADULT ANTISOCIAL BEHAVIOR

In the past 20 years, there has been a proliferation of studies investigating neighborhood explanations for crime and delinquency (Leventhal & Brooks-Gunn, 2000; Sampson, Morenoff, & Gannon-Rowley, 2002). Most of this research has concentrated on socio-demographic measures of neighborhood quality. Drawing upon social disorganization theory, the most widely used indicators have been poverty, income, unemployment, female-headed households, public assistance, and racial/ethnic heterogeneity. Several studies have reported that such variables are related to delinquency and crime and that their effect is, in large measure, indirect through their impact upon social ties and informal social control (Bursik & Grasmick, 1993; Sampson, 2012). Importantly, however, almost all of this research has focused upon adolescent males (Bellair & McNulty, 2005; Odgers et al., 2009; Simons et al., 2005). Thus it is not clear how well this model explains adult antisocial behavior, especially that of adult women (Belknap, 2007; Chesney-Lind & Pasko, 2013; Kroneman et al., 2004; Zahn & Browne, 2009). The first goal of the present study is to replicate findings of prior neighborhood studies using a sample of adult African American females.

In addition to neglecting the effect of community disorganization on women, neighborhood studies have tended to employ a macro focus that ignores individual variation (Leventhal & Brooks-Gunn, 2000). This approach cannot explain heterogeneity in the behavior

of those living in the same disadvantaged neighborhood. In contrast to purely structural models, multi-level studies find that neighborhood effects on delinquency and crime are moderated by individual characteristics and/or experiences (Barnes & Jacobs, 2013; Bush et al., 2010; Simons et al., 2005). Recently, a profusion of studies has reported that genetic variation often interacts with the environmental context to influence the probability of various behaviors (Caspi et al., 2003; Dick et al., 2011; Freese & Shostak, 2009; Guo, Roettger, & Cai, 2008; Shanahan et al., 2008). Most importantly, several recent scholars (e.g. Bakermans-Kranenburg & Van Ijzendoorn, 2011; Beach et al., 2012; Belsky et al., 2007, 2009, 2013; Kochanska et al., 2011; Simons et al., 2011; Simons & Lei, 2013; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012) have proposed the differential susceptibility view of gene - environment interaction. This perspective argues that the polymorphisms used in most G×E studies exert their influence by augmenting susceptibility to social context, whether that environment is adverse or supportive. Thus those persons most vulnerable to adverse social environments are the same ones who reap the most benefit from environmental support.

Building upon these findings, the present study investigates the extent to which variation in the serotonin transporter gene (*5-HTTLPR*), a gene that have been linked to adolescent and adult men's antisocial behavior (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Brody et al., 2009, 2011; Homberg & Lesch, 2010; Sakai et al., 2006; Simons et al., 2011, 2012; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012), moderates the effect of neighborhood disadvantage and social ties on adult women's antisocial behavior. The current study uses multilevel data from a sample of approximately 400 African American women. Such a sample is particularly relevant for examining these ideas given the wealth of data indicating that, in general, African-American women have higher rates of crime and antisocial behavior

than women in other ethnic groups (Belknap, 2007) and are more apt to reside in extremely disadvantaged neighborhoods (McNulty, 2001; Peterson & Krivo, 2010).

NEIGHBORHOOD CHARACTERISTICS AND ANTISOCIAL BEHAVIOR

Neighborhood studies can be traced back to the early twentieth century. Shaw and McKay ([1942] 1969) were among the first to argue that residing in a disadvantaged neighborhood increases the probability that an individual will engage in delinquency, crime and other antisocial behaviors. They argued that this association exists because informal social control is weak in disadvantaged neighborhoods. Their perspective gained popularity in the 1990s when sociologists and criminologists began to focus upon the avenues whereby community disadvantage might produce this effect (Sampson et al., 2002; Sampson 2012). Bursik and Grasmick (1993) argued that the greatest shortcoming of Shaw and McKay's social disorganization theory was its failure to consider relational networks that pertain to the public sphere of social control. They proposed that neighborhood disadvantage affects deviance and crime through its impact on neighborhood social ties and cohesion. Disadvantage makes it difficult for residents to establish the social cohesion, ties, and common values necessary to constrain individuals from engaging in crime and other deviant behaviors. In other words, the relationship between community characteristics and antisocial behavior is mediated by the level of cohesion or social ties that exist between residents in the area.

Several studies have reported support for this perspective (Rountree et al., 1999; Browning et al., 2004, 2008; Sampson, Raudenbush, & Earls, 1997). However, while neighborhoods consist of individuals of diverse ages, most researchers utilized child and adolescent samples (Bellair & McNulty, 2005; Odgers et al., 2009; Simons et al., 2005), largely

omitting adult antisocial behavior from their analysis. During the past decade, increasing evidence suggests that neighborhood social cohesion or ties mediate the association between neighborhood characteristics and adult physical and mental health problems (Valerie et al., 2011; Vartanian & Houser, 2010). Less is known about whether this neighborhood effect also holds for adult antisocial behavior.

Moreover, feminist scholars have claimed that the community disorganization perspective remains a "male theory" (Belknap, 2007; Chesney-Lind & Pasko, 2013; Zahn & Browne, 2009). They have charged that traditional neighborhood studies were presumed to be gender neutral, focused disproportionately on males, ignored women's experience, or simply used gender as a control variable. For instance, the data provided by Shaw and McKay ([1942] 1969) in support of their perspective focused only on boys. And, subsequent tests of the theory have relied almost exclusively upon male samples (e.g. Bares & Jacobs, 2013; Beaver et al., 2012).

Importantly, recent neighborhood studies have found evidence that females and males tend to have different experiences in their neighborhoods (Cobbina, Miller, & Brunson, 2008; Leventhal & Brooks-Gunn, 2004; Zimmerman & Messner, 2010). Therefore, it is uncertain as to whether the results of prior neighborhood studies generalize to women (Obeidallah et al., 2004; Zahn & Browne, 2009). Although the social disorganization framework has proven to be a powerful framework for explaining male antisocial behavior, it is not clear that it is a useful perspective for explaining variations in female offending (Chesney-Lind & Pasko, 2013; Zahn & Browne, 2009).

The first goal of the present study is to address this gap in the literature. Using a sample of adult African American women, I attempt to replicate findings of prior studies that

have found support for the community disorganization framework. Using multilevel data, I examine the extent to which women living in areas of concentrated disadvantage have higher levels of antisocial behavior than those living in more advantaged areas. Further, I investigate whether the influence of concentrated disadvantage on women's antisocial behavior is mediated by neighborhood social ties.

GENE-NEIGHBORHOOD INTERACTION

Neighborhood studies largely ignore individual-level factors, focusing instead on links among neighborhood structure, neighborhood processes, and human behavior (Leventhal & Brooks-Gunn, 2000; Sampson et al., 2002). The primary assumption guiding this research is that people who live in the same neighborhood are more similar to one another than to those who live in different neighborhoods. However, empirical studies reveal that not all individuals, indeed not even the majority of individuals, from disadvantaged neighborhoods become deviant or antisocial (Elliott et al., 2006). Thus, a central question remains: Why is there so much heterogeneity in the behavior of individuals residing in the same neighborhood? Understanding which individual characteristics influence the relationship between neighborhood contexts and human behavior is crucial to the advancement of neighborhood research.

In the past decade, a number of social scientists have attempted to make their models more precise and biologically integrated by incorporating genetic effects into their theoretical frameworks (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Beach et al., 2012; Beaver et al., 2012; Brody et al., 2011; Caspi et al., 2003; Guo, Roettger, & Cai, 2008; Moffitt, Caspi, Rutter, 2006; Shanahan et al., 2008; Simons et al., 2011). A growing body of literature suggests that genetic variability moderates the impact of the social environment on human behavior (Duncan

& Keller, 2011). Although such G×E research has increased dramatically in recent years, only a few studies have investigated gene-neighborhood interactions (e.g. Barnes & Jacobs, 2013; Beaver et al., 2012; Hart & Marmorstein, 2009; Simons et al., 2012). For example, using samples from the National Longitudinal Study of Adolescent Health (Add Heath), Barnes and Jacobs (2013) found that men with one or more copies of the risk allele for the dopamine receptor gene show higher levels of violent behavior than those with no copy when they live in disadvantaged neighborhoods. While these studies provide valuable insights regarding gene-neighborhood interactions, they are limited in that they exclude women in their analyses and ignore the role of neighborhood social ties and cohesion as an important mediator in social disorganization framework. The present study extends the focus of these studies in three respects.

First, the few G×E neighborhood studies that have been conducted focus upon children and adolescents. The current study extends this work by examining whether such G×E effects also operate for adult women. Second, whereas prior studies investigated the extent to which genes moderate the impact of neighborhood disadvantage on antisocial behavior, I go on to examine whether genetic variation also moderates the impact of neighborhood social ties on antisocial behavior. Finally, as part of this analysis, I examine whether the effect of G × neighborhood disadvantage on antisocial behavior is eliminated when the effect of G × neighborhood social ties is considered. In other words, I extend social disorganization theory by examining the extent to which the G × neighborhood disadvantage effect on antisocial behavior is mediated by the G × social ties effect on antisocial behavior.

Much of the research investigating the molecular genetic basis of aggressive and antisocial behavior has focused upon variation in the serotonin transporter gene (5HTT). The

serotonin transporter gene is involved in the regulation of serotonergic neurotransmission which has been linked to sensitivity to punishment and displeasure (see Carver, Johnson, & Joormann, 2008; Frank et al., 2007). It contains a functional polymorphism in the 5′ promoter region (*5-HTTLPR*) that consists of 14 or 16 repeats of a 20-22 base pair (bp) unit (Murphy, Lerner, Rudnick, & Lesch, 2004). Several studies have been shown that this polymorphism influences human behavior (Brody et al., 2009, 2011; Caspi et al., 2003; Homberg & Lesch, 2010; Sakai et al., 2006; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012). This research indicates that individuals with the short (s) allele, which is associated with reduced serotonin transporter expression and diminished mRNA for serotonergic neurotransmission, are more likely to engage in conduct disorder, aggression, and/or antisocial behavior than persons with the long (l) allele (Heils et al., 1996; Reif et al., 2007; Sakai et al., 2006).

In most cases, variation in this polymorphism does not show a direct effect on antisocial behavior; rather, they exert their influence by moderating the effect of the social environment. A number of studies have reported that the s-allele of *5-HTTLPR* increases the probability that an adverse social environment will lead to antisocial behavior (Beach et al., 2012; Brody et al., 2009, 2011; Caspi et al., 2003; Reif et al., 2007; Homberg & Lesch, 2010; Simons et al., 2011, 2012). Thus, in the present study, I expect that both neighborhood disadvantage and weak social ties will have a greater impact on the antisocial behavior of women with one or two copies of the s-allele of *5-HTTLPR* than upon those with no copies of this allele.

MODELS OF G×E INTERACTION

Genetically informed social science requires models of the manner in which genetic variables combine with environmental context to influence behavioral outcomes (Freeze, 2008;

Shanahan & Hofer, 2005, 2011; Simons & Lei, 2013). The model utilized in the vast majority of G×E studies of antisocial behavior, as well as of other adjustment problems, assumes that allelic variation in a particular gene amplifies the probability that exposure to some adverse social condition (e.g., abusive parenting, racial discrimination, economic hardship) will lead to antisocial behavior. In psychology and psychiatry, this is labeled the diathesis-stress perspective. This model asserts that some individuals possess alleles that operate as diatheses to amplify the effects of environmental stress or adversity. It assumes that some individuals are by nature more vulnerable than others as they possess dysfunctional "risk alleles" that foster maladjustment in the face of deleterious environmental conditions. Support for the diathesis-stress perspective is evident when a graph of the G×E effect shows a fan-shape such that increases in adversity are associated with a greater increase in antisocial behavior for those with the risk allele than for those without the risk allele (Belsky & Pluess, 2009; Simons & Lei, 2013). Figure 2.1a depicts a hypothetical example of this perspective.

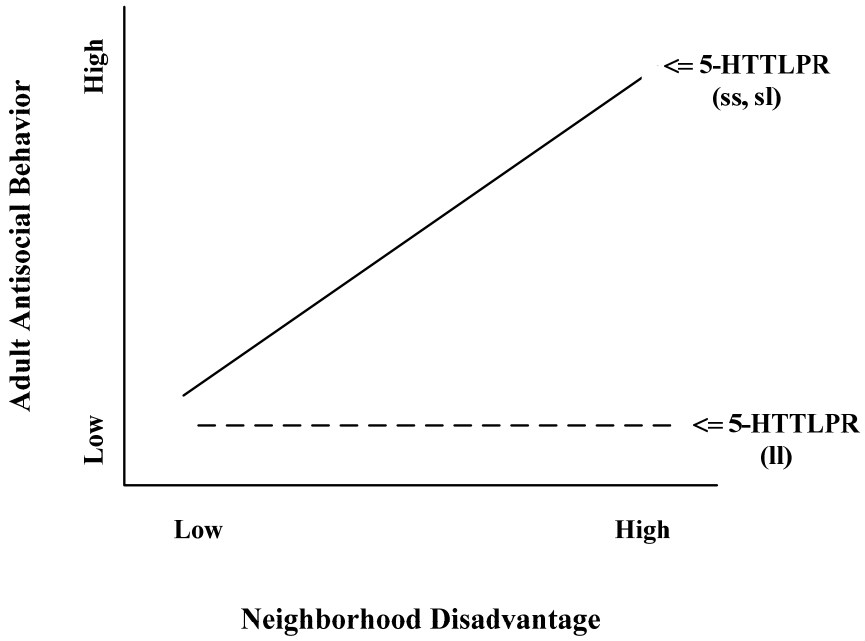

Figure 2.1a. The Diathesis-Stress Hypothesis

The diathesis-stress model, with its focus on risk alleles, is contradicted by the fact that over the past several thousand years evolution seems to have conserved these various alleles (Ellis, et al., 2011; Homberg & Lesch, 2011). While truly dysfunctional genetic variants should largely disappear over time, most of the so called risk alleles studied by behavioral science researchers are highly prevalent, often being present in 40 to 50 percent of the members of the populations being investigated (Ellis, et al., 2011). Thus contrary to the negative view usually taken of these alleles, this suggests that, at least in certain contexts, these genetic variants must provide advantages over other genotypes. This view is an essential component of the alternative model of gene by environment interaction recently proposed by Jay Belsky and his collaborators.

Belsky and colleagues (Belsky, Bakermans-Kranenburg, & von IJzendoorn, 2007; Belsky & Pluess, 2009; Ellis et al., 2011) argue that the polymorphisms used in most G×E studies of child and adolescent adjustment exert their influence by augmenting susceptibility to social context, whether that environment is adverse or supportive. Thus those persons most vulnerable to adverse social environments are the same ones who reap the most benefit from environmental support. Belsky and colleagues label this view of G×E the differential susceptibility perspective. Their model assumes that some individuals are programmed by their genetic make-up to be more sensitive to environmental influence than others. In other words, they are more "plastic." Indeed, Belsky and his colleagues often refer to genetic variants thought to enhance sensitivity to social context as plasticity alleles. Furthermore, they indicated that the more plasticity alleles one carries, the more susceptible one will be to environmental influence. For example, using a composite measure of cumulative genetic plasticity, Belsky and Beaver (2011) revealed that individuals with multiple plasticity alleles scored lower than others on self-

regulation when reared by hostile parents, whereas persons with this genotype scored higher than others on self-regulation when reared by supportive parents.

Support for the differential susceptibility or plasticity argument is evident when the slopes for a gene by environment interaction show a crossover effect with the susceptibility group showing worse outcomes than the comparison group when the environment is negative but demonstrating better outcomes than the comparison group when the environment is positive (Belsky et al., 2007; Belsky & Pluess, 2009; Simons & Lei, 2013). Figure 2.1b provides a hypothetical example of the differential susceptibility perspective. In a recent article, Belsky and Pleuss (2009) reviewed scores of studies reporting a G×E effect on child or adolescent adjustment. Many of these studies focused on outcomes involving conduct problems and related deviant behaviors. Although these studies appeared to support a diathesis-stress model, Belsky and colleagues concluded that a careful inspection of the results pointed to a different interpretation as all of the studies included in the review showed a cross-over effect.

Respondents with so-called risk alleles showed more problem behavior than other genotypes when their environment was adverse but manifested fewer problems than other genotypes when either their environment was more supportive. Thus, rather than simply showing that some individuals are more vulnerable to adverse conditions than others, the data supported the idea that some people are genetically predisposed to be more susceptible to environment influence than others. The findings suggested that what were assumed to be risk alleles are in actuality plasticity alleles. In most of these studies, however, this pattern was not recognized or discussed because the authors were operating out of the diathesis-stress paradigm.

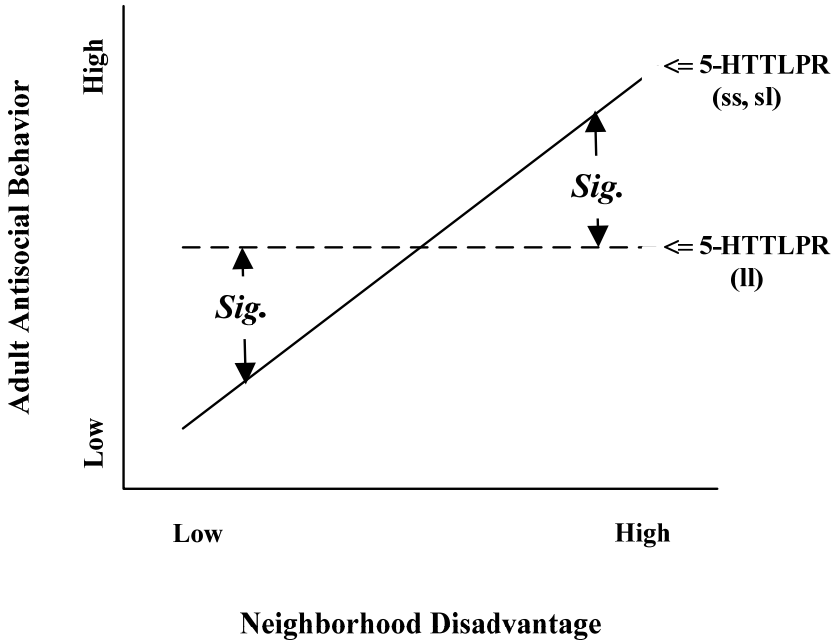

Figure 2.1b. The Differential Susceptibility Hypothesis

Recent meta-analyses of G×E studies (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012) have provided strong evidence that the genetic polymorphism of concern in the present study (*5-HTTLPR* s-allele) operate as susceptibility alleles. They are associated with increased problem behavior in adverse social environments but enhanced success in favorable social environments. I hypothesized that women with the s-allele of *5-HTTLPR* will show greater antisocial behavior than the l-allele of *5-HTTLPR* in response to an adverse neighborhood context, whereas they will show less antisocial behavior than the l-allele of *5-HTTLPR* when living in an advantaged neighborhood context.

METHODOLOGICAL CHALLENGE

In the present study, I graph all significant G×E interactions in an effort to determine whether the pattern best supports the diathesis-stress or differential susceptibility argument. Most recent studies have used the Johnson-Neyman technique or the pick-a-point approach to distinguish differential susceptibility from diathesis-stress. This procedure identifies regions of significance for interactions between genotypes and environmental variables (Bauer & Curran, 2005; Roisman et al., 2012; Simons, Lei & et al., 2011). However, research indicates that the statistical power to test differential susceptibility is often limited by range restrictions and variation in environmental measurements (Belsky, Pluess, & Widaman, 2013; Dick, 2011; Duncan & Keller, 2011). Testing for the differential susceptibility requires that one have data representing the full range of the social environment, from very adverse to very favorable, and this often is not the case. Indeed, I anticipate encountering this problem in the present study.

There is strong evidence that institutional racism has resulted in residential patterns where African Americans are over-represented in seriously disadvantaged neighborhoods while being under-represented in middle class areas (McNulty, 2001; Peterson & Krivo, 2010; Sampson, Morenoff, & Raudenbush, 2005). This skew in residential distribution is likely to result in there being too few cases, and hence limited variation and statistical power, to assess the interaction of genes with residence in advantaged neighborhoods.

To address the issue of statistical power with small sample sizes, Widaman et al. (2012) proposed a new procedure for evaluating the cross-over point of simple regression lines in order to determine the pattern of interaction effects. Unlike classical post-hoc tests, this new approach directly estimates the confidence interval of the cross-over point to test for G×E effects and to distinguish differential susceptibility from diathesis-stress. Support for the differential

susceptibility perspective requires that the simple regression lines cross within a range of values of the independent variable. Hence, if the confidence interval of the cross-over point includes that range of values on the independent variable, the data provides support for differential susceptibility. On the other hand, if the confidence interval is located outside of the range of values for the independent variable, the data supports the diathesis-stress model.

RESEARCH DESIGN FOR STUDY 1

Sample

I test my hypotheses using data from the Family and Community Health Study (FACHS), a multi-site investigation of neighborhood and family effects on health and development (see Beach et al., 2012; Simons et al., 2011). FACHS was designed to identify neighborhood and family processes that contribute to school-age African American children's development in families living in a wide variety of community settings outside the inner-city core. Each family includes a child who was in 5th grade at the time of recruitment. At the first wave, the FACHS sample consists of 889 African American children (411 boys and 478 girls) and their primary caregivers (PCs) (60 men and 829 women). At study inception, about half of the sample resided in Georgia and the other half in Iowa. The children averaged ten years of age (5th grade) at the beginning of the study in 1997-1998. Of the 889 PCs interviewed at Wave 1, 693 were interviewed again at Wave 5 (77.26% of the original sample). As part of wave 5 (2007-2008) data collection, PCs were asked to provide DNA for genotype analysis. Of the 693 participants, 549 (80%) agreed to DNA collection, and a saliva sample was obtained from 472 females. Successful genotyping for *5-HTTLPR* was achieved for 467 of these respondents (a call rate of 98.9%). Analyses indicated that those individuals who did not participate in the genetic

component of the study did not differ significantly from those who participated with regard to antisocial behavior, age, education, family structure, household income, or neighborhood characteristics.

Current Study Participants

The current study involves a two-level data structure, that is, individuals nested in neighborhoods. The neighborhood-level data was created using the 2000 census STF3A (Summary Tape File 3) that was geocoded with participant's residential addresses in 1996. Additional details regarding neighborhood data can be found in Stewart and Simons (2010). In the current study, analyses are based upon the 397 of 467 female respondents who were nested within 67 census tracts, were genotyped for *5-HTTLPR*, and provided data on all respondent measurements at the first wave. Participants included in the present study did not significantly differ from those excluded due to missing data with regard to neighborhood disadvantage and antisocial behavior at wave 1. Of the 397 respondents, fifty six percent self-identified as single parents, forty percent lived below the poverty line, and sixty two percent did not hold a high school diploma. The resulting sample had a mean age of 36.98 years, $SD = 7.92$, and an average per capita annual income of $6,456.

Procedures

The measures of neighborhood characteristics were created using the 2000 census data which was geocoded with participant's residential addresses at Wave 1. The questionnaires were administered in the respondent's home and took on average 2 hours to complete. The instruments were presented on laptop computers. Questions appeared in sequence on the screen, which both

the researcher and participant could see. The researcher read each question aloud and the participant entered an anonymous response using a separate keypad. In addition, participants were also asked to provide a saliva sample at Wave 5. It was frozen and shipped via courier to a laboratory at the University of Iowa.

Measures

Adult antisocial behavior was assessed with the Diagnostic Interview Schedule (DIS; Robins et al., 1997; Shaffer et al., 1993) which focuses on the symptoms of antisocial personality disorder (ASPD) listed in the Diagnostic and Statistical Manual of Mental Disorders, Version 4 (DSM-IV; American Psychiatric Association 1994, pp. 649-650). The measure consisted of 35 items (e.g. "have you stolen things or money by holding someone up," "have you sometimes pretended you were sick or injured to collect insurance, worker's compensation, or disability pay," "have you sometimes used a stick, knife, gun, bottle, or bat to hurt someone," and "have you often driven when you were high or drowsy on alcohol or drugs") rated on a dichotomous scale (0 = no, 1 = yes) designed to assess the seven ASPD lifetime symptoms under Criterion in the DSM-IV manual. The items were scored and clustered into the seven symptoms using diagnostic algorithms corresponding to DSM-IV criteria developed by the Division of Child and Adolescent Psychiatry at Columbia University (Lahey et al., 1996). Finally, a symptom count was obtained by summing scores for the seven diagnostic symptoms (0 = symptom is absent, 1 = symptom is present): 1) failure to conform to social norms with respect to lawful behaviors, 2) deceitfulness, 3) impulsivity, 4) irritability and aggressiveness, 5) reckless disregard for safety of self or others, 6) consistent irresponsibility, and 7) lack of remorse for the mistreatment of others. The maximum possible score of seven corresponds to a subject reporting that she had engaged in

acts pertaining to all of the different symptoms. Confirmatory factor analysis of the seven symptoms used to assess antisocial behavior produced factor loadings that ranged from .46 for impulsivity to .66 for irritability and aggressive. Coefficient alpha for the scale was .70.

Neighborhood social ties was assessed from four items, such as "You can count on adults in your neighborhood to watch out that children are safe and don't get in trouble," "Parents in your neighborhood know their children's friends," "Adults in your neighborhood know who the local children are," and "Parents in this neighborhood generally know each other." The response format for all these items was "1 = true," and "0 = false." Following the example of Raudenbush and Sampson (1999), a neighborhood-level measure of social ties was created summing and averaging across respondents within each of the 67 census tracts. A higher score indicates a higher level of neighborhood social ties. Coefficient alpha for the scale was .82.

Concentrated disadvantage was assessed with 2000 STF3A census tract data. Following previous studies (Sampson, Raudenbush, & Earls, 1997; Simons et al., 2005), the scale include five items: average per-capita income, the percentage of unemployment, the percentage of residents below the poverty threshold, the percentage of female-headed households, and the percentage of those receiving public assistance. To provide equal weight for each item, per capita income was reverse-coded, and I used factor scores obtained through principal-components analyses to form the scale. Factor loadings ranged from .74 for per capita income to .88 for the percentage of residents below the poverty threshold. Coefficient alpha for the measure was .89.

Heterogeneity of Racial composition was assessed by using census data regarding the percentage of white residents in the respondent's census tract in 2000 (M = 57.72, *SD* = 28.37) (Stewart et al., 2009). Previous studies have indicated that African Americans are more likely

than whites to experience anxiety, racial discrimination and to commit crime/deviance in predominately white affluent neighborhoods (Pattillo, 1998; Tatum, 1999).

Control variables. This study included five control variables that might influence the relationships among neighborhood variables and adult antisocial behavior, including high school graduation, single family status, age, residential history (moved =1), and family income below the federal poverty line (http://www.census.gov/hhes/www/poverty/data/threshld).

Genotyping. Participants were asked to contribute a saliva sample using Oragene™ DNA kits (Genotek; Calgary, Alberta, Canada). Those who chose to participate rinsed their mouths with tap water, and then deposited 4 ml of saliva in the Oragene sample vial. The vial was sealed, inverted, and shipped via courier to a laboratory at the University of Iowa, where samples were prepared according to the manufacturer's specifications. Genotyping was performed for VNTR (Variable Number Tandem Repeat) polymorphisms in *5-HTTLPR*.

Genotype at the *5-HTTLPR* located on chromosome 17q11.1-q12 has a functional polymorphism in the variable repeat sequence in the promoter region (Bradley, Dodelzon, Sandhu, & Philibert, 2005). The homozygous long allelic variant (16 or 18 repeats) is related to higher concentrations of 5-HTT messenger RNA and a greater rate of reuptake than the homozygous short allelic variant (14 repeats). A number of studies have provided evidence that the short (s) allele of *5-HTTLPR* is associated with conduct disorder, aggression, and/or antisocial behavior (Caspi et al., 2010; Sakai et al., 2006; Simons et al., 2011).

There are three main models for coding gene sequences (Lewis, 2002). Using the dominant model, individuals receive a score of 1 if they are either heterozygous or homozygous for the plasticity allele and a score of 0 if they are homozygous for the non- plasticity allele. The additive model counts the number of plasticity alleles for the gene (i.e., 0, 1, 2). Thus those

heterogeneous for the plasticity allele receive a 1 and those homogeneous for the allele received a two. Finally, using the recessive model individuals receive a score of 1 if they are homozygous for the plasticity allele and otherwise receive a zero 0. Consistent with prior research (Beach et al., 2012; Belsky & Beaver, 2011; Simons et al., 2011, 2012; Van IJzendoorn, Belesky, & Bakermans-Kranenburg, 2012), the current study used the dominant model. I treated *5-HTTLPR* as dichotomous variables where individuals received a score of 1 if they were carrying at least one copy of the plasticity allele and a score of 0 if they were homozygous for the non-plasticity allele.

Thus for *5-HTTLPR*, 1 = at least one short allele (ss/sl); 0 = pair of long alleles (ll). Using this criteria, 177 (44.6%) had at least one copy of the short allele. Among the 397 respondents, 7.3% were homozygous for the short allele (ss), 37.3% were heterozygous (sl), and 55.4% were homozygous for the long allele (ll). Using the Hardy-Weinberg equilibrium test, the observed distribution of *5-HTTLPR* polymorphism did not differ significantly (chi-square = .06, $df = 1$, $p = .82$) from that predicted on the basis of simple Mendelian inheritance.

Analytic Strategy

Because respondents are clustered within neighborhoods, the error terms of regression models are not independent, which leads to an underestimation of standard errors. To avoid this problem and given that antisocial behavior is a count variable, I used the multilevel Poisson model (Snijders & Bosker, 2012) available in the non- linear mixed effects function of the "nlme" R-package (Pinheiro et al., 2009). This model allowed us to generate a simultaneous estimation of relationships across hierarchical levels and to decompose the total variation in the dependent variable into variances at individual and neighborhood levels.

Using the multilevel Poisson model begins with the unconditional model to estimate how much variability in adult antisocial behavior exists at each level. This model has no predictors at the respondent and neighborhood levels, as shown in the following equation:

$$\log(\lambda_{ij}) = \gamma_{00} + u_{oj} \qquad u_{0j} \sim N(0, \sigma^2_{u0}) \quad \text{var}(e_{0i}) = 1$$

where $\log(\lambda_{ij})$ represents the log of the count of antisocial behaviors for individual *i* in neighborhood *j*, γ_{00} is the grand mean, the level-one residual variance e_{0i} is constrained to be 1, and u_{oj} is the variance of neighborhoods.

Models 1 through 3 in Table 2.3 and model 1 in Table 2.4 include main effects and all control variables. They are used to test for social disorganization theory and genetic effect. The general equation is

$$\log(\lambda_{ij}) = \gamma_{00} + \gamma_{10} X_{ij} + \gamma_{01} W_j + \gamma_{mo} C_{ij} + u_{oj} \qquad u_{0j} \sim N(0, \sigma^2_{u0}) \quad \text{var}(e_{0i}) = 1$$

where γ_{10} represents the fixed effect of individual-level predictors, X_{ij} (*5-HTTLPR*), γ_{01} is the fixed effect of neighborhood-level predictors, W_j (neighborhood measures), γ_{m0} is the fixed effect of individual-level control variables (C_{ij}), and other symbols are the same as in the equations above. Finally, models 2 through 3 in Table 2.4 include cross-level interaction effects that test for G×E effects. The general equation is

$$\log(\lambda_{ij}) = \gamma_{00} + \gamma_{10} X_{ij} + \gamma_{01} W_j + \gamma_{11} X_{ij} W_j + \gamma_{mo} C_{ij} + u_{oj} \qquad u_{0j} \sim N(0, \sigma^2_{u0}) \quad \text{var}(e_{0i}) = 1$$

where γ_{11} represents the effect of cross-level interaction between X_{ij} (*5-HTTLPR*) and W_j (neighborhood measures), and other symbols are defined above.

Neighborhood variables were standardized before the interaction terms are calculated. The benefits of utilizing standardized scores in the interaction model include making coefficients

easier to interpret, reducing multicollinearity, and making the simple slope easier to test (Dawson & Richter, 2006). In addition, the variance inflation factors (VIF) and the tolerance statistics are used to detect whether multicolinearity exists among variables. To account for neighborhood measures that could provide plausible rival explanations and to avoid individual-level propensities, all analyses controlled for individual socioeconomic variables, residence history and age. When interactions effects were present, post-hoc analyses of significant interaction terms were conducted using the simple slope test (Aiken & West, 1991; Bauer & Curran, 2005). Furthermore, I used the confidence interval of the cross-over point of the simple regression lines to distinguish differential susceptibility from diathesis-stress (Widaman et al., 2012). The cross-over point is calculated by the following equation:

$$C = -\frac{\gamma_{10}}{\gamma_{11}}$$

where C is the cross-over point of the simple regression lines, and γ_{10}, γ_{11} were defined above. To make statistical inference on the point estimates of C, the confidence interval for the estimates of C is required. According to Widaman et al. (2012), the standard error of C is estimated by the re-parameterized equation using the non-linear regression and is used to construct the confidence intervals of C. Because multilevel Poisson models are used in the current study, the re-parameterized equation is calculated by generalized nonlinear mixed models (PROC NLMIXED in SAS, see appendix A) (SAS Institute, Cary, NC). For the study, if the 95% confidence interval of the cross-over point is within the range of the neighborhood measure, the differential susceptibility perspective is supported.

RESULTS FOR STUDY 1

Initial Findings

The American Psychiatric Association (DSM-IV; American Psychiatric Association 1994, pp. 649-650) specifies seven symptoms of ASPD: failure to conform to social norms, deceitfulness, impulsivity, aggressiveness, reckless, irresponsibility, and a lack of remorse. Frequency analysis indicated that 34.8% of respondents did not have any symptoms of antisocial behavior, 37.3% had one or two symptoms, and 27.9% had three or more symptoms. As can be seen in Table 2.1, the most frequently presented symptoms involved irresponsibility (43.1%), lack of remorse for the mistreatment of others (34.8%), irritability and aggressiveness (32.7%), and reckless disregard for safety of self or others (31.7%). Deceitfulness (5.5%) was a low frequency symptom. The mean symptom count for antisocial behavior was 1.68 (*SD* = 1.67). Moreover, respondents had lived in their neighborhoods an average of over twenty years.

Table 2.1.
Descriptive Statistics for Antisocial Behavior

	1= Presence		0 = Absent	
Symptoms	*Freq.*	%	*Freq.*	%
1. Failure to conform to social norms	41	10.3	356	89.7
2. Deceitfulness	22	5.5	375	94.5
3. Impulsivity	40	10.1	357	89.9
4. Irritability and aggressiveness	130	32.7	267	67.3
5. Reckless	126	31.7	271	68.3
6. Consistent irresponsibility	171	43.1	226	56.9
7. Lack of remorse	138	34.8	259	65.2

Table 2.2.
Correlation Matrix for the Study Variables of Study 1.

	1	2	3	4	5	6	7	8	9	10
1. Antisocial behavior	——									
2. Concentrated disadvantage	.145**	——								
3. Racial composition	.060	-.411**	——							
4. Neighborhood social ties	-.209**	-.390**	.017	——						
5. *5-HTTLPR* (1 = ss/sl)	.082	-.051	.034	-.031	——					
6. High school diploma	-.049	-.160**	.122*	.161**	-.023	——				
7. Single family status	.020	.119*	-.034	-.079	-.064	-.038	——			
8. Age	-.203**	-.093†	-.012	.061	-.050	-.079	.053	——		
9. Family poverty	.089†	.184**	-.044	-.089†	-.039	-.232**	.167**	-.107*	——	
10. Residence history	.082	-.002	.041	-.139**	.006	-.099*	.012	-.140**	.080	——

**$p \leq .01$; *$p \leq .05$; †$p < .10$ (two-tailed tests); $N = 397$

The zero order correlations among the study variables are presented in Table 2.2. As expected, living in a neighborhood with concentrated disadvantage or weak social ties is associated with increased risk of antisocial behavior. In addition, structurally disadvantaged neighborhoods have weak neighborhood social ties. On the other hand, there is no significant correlation between *5-HTTLPR* and antisocial behavior. Consistent with previous molecular genetic studies (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Caspi et al., 2003; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012), the s-allele *5-HTTLPR* shows no association with antisocial behavior.

Table 2.3.
Poisson Multilevel Regression Models Examining Neighborhood Measures as Predictors of Adult Antisocial Behavior

	Model 1		**Model 2**		**Model 3**	
Fixed effect	*Coeff.* (SE)	Odds Ratio	*Coeff.* (SE)	Odds Ratio	*Coeff.* (SE)	Odds Ratio
Intercept	.879**	2.409	.853**	2.347	.827**	2.286
	(.136)		(.135)		(.134)	
Between-Neighborhood						
Concentrated Disadvantage			.130*	1.139	.078	1.081
			(.063)		(.067)	
Racial Composition			.113	1.120	.095	1.100
			(.070)		(.068)	
Neighborhood Social Ties					-.141*	.868
					(.068)	
Between-Person						
High school diploma	-.123	.884	-.113	.893	-.086	.918
	(.093)		(.092)		(.093)	
Single Family Status	-.016	.984	-.020	.980	-.019	.981
	(.085)		(.085)		(.085)	
Age	-.029**	.971	-.027**	.973	-.027**	.973
	(.006)		(.006)		(.006)	
Family Poverty	.078	1.081	.065	1.067	.073	1.076
	(.089)		(.089)		(.089)	
Residence history	.042	1.043	.047	1.048	.035	1.036
	(.089)		(.088)		(.088)	
Random effect (Variance Component)						
τ_{00}	.124		.097		.083	
Deviance	690.5		685.7		681.6	

Note: Unstandardized coefficient and odds ratio shown with robust standard errors in parentheses; family poverty and age are group centered; the measures of concentrated disadvantage and neighborhood social ties are standardized by z-transformation (mean = 0; *SD* =1); $N_{(persons)}$ = 397 and $N_{(neighborhoods)}$ = 67
**$p \leq .01$; *$p \leq .05$, †$p < .10$ (two-tailed tests)

Prior to beginning the multilevel modeling, I tested for gene-environment correlation (rGE) as it is possible that genotype may influence selection into different types of neighborhoods. The presence of rGE may further confound the examination of G×E effects (Caspi & Moffitt, 2006; Freese & Shostak, 2009). Table 2.2 shows that there were no significant zero-order correlations between the neighborhood measures (either concentrated disadvantage or neighborhood social ties) and *5-HTTLPR*. Furthermore, in analyses not shown, *5-HTTLPR* genotype was not related to either concentrated disadvantage or neighborhood social ties. Thus, there is no evidence of an active rGE effect whereby people seek out or evoke environments that are compatible with their genetic predispositions, indicating an absence of rGE effects in the current study.

Tables 2.3 and 2.4 show the results of using multilevel Poisson modeling to examine the effects of concentrated disadvantage, neighborhood social ties, and 5HTTLPR on women's antisocial behavior, controlling for education, family structure, age, family poverty, and residential history. I first checked for potential multicollinearity among variables. VIF scores ranged between 1.010 for cumulative plasticity alleles and 1.527 for concentrated disadvantage, and all measures of tolerance were above .60, indicating no evidence of multicollinearity (VIF <10 and tolerance > .20, Cohen, Cohen, West, & Aiken, 2003) among the study variables. The results of an unconditional model indicate that the neighborhood random effect is significant. Approximately 11% (ICC = .111) of the total variance occurs across neighborhoods. This result is consistent with previous research reporting that there is substantial variation across neighborhoods in levels of antisocial behavior.

Table 2.4.
Poisson Multilevel Regression Models Examining Neighborhood Measures and Genetic Diversity as Predictors of Adult Antisocial Behavior

	Model 1		Model 2		Model 3	
Fixed effect	*Coeff.* (SE)	Odds Ratio	*Coeff.* (SE)	Odds Ratio	*Coeff.* (SE)	Odds Ratio
Intercept	.736**	2.088	.790**	2.203	.760**	2.138
	(.143)		(.146)		(.145)	
Between-Neighborhood						
Concentrated Disadvantage	.084	1.088	.026	1.026	.066	1.068
	(.069)		(.080)		(.081)	
Racial Composition	.096	1.101	-.002	.998	.087	1.091
	(.068)		(.086)		(.068)	
Neighborhood Social Ties	-.134*	.875			.003	1.003
	(.068)				(.093)	
Between-Person						
5-HTTLPR (1 = ss/sl)	.160†	1.174	.129	1.138	.091	1.095
	(.082)		(.092)		(.091)	
High school diploma	-.080	.923	-.112	.894	-.068	.934
	(.093)		(.093)		(.093)	
Single Family Status	-.009	.991	-.009	.991	-.011	.989
	(.085)		(.085)		(.085)	
Age	-.026**	.974	-.028**	.972	-.028**	.972
	(.006)		(.006)		(.006)	
Family Poverty	.082	1.085	.087	1.091	.095	1.100
	(.089)		(.089)		(.089)	
Residence history	.036	1.037	.033	1.034	.039	1.040
	(.088)		(.089)		(.089)	
Cross-Level Interaction						
Concentrated Disadvantage			.203*	1.225	.030	1.030
× *5-HTTLPR*			(.090)		(.090)	
Neighborhood Social Ties					-.246*	.782
× *5-HTTLPR*					(.109)	
Random effect						
(Variance Component)						
τ_{00}	.084		.100		.083	
Deviance	677.8		674.6		670.2	

Note: Unstandardized coefficient and odds ratio shown with robust standard errors in parentheses; family poverty and age are group centered; the measures of concentrated disadvantage and neighborhood social ties are standardized by z-transformation (mean = 0; *SD* =1); $N_{(persons)}$ = 397 and $N_{(neighborhoods)}$ = 67
**$p \leq .01$; *$p \leq .05$, †$p < .10$ (two-tailed tests)

Concentrated Disadvantage, Social Ties, and Adult Antisocial Behavior

As shown in Table 2.3, the first model includes all individual-level control variables. The second model adds two neighborhood measurements: concentrated disadvantage and racial composition. A comparison of model 1 with model 2 indicates that the neighborhood variance is reduced by 21.77% when these two neighborhood measurements are included. The table shows

that an increase of one standard deviation in concentrated disadvantage is associated with an increase of 14 percent in the odds of adult antisocial behavior (odds ratio = 1.139, 95% CI [1.001, 1.291], $p = .041$). In contrast, racial composition is not significantly related to antisocial behavior.

Model 3 adds the variable neighborhood social ties. The difference in deviance between model 2 and model 3 is significant (Δ chi-square = 4.10, $df = 1$, $p = .428$), implying that the measure of neighborhood social ties improves the model fit. Consistent with the mediation argument, the effect of concentrated disadvantage on antisocial behavior is no longer significant when neighborhood social ties is included in the model. Comparing Model 2 with Model 3, neighborhood variation is reduced by 14.43% when neighborhood social ties is added to the model. Therefore, social ties explains a substantial variation in antisocial behavior. A standard deviation increase in neighborhood social ties is related to a 13 percent decrease in the odds of adult antisocial behavior (odds ratio = .868, 95% CI [.761, .993], $p = .039$). This pattern of results suggests that neighborhood social ties is a mediator of the effect of concentrated disadvantage on antisocial behavior. Thus, as expected, the results with a sample of adult women replicate prior research with children and adolescents.

The Effect of Genetic Variations on Adult Antisocial Behavior

Table 2.4 presents multilevel models including *5-HTTLPR*. Controlling for all individual-level demographic predictors and neighborhood measures, *5-HTTLPR* is not significantly related to adult antisocial behavior (odds ratio = 1.174, 95% CI [.999, 1.378], $p = .060$) at the .05 level. This finding is consistent with prior molecular studies indicating that so-called risk alleles

generally have little main effect on problem behavior (Caspi et al., 2003; Moffitt, Caspi, & Rutter, 2006; Simons et al., 2011).

The Effect of G×E on Antisocial Behavior

The next set of models examines the extent to which variation in *5-HTTLPR* gene moderates the impact of the neighborhood variables. Model 2 adds the interactions of *5-HTTLPR* with concentrated disadvantage. The interaction term for *5-HTTLPR* and concentrated disadvantage is statistically significant (odds ratio = 1.225, 95% CI [1.027, 1.461], $p = .025$).

To further examine the interaction of concentrated disadvantage with *5-HTTLPR*, I graphed the effect in Figure 2.2 for levels of concentrated disadvantage that range from -2 to +2 standard deviations from the mean. Using a simple slope test (Bauer & Curran, 2005), the graph shows that the association between concentrated disadvantage and adult antisocial behavior is significantly steeper for respondents who carry one or two copies of the s-allele (b = .229, 95% CI [.076, .038], $p = .003$) than for those who do not (b = .026, 95% CI [-.131, .183], $p = .746$).

First of all, based on a pick-a-point approach (Aiken & West, 1991), those with one or two copies of the S-allele show significantly more antisocial behavior than those with no copies when concentrated disadvantage is greater than 1.5 standard deviations above the mean (b = 1.156, p =.046). The slopes show a cross-over pattern consonant with the differential susceptibility perspective. However, in advantaged neighborhoods there is no significant difference in antisocial behavior between respondents who carry a copy of the S-allele and those who do not. As mentioned above, the reason is that the distribution of the sample across types of communities does not provide sufficient statistical power to provide a valid test of the significance of this difference.

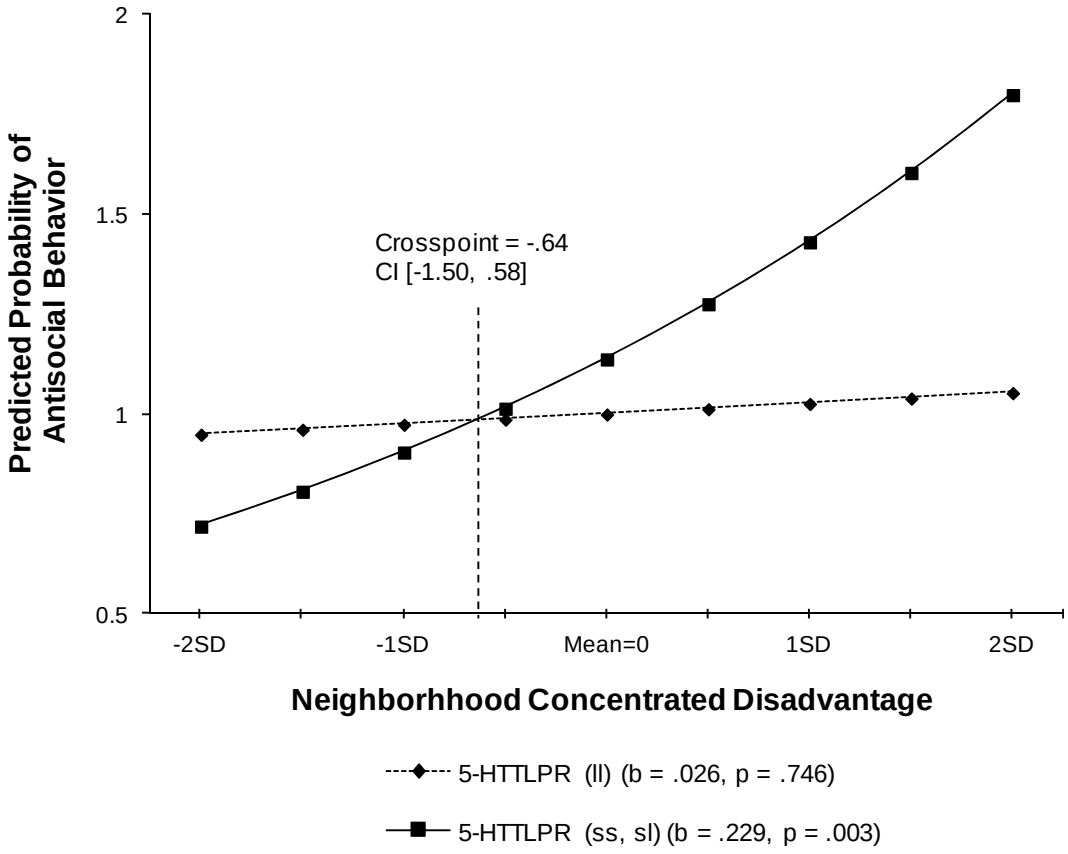

Figure 2.2.
The Effect of Neighborhood Concentrated Disadvantage on Antisocial Behavior by *5-HTTLPR*.

Turning to the new post-hoc test (Widaman et al., 2012), the differential susceptibility explanation for this G×E effect was tested using the confidence interval for the crossover point of the simple regression lines. If the confidence interval includes the range of observed values on the independent variable, a crossing pattern exists and the differential susceptibility is supported. As shown in Figure 2.2, the crossover point for the simple regression lines is -.64 [$C = -1 \times (.129/.203)$]. The 95% confidence interval of the crossover point ranges from -1.50 to .58. It covers the mean score of concentrated disadvantage (mean = 0) and a range of possible values of

concentrated disadvantage (-1.80 to 2.60). These results present the expected crossing pattern and provide strong support for the differential susceptibility perspective.

The last model in Table 2.4 incorporates the interaction term between neighborhood social ties and *5-HTTLPR*. The difference in deviance between model 2 and model 3 is significant (Δ chi-square = 4.4, $df = 1$, $p = .036$), indicating that the cross-level interaction of neighborhood social ties and *5-HTTLPR* improves the fit of the model. The interaction effect of neighborhood social ties and *5-HTTLPR* is significant (odds ratio = .782, 95% CI [.631, .967], $p = .023$). Importantly, inclusion of this interaction term reduces the interaction effect of concentrated disadvantage and *5-HTTLPR* to non-significance ($p > .05$). This suggests a pattern of mediation where the effect of the interaction of genetic plasticity × concentrated disadvantage is explained by the interaction of genetic plasticity × social ties.

Figure 2.3 shows the graph of the interaction of *5-HTTLPR* and social ties. Using the simple slope procedure, the slope for respondents with at least one S–allele is significantly different from zero (b = -.219, 95% CI [-.384, -.005], $p = .009$), whereas the slope for non-carriers is not (b = .003, 95% CI [-.179, .185], $p = .976$). In other word, the effect of neighborhood social ties on antisocial behavior is significantly greater for adults with at least one S-allele than for those who do not have this allele.

As Figure 2.3 shows, the crossover point of the simple regression lines is .37 [$C = -1 \times (.091/-.246)$]. I estimate the 95% confidence interval of this crossover point using the re-parameterized equation to be between -.48 and .93. The range includes the mean score and the possible range of observed values on the measure of neighborhood social ties (-.45 to 1.18). Thus, as was the case for the interaction of *5-HTTLPR* and concentrated disadvantage, the slopes

depicted in Figure 2.3 show a cross-over pattern consonant with the differential susceptibility perspective.

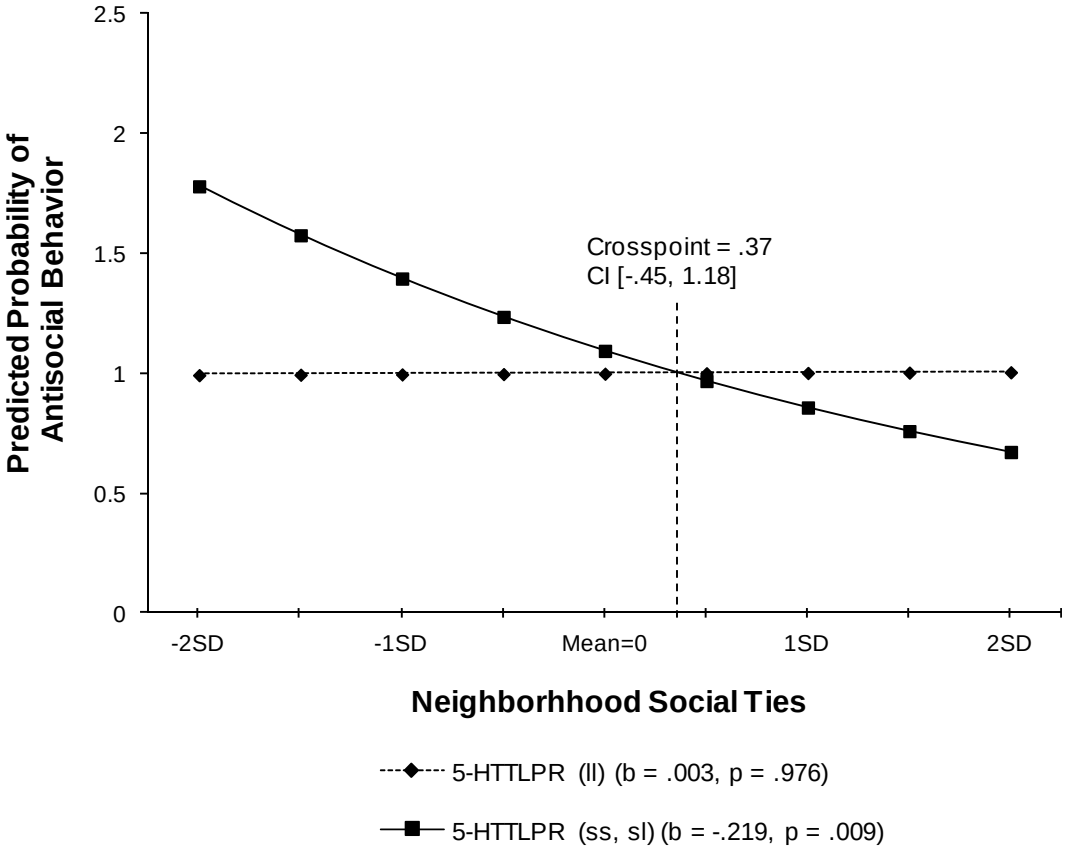

Figure 2.3.
The Effect of Neighborhood Social Ties on Antisocial Behavior by *5-HTTLPR*.

Supplementary analysis

Studies have indicated that population genetic admixture may confound G×E findings (Halder et al., 2009; Reiner et al., 2005). I employed the Structure program version 2.3.4 (Falush et al., 2007) with a panel of 24 ancestry informative markers (AIMs) to infer the number of ancestral populations and to estimate an ancestry proportion of each participant. The average proportion of African ancestry in the sample is 94.7%. Including the ancestry proportion as a

covariate into research models does not change the results. There is no evidence for genetics admixture as a potential confound in the present study.

DISCUSSION FOR STUDY 1

Prior research has provided rather strong support for the community disorganization perspective. These studies indicate that concentrated disadvantage increases the probability of antisocial behavior and it does so by disrupting social ties and informal social control (Leventhal & Brooks-Gunn, 2000; Sampson et al., 2002). However, most of this research has focused on children and adolescents and the few studies conducted on adult studies have only included males. As a result, some feminist scholars have asserted that there is virtually no evidence that community disorganization theory is applicable to females (Belknap, 2007; Chesney-Lind & Pasko, 2013; Kroneman et al., 2004; Zahn & Browne, 2009). The present study attempted to address this limitation in past research by testing the community disorganization model using a sample of African American women. Consonant with the findings from prior research, the results indicated that concentrated disadvantage is associated with increased involvement in antisocial behavior and that this effect is mediated, in large measure, by neighborhood social ties. This finding suggests that the theory is not simply a theory of adolescent or male deviant behavior. It is also explains the antisocial behavior of adult women.

A second limitation of past research on social disorganization theory is that it has paid little attention to personal differences in the way that individuals respond to neighborhood concentrated disadvantage and social ties (Leventhal & Brooks-Gunn, 2000). I attempted to address this issue by examining the extent to which variation in genes moderates the effect of community disorganization on antisocial behavior. Past research has reported that

polymorphisms in both *DRD4* and *5-HTTLPR* interact with other aspects of the environment, such as parenting and discrimination (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Belsky & Pleuss, 2009; Brody et al., 2011; Simons et al., 2011, 2012; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012), to influence the development of antisocial behavior. The present study extended this research by examining the extent to which an accumulation of these genetic variants also moderates the impact of community disorganization. The results indicated that this is the case. Although the *5-HTTLPR* gene had no direct effect on antisocial behavior, they moderated the association of both concentrated disadvantage and social ties with increased involvement in antisocial behavior. Further, the results indicated a pattern of spurious relationship in that the moderating effect of genetic variation on the relationship between concentrated disadvantage and antisocial behavior was explained by the interaction of genetic variation with social ties. These findings suggest that variation in genes involved in regulation of the serotonergic and dopaminergic neurotransmitter systems is an individual difference that accounts, at least in part, for dissimilarities in the way that people respond to neighborhood influences.

Further, several recent studies have reported evidence indicating that the serotonin transporter genes interact with the environment in the manner predicted by the differential susceptibility perspective (Bakermans-Kranenburg & Van Ijzendoorn, 2011; Belsky & Pleuss, 2009; Simons et al., 2011, 2012; Van IJzendoorn, Belsky, & Bakermans-Kranenburg, 2012). This perspective asserts that persons with plasticity alleles are more sensitivity to the effects of both favorable and adverse social environments. They are more likely than those without these alleles to be influenced by both the stressful and supportive conditions extant in their social environment. One of the goals of the present study was to test whether the interaction of *5-*

HTTLPR with variations in the neighborhood environment also conform to the pattern predicted by differential susceptibility.

Tests of the differential susceptibility perspective usually relay upon the Johnson-Neyman technique or the pick-a-point approach to evaluate the G×E effect. These procedures require data, however, representing the full range of values on the environmental variable of interest, from adverse to favorable, and enough variation in the measures to estimate the confidence region (Belsky, Pluess, & Widaman, 2013; Dick et al., 2011). The data could not meet these requirements as African American families tend to be overrepresented in disadvantaged neighborhoods (McNulty, 2001; Peterson & Krivo, 2010; Sampson, Morenoff, & Raudenbush, 2005). Fortunately, Widaman et al. (2012) recently developed the re-parameterized model as a method for testing the differential susceptibility perspective under such circumstances. Using their approach, the findings provide evidence for the cross-over effect predicted by the differential susceptibility model. Women with the focal alleles showed poorer adjustment than other genotypes when the neighborhood environment was adverse but better adjustment than other genotypes when the neighborhood environment was favorable.

From a theoretical standpoint, these results replicate and extend the existing neighborhood literature. Consistent with past findings for adolescents and young men (Sampson, 2012), I found that neighborhood disadvantage increases the probability of antisocial behavior among African American women and that this relationship is explained, in large measure, by neighborhood social ties. It was also the case, however, that there was much heterogeneity in antisocial behavior among individuals residing in the same neighborhood. The results suggest that some individuals are genetically predisposed to be more sensitive to neighborhood conditions than others. Individuals with particular variants of the serotonin transporter gene

showed higher rates of antisocial behavior than other genotypes when the neighborhood environment was adverse but demonstrated less antisocial behavior than other genotypes when they resided in advantaged neighborhoods with strong social ties.

An advantage of the present study is that the data set included genetic data and both census and process measures of neighborhood characteristics. In addition, the sample consisted of families nested within neighborhoods which allowed us to perform multilevel analyses. Nevertheless, the study also suffered from limitations that need to be noted. First of all, given that the adults in the sample were selected because of their status as primary caregivers, virtually all of them were women. The lack of men in the sample prevented us from being able to assess the extent to which the results might differ by gender. There is certainly a need for neighborhood studies that focus upon women given their exclusion in prior research. However, it is also important that the results be replicated with samples that include both men and women so that gender differences can be investigated.

The fact that all of the women in the sample were African Americans might be viewed as a strength as well as a limitation. On the one hand, neighborhood studies of African-American women are important for theoretical and policy reasons given that they have been shown to have higher rates of antisocial behavior than women in other ethnic groups (Belknap, 2007) and are more apt to reside in extremely disadvantaged neighborhoods (McNulty, 2001; Peterson & Krivo, 2010). On the other hand, the results obtain in the present study clearly need to be replicated with women from other ethnic groups.

Finally, community research shows that people select themselves into neighborhoods based upon personal characteristics (Sampson et al., 2002; Sampson, 2012), and genetic studies (Caspi & Moffitt, 2006) have found rGE effects in which people select environments that are

compatible with their genetic predispositions. Unfortunately, selection bias is nearly impossible to completely rule out in non-experimental studies. To reduce neighborhood selection bias in the present study, individual demographic variables were included as controls in all of the models. Further, the analyses indicated that there were no associations between variations in *5-HTTLPR* and the neighborhood measures. Thus, while selection bias cannot be completely ruled out in the present study, it is unlikely that it exerted a significant effect upon the findings.

In summary, research on gene-neighborhood interactions provides an alternative framework for understanding the relationships between neighborhood influences and human behaviors. Findings from this study extend neighborhood studies to women and support the conclusion that particular genetic polymorphisms amplify sensitivity to neighborhood context. Specifically, this study suggests that the s-allele of *5-HTTLPR* enhance susceptibility to concentrated disadvantage and the absence of neighborhood social ties.

CHAPTER 3

STUDY 2: NEIGHBORHOOD DISORDER AND HEALTH

Over the past two decades, there has been increasing interest in how social conditions influence health and disease (Link & Phelan, 1995; Phelan, Link, & Tehranifar, 2010). For example, recent research indicates that lower socioeconomic status in childhood is associated with a higher prevalence of chronic illnesses, including diabetes, hypertension, and cardiovascular disease (Miller, Chen, & Parker, 2011). Exposure to childhood trauma may increase the risk of chronic diseases and the dysregulation of the immune system (Fagundes, Glaser, & Kiecolt-Glaser, 2013). Youth who perceive multiple forms of discrimination have worse mental and physical health (Grollman, 2012).

In particular, the neighborhoods in which youth live are fundamental contexts for understanding stress and health. Numerous studies have provided evidence suggesting that the neighborhood context often contains a variety of stressful stimuli and structural elements that can impact health outcomes because both social and economic stressors tend to cluster in disordered neighborhoods where unequal socioeconomic opportunity structures exist (Hill, Ross, & Angel, 2005; Pearlin, 1989; Aneshenel, 1992; Ross & Mirowsky, 2001). Based on the stress process model, the literature has supported that neighborhood disorder is related to self-reported health status and such relationships are mediated by psychological processes (Aneshensel & Sucoff, 1996; Bowling & Stafford, 2007; Browning & Cagney, 2003; Cohen et al., 2003; Diez-Roux et al., 2001; Franzini et al., 2005; Idler, Russell, & Davis, 2000; Robert, 1998; Ross & Mirowsky,

2001; Schootman et al., 2006; Turner, 2010). While studies have indicated that physiological mechanisms provide a way of measuring bodily reactions that are associated with how individuals respond to stress (Miller, Chen, & Parker, 2011), little is known about the stress process mechanism through which neighborhood contexts impact health.

During the past decade, an increasing number of studies have found that social stressors are associated with elevations in circulating markers of inflammation. These studies have proposed that inflammation markers as physiological stress-response mechanisms are linked to deregulated biological systems that predict the likelihood of certain cancers, cardiovascular diseases, atherosclerosis, diabetes, and chronic illnesses (Black, 2006; Chen et al., 2006; Danesh et al., 2000; Gallo et al., 2012; Miller, Chen, & Parker, 2011). In other words, while living in disordered neighborhoods is a common source of chronic stress (Ross & Mirowaky, 2001; Schulz et al., 2000), it is unclear how neighborhood contexts influence self-reported health status through inflammatory responses. One of the goals of this study is to extend prior research of neighborhood disorder and health by examining objective inflammation biomarkers as well as self-reported health status. Using these biomarkers, I attempt to better understand the physiological pathways linking neighborhood disorder and self-reported health.

On the other hand, a handful of existing studies have reported that genetic effects on health are sensitive to environmental contexts as well as individual genetic variants, which may influence physical health status (Bamshad, 2005; Beach et al., 2012; South & Krueger, in press). Although some studies find the link between neighborhood disorder and physical health, few studies have been able to examine whether such relationships are moderated by genetic makeup (Miller, Chen, & Parker, 2011). In particular, no study has focused on the manner in which both neighborhood disorder and genetic variation combine to influence inflammation responses.

Therefore, how genes moderate the association between neighborhood disorder and inflammation markers remains unclear.

A number of studies have indicated that social support can serve as a buffer for the impact of adverse social conditions on health, and these studies have documented that marital status is a central source of social and emotional support that relates to health and buffers the effects of stress (Cohen, 1988; Hughes & Waite, 2009; Lin & Ensel, 1989; Schwerdtfeger & Friedrich-Mai, 2009; Taylor, 2011). For instance, studies have indicated that marriage may buffer the negative effect of stress on physical health and that married people have better health than their unmarried counterparts (Bookwala & Fekete, 2008; Kaplan & Kronick, 2006; Thoits, 2010). However, few studies have examined the extent to which neighborhood context on systemic inflammation are buffered by marital status. More importantly, no studies have examined whether the multiplicative effect of genetic variants by neighborhood context on systemic inflammation vary by marital status. Therefore, it is important to extend this line of research to include both genotype and marital status, and to examine the mechanisms that trigger inflammatory responses across neighborhoods.

While studies provide evidence suggesting that African Americans are more vulnerable to physiological deterioration (Geronimus et al., 2006; Warner & Hayward, 2006; Thoits, 2010) and are more likely than other ethnic groups to live in neighborhoods with high crime and poverty rates (Peterson & Krivo, 2010), there is no work that links neighborhood disorder as an objective indicator of inflammation burden among African Americans. In particular, traditional neighborhood studies assume that neighborhood effects are the same for women and men and that findings based on male samples can be used to generalize to women (Belknap, 2007; Chesney-Lind, 1989; Jacob, 2006; Zahn & Browne, 2009). It is important to examine whether

neighborhood factors influence female health. To address these neglected issues, this study incorporates systematic social observation, a self-reported survey, and measures of inflammation biomarkers to examine neighborhood disorder and health among a sample of adult African American women.

NEIGHBORHOOD DISORDER AND HEALTH

Studies concerning neighborhood context have suggested that individual characteristics alone may not explain health inequalities (Browning & Cagney, 2003). Individuals are embedded in neighborhoods that shape everyday life. Most studies of social disorganization theory have found that people who live in disadvantaged neighborhoods have higher proportions of chronic illnesses and mortality than those who live in more advantaged neighborhoods (Diez-Roux et al., 2001; Schootman et al., 2006). In addition to economic-based neighborhood structures, Wilson and Kelling (1982) indicated that disordered neighborhoods display more visual signs of physical disorder including vacant buildings and buildings marked by graffiti and litter. They proposed that exposure to visible signs of neighborhood disorder lead to high crime rates because of the psychological suggestion of disrespect and hopelessness. Recent studies have used neighborhood disorder to explain physical or mental health problems (Browning et al., 2013; Hill, Ross, & Angel, 2005; Ross & Mirowsky, 2001). They indicate that exposure to disordered neighborhoods increases the risk of asthma (Rosenbaum, 2008), morbidity (Diez-Roux, 2001), gonorrhea (Cohen et al., 2000), and cardiovascular disease (Nordstrom et al., 2004). More importantly, Ross and Mirowsky (2001) revealed that the effect of concentrated disadvantage on self-reported health is mediated by disorder in the neighborhood. These findings suggest that neighborhood

disorder is a fundamental role in health inequalities and can explain why neighborhood disadvantage influences physical health.

While individuals living in disordered neighborhoods are more vulnerable to health problems and chronic illness, how such neighborhoods effect physical health remains unclear. In the past two decades, scholars have proposed that the stress process model provides a framework for understanding neighborhood effects (Foster & Brooks-Gunn, 2013). According to the stress process model, health inequalities are caused by differential exposure to social stress environments that impair immune system's ability, damage heart tissue, and increase the risk of chronic diseases because prolonged exposure to chronic stress can produce a physiological and psychological arousal reaction that wear down the body's ability to resist disease (Pearlin et al., 2005; Pollitt et al., 2008; Turner, 2010). For example, Hill, Ross, and Angel (2005) revealed that women living in disadvantaged neighborhoods experienced more physiological distress and reported worse physical health status. Evan et al. (2007) found that poverty and low socioeconomic status lead to physiological dysregulation, which in turn increase risk for cardiovascular disease. Wendy and colleagues (2003) revealed that slow cardiovascular recovery among adults is associated with environmental stressors and conditions. Furthermore, Thoits (2010) indicated that since poor health is likely related to persistent and repeated stressful social contexts, health inequality is accounted for by the uneven distribution of stress across individuals in the population.

In particular, a number of studies have suggested that chronic stress tends to cluster in disordered neighborhoods (Aneshenel, 1992; Hill, Ross, & Angel, 2005; Latkin & Curry, 2003; Pearlin 1989; Pearlin et al., 2005). This is because residents living in those neighborhoods face multiple social and economic challenges. Evidence from neighborhood studies indicates that

disadvantaged neighborhoods offer few opportunities for economic and educational success, which result in increased economic stress (Sampson, 2012). Neighborhoods with signs of physical disorder (e.g. graffiti, litter, and abandoned commercial buildings) are often a signal indicating commercial decline (Browning et al., 2006). In addition, physical disorder in neighborhoods have been consistently shown to lead to increased fear of crime, victimization, social insecurity, lack of control, and structural discrimination (Browning, Cagney, & Iveniuk, 2012; Ross & Mirowsky, 2009; Sampson & Raudenbush, 2004; Wilson & Kelling, 1982). Therefore, disordered neighborhoods have been found to be associated with ambient threat, depression, social strain, psychological distress, and a prevalence of stressful life events for the people living in these areas (Aneshensel & Sucoff, 1996; Hill, Ross, & Angel, 2005; Latkin & Curry, 2003; Natsuaki et al., 2007; Ross & Mirowsky, 2009; Weich, 2002). Consequently, residents living in disordered neighborhoods are likely to experience hyper-activation of adrenaline responses, which contribute to feelings of tension, distrust, and social isolation, which in turn can threaten physical well-being (Broyles, 2012; Hill, Ross, & Angel, 2005; Ross & Mirowsky, 2001).

In summary, exposure to stress is unevenly distributed across neighborhoods, and, in particular, chronic stressors are clustered in disordered neighborhoods because of the opportunity structures present therein. Classical neighborhood studies and the stress process perspective argue that neighborhood disorder plays a fundamental role in health inequalities because individuals living in these environments experience an erosion of their physiological stress-regulatory functions. In particular, studies have shown that African Americans are more likely than other racial groups to reside in extremely disorderly or disadvantaged neighborhoods (McNulty, 2001; Peterson & Krivo 2010), and have higher levels of chronic and heart disease

morbidity and mortality than other racial groups (Alder & Rehkopf, 2008). In addition, while neighborhoods consist of individuals of diverse ages, most studies employ child and adolescent samples (Aneshensel & Sucoff, 1996; Boardman, 2004; Glass & Balfour, 2003; Wickrama, Noh, & Bryant, 2005), relatively neglecting adult health from their analysis.

Finally, traditional neighborhood scholars have tended to operate as if results pertinent to men can be generalized to women as well. This approach has been criticized by many feminist scholars (e.g. Brooks-Gunn et al., 1993; Chesney-Lind & Bloom, 1997; Fagan & Wright, 2012; Zahn & Browne, 2009). These feminist scholars have indicated that the conceptualization of gender is embedded in social systems and women and men have unique life experiences in their neighborhoods (Cobbina, Miller, & Brunson, 2008; Miller & White, 2006). They have further suggested that females are ignored in neighborhood studies (Chesney-Lind & Pasko, 2013; Zahn & Browne, 2009) even though the impact of neighborhood characteristics on health is more pronounced for women than for men (Stafford et al., 2005). Using a sample of adult African American women, the first goal of the present study is to replicate findings of previous studies and proposes that the effect of neighborhood disorder is associated with physical health.

NEIGHBORHOOD DISORDER, INFLAMMATORY BURDEN, AND HEALTH

Although there is much research on the effect of neighborhood context on self-reported distress response and health status (Browning & Cagney, 2003; Franzini et al., 2005; Lee & Cubbin, 2003; Ross & Mirowsky, 2001; Schootma et al., 2006; Wen, Browning, & Cagney, 2003), it is surprising that very few studies have examined biomarkers from blood samples as objective health indicators. This is particularly the case for research that respondents are more likely to misperceive their own health status (Vandelanotte et al., 2011). Moreover, all study

measures are derived from self-report data, which are vulnerable to the inflation of the relationships between variables by "common method variance" biases (Podsakoff et al., 2003). In address this issue, this study focuses not only on subjective self-reported health but also on objective biomarkers.

Recently, molecular biologists have indicated that a number of inflammatory biomarkers are a sign of physiological distress (Danesh et al., 2000). There is growing evidence that chronic exposure to negative environments produce dysregulated physiological stress responses as well as elevated markers of inflammation and these biomarkers have been associated with increased likelihood of certain cancers, diabetes, cardiovascular diseases, hypertension, periodontal disease, rheumatoid arthritis, and general wear and tear on the body's systems (Black, 2006; Gouin, Hantsoo, & Kiecolt-Glaser, 2011). More importantly, several studies have reported that repeated exposure to stressors results in dysregulation of the inflammatory response (Holmes & Marcelli, 2012; Pollitt et al., 2007). For instance, studies have indicated that children growing up in low socioeconomic families display elevated levels of inflammation (Chen et al., 2006; Miller et al., 2009) because they are more likely to experience economic hardships, education failure, and family instabilities. Similarly, Gouin and colleagues (2012) found that multiple stressors led to elevations in circulating inflammatory burden in adults. Given these results, inflammation markers, a physiological response to a variety of harmful stimuli, can explain the relationship between chronic stress and health problems.

Drawing on the growing stress, inflammation, and health literature (Browning, Cagney, & Iveniuk 2012; Gouin et al., 2011; 2012), researchers have determined that there are two main inflammatory markers: C-reactive protein (CRP) and soluble interleukin-6 receptors (sIL6R). CRP is a biomarker of vascular and systemic inflammation that is an acute phase serum protein

and is produced by the liver. There is evidence that CRP is a prototypic marker of inflammation and is an important risk factor for diabetes, hypertension, atherosclerosis and coronary heart disease (Danesh et al., 2000). In addition, several studies link neighborhood characteristics and CRP. Using a sample of 1410 adults living in Dallas, Browning and colleagues (2012) found that men living in neighborhoods with higher burglary rates exhibited elevated CRP levels. Broyles et al. (2012) found that children living in disadvantaged or unsafe neighborhoods were more likely to have elevated CRP levels than those living in advantaged or safe neighborhoods. Gallo et al. (2012) found that neighborhood disadvantage is positively related to CRP in Mexican American women. Using samples from unauthorized Brazilian migrants, Holmes and Marcelli (2012) also found that neighborhood disorder was positively associated with greater levels of CRP.

The soluble receptor of IL-6 (sIL6R) is assessed through blood plasma and is a marker of chronic cytokine activity (Morley & Baumgartner, 2004). Previous studies have indicated that the marker plays a role in the regulation of IL-6 and is associated with diabetes, chronic illness, certain cancers, and neurological disorders (Gouin, Hantsoo, & Kiecolt-Glaser, 2011; Hurst et al., 2001; Jones et al., 2001; Jones & Rose-John, 2002; Shariat et al., 2001). Similar to CRP, there is also evidence that greater chronic stress exposure (Friedman et al., 2007; Kallen, 2002) or living in disordered neighborhoods (Nazmi et al., 2010) is associated with elevated sIL-6R levels after controlling for individual characteristics. While several studies have focused on the association between each inflammatory marker and chronic stress, others have reported that the inflammatory burden, a combination of inflammatory markers, has substantially more variation, predictive ability, and sensitivity to detect the effect of interest (Anuurad et al., 2011; Ikonomidis et al., 2012; Pollitt et al., 2007; Slopen et al., 2010).

Although a growing number of studies have suggested that neighborhood physical and social disorder hamper physical health and are linked to an elevated circulating systemic inflammation burden, the results remain inconclusive. Some studies found that the effects of neighborhood context on health outcomes were no longer significant after controlling for individual SES (Bowling & Stafford, 2007; Gallo et al., 2012; Petersen et al., 2008). This suggests that neighborhoods may not matter for physical health. Any significant neighborhood effects may instead be a product of self-selection processes through individual SES. In contrast, mounting evidence indicates that neighborhood factors influence physical health and inflammatory burden, even after controlling for individual socioeconomic status (Aneshensel & Sucoff, 1996; Cohen et al., 2003; Diez-Roux et al., 2001; Franzini et al., 2005; Holmes & Marcelli, 2012; Lee & Cubbin, 2003; Morenoff, 2003; Robert, 1998, 1999; Ross & Mirowsky, 2001). Therefore, these studies suggest that neighborhood effects influence health outcomes beyond the effect of individual characteristics. Given this mixed evidence, it remains unclear as to whether living in disordered neighborhoods has a unique effect on health outcomes. In the present study, I assess if neighborhood context matters for physical health by assessing the influence of disordered neighborhood contexts on self-reported health and inflammatory burden while controlling for the effects of individual characteristics.

Additionally, the stress process model assumes that the association between environmental contexts and physical health is mediated by distress responses (Pearlin, 1989). Previous studies have shown a psychological stress-response mechanism (Diez-Roux & Mair, 2010; Hill, Ross, & Angel, 2005; Ross & Mirowsky, 2001, 2009). For instance, Hill, Ross, and Angel (2005) found that living in disordered neighborhoods, which are characterized by abandoned buildings, graffiti, vandalism, and high crime rates, are associated with more mental

distress, which in turn increases the likelihood of poor physical health. In contrast to the psychological stress-response, a stress response in the body is unseen and cannot be collected by traditional survey methodology. For this reason, very few studies have focused on physiological stress-response mechanisms. It seems possible that elevated inflammatory burden is an underlying physiological mechanism that bridges the gap between neighborhood stress and physical health, because the inflammatory burden is a sign of immune dysfunction and change in major physiological systems (Broyles et al., 2012; Brenner et al., 2013). To fill the gap in the literature on the stress process model, this study hypothesizes that the effect of neighborhood disorder on self-reported health is mediated by inflammatory burden.

GENE-NEIGHBORHOOD INTERACTION

According to the classical neighborhood assumption, people from the same neighborhood are more similar to each other than to those from different neighborhoods (Foster & Brooks-Gunn, 2013). Interestingly, studies have found that many people living in disadvantaged neighborhoods do not become unhealthy, while others living in the same neighborhoods do (Ross & Mirowsky, 2001; Schootman et al., 2006). This leaves an essential question unanswered: Why do some people living in disordered neighborhoods remain healthy while others do not? Understanding which individual characteristics influence the relationship between neighborhood contexts and physical health is fundamental to the advancement of neighborhood research.

During the last decade, investigators have turned their attention to the interaction between genes and the environment (Barnes & Jacobs, 2013; Beaver et al., 2012; Belsky & Beaver, 2011; Caspi et al., 2010; Simons & Lei, 2013), and mounting evidence has suggested that genetic

variation often interacts with the environmental context to influence physical health and inflammatory burden (Beach et al., 2012; Brody et al., 2013; Caspi et al., 2010; Hartley et al., 2012; Khoury, Burke, & Thomson, 2000; Romanowicz et al., 2012). However, no research has examined gene by neighborhood interactions on inflammatory burden and physical health, even though neighborhood effects on human behavior and health problems should be moderated by individual characteristics and/or experiences (Bush, Lengua, & Colder, 2010; Simons et al., 2005), and genetic differences are potential source of inflammatory response (Miller, Chen, & Parker, 2011; Taylor, Way, & Seeman, 2011).

To address these issues, this study employs a single nucleotide polymorphism (SNP) *rs2228145* (also known as rs8192284) in the IL-6 receptor gene. This gene located at the proteolytic cleavage site results in a substitution of aspartic acid to alanine (Abeywardena et al., 2009; Smith et al., 2012). The minor C allele of *rs2228145* is associated with an elevated serum levels of sIL6R and CRP (Curocichin et al., 2011; Galicia et al., 2004; Hingorani & Casas, 2012; Qi, Rifai, & Hu, 2007; Ridker et al., 2008; Sarwar et al., 2012).

Within health-related studies (e. g. Manuck & McCaffery, 2010; Miller, Chen, & Parker, 2011; Taylor, Way, & Seeman, 2011), the diathesis-stress perspective has been proposed to explain how genetic predispositions to health outcomes are expressed by a variety of stressors. This perspective claims that some individuals are more vulnerable than others when exposed to adverse social environments. Graphing this type of interaction produces a fan-shaped pattern where the effect of adverse social environments on health problems becomes greater as individuals with a copy of a particular risk allele (Caspi et al., 2010; Simons & Lei, 2013). Based on previous theory and research, the present study hypothesizes that people carrying the minor C

allele of *rs2228145* will experience an amplification of the probability that exposure to disordered neighborhoods will lead to an elevated systemic inflammatory burden.

THE STRESS-BUFFERING HYPOTHESIS

In addition to gene-environment interaction influences, one possible reason why not all individuals living in disordered neighborhoods experience elevated levels of circulating inflammatory burden or feel unhealthy is that some individuals may have access to social support. These support networks can act as anti-inflammatory mechanisms. In fact, social scientists have long been interested in the extent to which social support affects health (Phelan, Link, & Tehranifar, 2010; Turner & Marino, 1994), and have proposed the stress-buffering hypothesis which argues that social support buffers the relationship between stress and health (Cohen, 1988; Lin & Ensel, 1989). Individuals with high levels of social support are able to protect themselves from the negative effects of stress, which in turn protects their physical health. Recently, neighborhood research has demonstrated that social support buffers the effects of neighborhood disorder or disadvantage on health and well-being (Diez-Roux & Mair, 2010; Foster & Brooks-Gunn, 2013).

Of the relationships considered in the stress buffering literature, marriage has been determined to be one of the most important sources of social support. Married people are more likely than unmarried people to receive and to perceive instrumental emotional support from their partners (Bookwala & Fekete, 2008; Kiecolt-Glaser, & Newton, 2001; Thoits, 2010; Taylor, 2011). For instance, there is evidence that married people tend to be in better physical health because those people are likely to have close family ties that provide a source of emotional support, material sources, and social connectedness (Kiecolt-Glaser, & Newton, 2001;

Soons & Liefbroer, 2008; Turner & Roszell, 1994; Waldron, Hughes, & Brooks, 1996). In contrast, unmarried individuals report more psychological distress and have more chronic illnesses (Hughes & Waite 2009; Wickrama et al., 1997).

The death rate is also higher for single people than for those who are married (Kaplan & Kronick, 2006). Therefore, marriage has beneficial effects on health and the functioning of biological stress-regulation systems. Most importantly, several studies have found that marital status could buffer negative effects of neighborhood disorder on health indicators (Bierman, 2009; Thoits, 2010). This suggests that the relationship between neighborhood disorder and health problems is stronger for never-married adults than for married adults.

Summarizing the literature on stress and health, marital status as a kind of social support may play an important role in moderating the effects of stress on health outcomes because marriage could protect individuals from the deleterious effects of stressors. If the neighborhood-gene interaction is correct, it is unclear whether both marital status and genetic makeup simultaneously moderate the effect of neighborhood disorder on inflammatory burden. Building on the stress-buffering hypothesis, the current study hypothesizes that the association between neighborhood disorder and inflammatory burden will be strongest among unmarried women carrying the risk allele of *rs2228145*.

RESEARCH DESIGN FOR STUDY 2

Sample

Data for this study are drawn from Wave 5 of the Family and Community Health Study (FACHS), a multi-site investigation of neighborhood and family effects on health and development. FACHS was designed to identify neighborhood and family processes that

contribute to the development of African American children. The sample strategy was intentionally designed to generate families representing a range of socioeconomic statuses and neighborhood settings. Each family included a child who was in 5th grade at the time of recruitment. At the first wave, the FACHS sample consisted of 889 African American children with their primary caregivers (PCs). Of the 889 PCs interviewed at Wave 1, 693 were interviewed again at Wave 5 (77% of the original sample). At Wave 1, the sample had an average family per capita income of $6956/year. Thirty six percent of the families were below the poverty line, and fifty one percent of the respondents lived in a single-parent family. As part of Wave 5 data collection, PCs were asked to provide blood samples. Of the 693 participants, 489 PCs (71%) agreed to biomarkers collection. Successful genotyping for inflammation markers was achieved for 460 PCs.

Current Study Participants

The current study involves both individual and neighborhood characteristics. The measures of neighborhood characteristics were created using the 2000 census Summary Tape File 3 (STF3A) which was geocoded with participant's residential addresses. Additional details regarding neighborhood data can be found in Simons and colleagues (2005). The current study is based upon the 342 respondents who classified by ancestry proportions (Reiner et al., 2005), were nested within 89 census tracts, agreed to provide a blood sample, and provided data on the systemic social observation of neighborhoods and all responded measurements at Wave 5. The resulting sample had a mean age of 47.13 years, SD = 8.01. Comparisons of those participants excluded from the present study but retained in the sample did not display any significant differences with regard to neighborhood and individual characteristics. To further assess attrition

bias, I used Heckman's (1979) two-step procedure to estimate sample selection bias. The results showed that the inverse Mills ratio was not significant, and including this ratio parameter in the models did not change the findings. There were no significant differences between those remaining in the panel and those dropping out with regard to a variety of measures. For the 89 census tracts based on the 2000 Census, 58% of the neighborhoods were urban areas, and 32% had a population more than half of which was African American. The average poverty rate in 2000 was 28 percent (*SD* = .14).

Procedures

The measures of neighborhood characteristics were created using the 2000 census data which was geocoded with participant's residential addresses at Wave 5. Respondents had lived in their neighborhoods an average of over twenty years. At Wave 5, computer assisted interviews were administered in the respondent's home and took on average about 2 hours to complete. The instruments were presented on laptop computers. Questions appeared in sequence on the screen, which both the researcher and participant could see. The researcher read each question aloud and the participant entered an anonymous response using a separate keypad.

When visiting the participant families at Wave 5, two interviewers also rated the face-block level neighborhoods on physical appearance and dilapidation. The face block is the unit of observation and is defined by the block segment on one side of a street (Sampsons & Raudenbush, 1999).

Participants were also asked to provide a blood sample at Wave 5. After blood was drawn into serum separator tubes by certified phlebotomists, it was frozen and shipped via courier to a laboratory at the University of Iowa. Serum levels of CRP and siL6r were determined using a

Duo Set Kit (DY1707; RandD Systems, Minneapolis, MN, USA) according to manufacturer's specifications.

Measures

An inflammatory burden was measured with two biomarkers of inflammation. C-reactive protein (CRP), a biomarker of vascular and systemic inflammation from a blood sample, was measured at Wave 5. A normal concentration of CRP in healthy human serum is usually lower than 10 mg/L (Holmes & Marcelli, 2012). Approximately 7.6% of women in the sample had CRP levels outside the normal range. Because CRP displayed a skewed distribution (skewness = 3.892, kurtosis = 18.496), it was transformed using log transformation to meet the assumption of linearity for ordinary least squares regression (skewness = -.96, kurtosis = 2.70 after the transformation). Soluble IL-6 receptor (sIL-6R) which is the cognate receptor for IL-6 was assayed at Wave 5. Similar to CRP, siL-6R displayed a skewed distribution (skewness = 1.79, kurtosis = 9.24). I applied a log transformation to normalize the index (skewness = -.021, kurtosis = 2.03 after the transformation). After that, I combined both of them in one index according to the canonical weights method (Singer et al., 2004). An inflammatory burden was calculated by summing the standardized log-transformed biomarker scores.

Self-reported health status was assessed with an item (Idler, Russell, & Davis, 2000) that asked, "In general, would you say your physical health is…" The response format for the item ranged from 0 (excellent) to 4 (poor). Higher scores indicated worse health status (M = 1.936, *SD* = .996). Roughly 32% of the sample considered themselves in poor or fair health.

Neighborhood disorder was assessed using the observers' ratings of the participants' neighborhoods because the census data did not provide detailed information about physical

incivilities in neighborhoods (Sampson, Morenoff, & Gannon-Rowley, 2002). Observers trained in the use of the systemic social observation survey (Sampsons & Raudenbush, 1999) rated three dimensions of physical signs in the neighborhoods, such as graffiti, vandalism, and abandoned buildings. When visiting the participant families, two trained observers also visited the participants' neighborhoods and observed the face block on which the block segmented on one side of a street. The intra-class correlation for these two observers was .62, which indicated good agreement according to the guidelines for evaluating the inter-rater reliability coefficients (Cicchetti, 1994). For these three dimensions, the rating format for each scale ranged from 1 (No) to 4 (Yes, a considerable amount). Scores were summed the three rating scales to form a measure of neighborhood disorder. Higher scores indicated higher degrees of neighborhood disorder.

Concentrated disadvantage was assessed with 2000 STF3A census tract data. Following previous studies (Sampson, Raudenbush, & Earls, 1997), the scale include items: average per-capita income, the percentage of unemployment, the percentage of residents below the poverty threshold, the percentage of residents without high school degree, the percentage of female-headed households, and the percentage of those receiving public assistance. To provide equal weight for each item, per capita income was reverse-coded, and I used factor scores obtained through principal-components analyses to form the scale. The coefficient alpha for the measure was .87.

Residential stability was measured using two items from the census STF3A(Sampson, Raudenbush, & Earls, 1997): 1) the percentage of residents living in the same house over five years; and 2) the percentage of owner-occupied homes. The two items were standardized and summed. The coefficient alpha for the measure was .82.

Social isolation. Consistent with previous studies (Wilson, 1987, 1999), when factories and companies migrated from urban to suburban, working- or lower- class African Americans living in disadvantaged neighborhoods had minimal access to economic resources and found it more difficult to obtain jobs and to relocate. Thus, African Americans living in disadvantage neighborhoods with high residential stability contributed to feelings of tension, distrust, and social isolation. As a result, social isolation was assessed by the interaction between concentrated disadvantage and residential stability in local neighborhoods (Browning & Cagney, 2003).

Neighborhood cohesion was measured at Wave 5 using a revised version of the Social Cohesion and Trust Scale (PHDCN; Sampson, Raudenbush, & Earls, 1997). The 15- item measure asks respondents to report the extent to which neighborhood residents agreed that neighbors get together to deal with local problems; their neighborhood is close knit; no one in the neighborhood cares much about what happens there; there are adults in the neighborhood children can look up to; people are willing to help their neighbors; people do not get along (reverse scored); people provide social support to each other (three items); people in this neighborhood can be trusted; people share the same values; people do favors for each other; people watch over each other's property when they are away; and the number of friends the respondent has in the neighborhood. The scores were standardized and then summed to form a measure of neighborhood cohesion. The coefficient alpha was .90.

Marital status was coded as a binary variable (1 = married; 0 = unmarried). Among the 342 women used in analysis, 29.5% of subjects reported that they were married.

Control variable. To avoid overestimated results, the analyses controlled for four demographic measures. Family socioeconomic status (SES) was a composite measure based on education and family income (M = .00, *SD* = .78). At Wave 5, the samples in the study had a

mean age of 46 years, with a range of 35-65 years. Given the wide age range observed in this sample, age is included in all analyses. Region was coded 1 for respondents living in the South (67.5 percent) and 0 for those living in other areas of the country. Mean length of residence for respondents was over ten years.

Genotyping. All participants were genotyped using TaqmanR MGB assays (Applied Biosystems, Foster City, CA) and Fluidigm Biomark Genetic Analysis System (Fluidigm, South San Francisco, CA). The SNP *rs2228145* (Asp358Ala) is located on exon 9 of the IL-6r gene on chromosome 1q21. Among the 342 respondents, 2.34% were homozygous for the C allele (CC) at *rs2228145*, 20.76% were heterozygous (AC), and 76.90% were homozygous for the A allele (AA). Using the Hardy-Weinberg equilibrium test, the observed distribution of *rs2228145* did not differ significantly (chi-square = 1.444, $df = 1$, $p = .230$) from that predicted on the basis of simple Mendelian inheritance. Studies have indicated that population genetic admixture may confound genetic findings (Halder et al., 2009). This study employed the Structure program version 2.3.4 (Falush et al., 2007) with a panel of 24 ancestry informative markers (AIMs) to infer the number of ancestral populations and to estimate an ancestry proportion of each participant. The average proportion of African ancestry in the sample is about 99%. There is no evidence for genetics admixture as a potential confound in the present study

Analytic Strategy

The study used multi-site samples to examine the current study models, but the multi-site samples were not independently selected. If samples were directly estimated by a general regression model, non-independent samples would over-estimate the results (Muthén & Satorra, 1995). To avoid this problem, this study used a complex sampling design model available in the

M*plus* 7.0 statistical software (TYPE=COMPLEX function, Muthén & Muthén, 2012). This model allowed the study to estimate actual standard errors for clustered data in complex mediation models (MacKinnon, 2007).

In addition, the measure of neighborhood disorder was standardized (mean of 0 and a standard deviation of 1) before interaction terms were calculated. Some advantages of using standardized scores in the interaction models include making coefficients easier to interpret, reducing multicollinearity, and making the simple slope easier to test (Dawson and Richter, 2006). Because an inflammatory burden displayed a strong positive skew, it was transformed using log transformation to meet the assumption of linearity for ordinary least squares regression.

To test the hypotheses, Poisson regression with a complex sampling design was used for self-reported health status because this measure is a count variable. Regarding self-reported health status, Model 1 in Table 3.2 was used to test for the main effect of neighborhood disorder. Model 2 in Table 3.2 included an inflammatory burden index to test the mediating model. Turing to an inflammatory burden, the studies included four regression models to test for the main effect of neighborhood disorder and the moderation effect of gene and neighborhood. Based upon the different coding schemes, the dominant genetic model used the first two models, and the additive model used in the last two models. In these four models in Table 3.3, Models 1 and 3 were used to test for main effects of neighborhood disorder, and Models 2 and 4 included the interaction terms necessary to test the moderating hypotheses. When the interaction effects were significant, post hoc analyses of interaction terms were conducted using the simple-slope test (Aiken & West, 1991) and the proposed proportion of interaction (PoI) index (Roisman et al., 2012). Furthermore, the mediated-moderation model examines the inflammatory burden as possible mediators of the two-way interaction effect of neighborhood disorder and genotype on health

status. The logic of the mediated-moderation model is similar to traditional mediated models except that it focuses only on the relationships among an interaction term, mediator, and outcome rather than among other independent variables (Preacher, Curran, & Bauer, 2006). Finally, to test the stress-buffering hypothesis, the study added a three-way interaction. When interaction effects were present, post hoc analyses of significant interaction terms were conducted (Dawson & Richter, 2006).

RESULTS FOR STUDY 2

Descriptive and Association Analysis

A substantial proportion of participants reported that they were in poor/fair health status. For example, four percent of respondents reported poor health, and 28% indicated fair health status. Using the objective biomarker measure, 14% of respondents in the sample scored above one standard deviation from the mean on the inflammatory burden.

Consistent with previous molecular genetic studies (Galicia et al., 2004; Hingorani & Casas, 2012; Qi, Rifai, & Hu, 2007; Ridker et al., 2008; Sarwar et al., 2012), Table 3.1 reveals that individuals carrying the A allele of *rs2228145* genotype demonstrate significantly higher levels of inflammatory burden (r = .242, p = .000006). Importantly, studies have indicated that the presence of gene-environment correlation (*r*GE) is likely to confound gene-environment interaction effects (Caspi & Moffitt, 2006; Shanahan & Hofer, 2005). As shown in Table 3.1, there is no significant relationship between neighborhood disorder and *rs2228145* genotype. This finding suggests the absence of an active *r*GE effect whereby people seek out neighborhoods that are compatible with their genetic predispositions. Furthermore, in analyses not shown, all associations of parent or child genotype with neighborhood disorder or health measures are

Table 3.1.

Correlation Matrix for the Study Variables of Study 2.

	1	2	3	4	5	6	7	8	9	10	11	
1. Self-reported health	——											
2. Inflammatory burden	.125*	——										
3. Neighborhood disorder	.108*	.121*	——									
4. *rs2228145* (1 = C:C or A:C)	-.069	.242**	-.005	——								
5. Family socioeconomic status	.143**	.107*	-.016	.008	——							
6. Age	-.018	.053	-.025	.023	-.108*	——						
7. South	-.020	-.102	-.156**	.009	-.079	-.064	——					
8. Length of residence	-.041	.073	.041	.017	.038	.372**	.151**	——				
9. Neighborhood cohesion	-.051	.064	-.112*	.035	.078	.099†	.170**	.232**	——			
10. Neighborhood disadvantage	.094†	.110*	.145**	-.036	-.221**	-.103†	-.199**	-.073	-.209**	——		
11. Residential stability	-.078	.138*	-.012	.018	.078	.068	.189**	.262**	.156**	-.258**	——	
12. Marital status	-.113*	.102†	-.047	.041	.313**	-.050	-.003	.060	.046	-.142**	.139**	——
Mean	1.936	-.058	0	.231	0	.012	.675	8.970	-.366	0	0	.295
SD	.996	1.461	1	.422	.779	8.012	.469	9.201	9.562	1.028	.995	.457

**$p \leq .01$; *$p \leq .05$ (two-tailed tests); n = 342

nonsignificant, ruling out potential confounding effects of passive and evocative *r*GE attributable to *rs2228145* genotype on selection into neighborhoods.

To test the hypotheses and to correct for clustering bias, regression models with the COMPLEX option in M*plus* and robust maximum likelihood estimators are used in the following analyses.

The Effect of Neighborhood Disorder on Self-Reported Health Status

Poisson regression is used in Table 3.2 because the measure of self-reported health is a count variable. I first checked for potential multicollinearity among variables. The variance inflation factor (VIF) of the independent variables is lower than 1.5, and thus multicolinearity is not identity. To understand the contributions of neighborhood disorder to self-reported health independent of individual and neighborhood socioeconomic status I controlled for family socioeconomic status and neighborhood characteristics in all models.

Model 1 presents the results of regressing self-reported health on the observers' ratings of neighborhood disorder and control variables. As hypothesized, neighborhood disorder is related to self-reported health (odds ratio = 1.049, 95% CI [1.001, 1.100], $p = .045$) even after controlling for the demographic measures and neighborhood characteristics. No variables other than family socioeconomic status (odds ratio = .919, 95% CI [.854, .988], $p = .020$) show a significant effect. The results are consistent with numerous studies (Hill, Ross, & Angel, 2005; Ross, & Mirowsky, 2001, 2009), which have found that residing in disordered neighborhoods has a deleterious effect on physical health.

Table 3.2.
Observer Ratings of Neighborhood Disorder and the Inflammatory Burden as Predictors of self-reported health status

Dependent variable:	Model 1		Model 2	
Self-reported health status (0-4)	*Coeff.* (SE)	Odds Ratio	*Coeff.* (SE)	Odds Ratio
Independent variable				
Observer ratings of neighborhood disorder	.048*	1.049	.038	1.039
	(.024)		(.027)	
Mediator				
Inflammatory burden			.054**	1.055
			(.016)	
Control variables				
SES	-.085*	.919	-.091*	.913
	(.037)		(.036)	
Age	-.001	.999	-.001	1.001
	(.005)		(.005)	
South	.005	1.005	.029	1.029
	(.071)		(.070)	
Length of residence	-.001	.999	-.001	.999
	(.004)		(.004)	
Neighborhood cohesion	.000	1.000	-.001	.999
	(.003)		(.003)	
Concentrated disadvantage (CD)	.018	1.018	.026	1.026
	(.025)		(.024)	
Residential stability (RS)	-.024	.976	-.032	.969
	(.030)		(.032)	
Social isolation (CD × RS)	-.006	.994	-.010	.990
	(.024)		(.024)	
Constant	.659**		.645**	
	(.064)		(.062)	

Notes: Unstandardized coefficients are shown with robust standard errors in parentheses; SES, age, neighborhood cohesion, concentrated disadvantage, and residential stability are centered by mean subtraction; observer ratings of neighborhood disorder is standardized by z-transformation (mean = 0 and *SD* =1); n = 342.
† $p \leq .10$; * $p \leq .05$; ** $p \leq .01$ (two-tailed tests)

Model 2 adds the measure of inflammatory burden to the model. As predicted, inflammatory burden is significantly related to self-reported health (odds ratio = 1.055, 95% CI [1.023, 1.089], $p = .001$). Moreover, consistent with the mediation argument, the relationship between neighborhood disorder and self-reported health status is no longer significant (odds ratio = 1.039, 95% CI [.985, 1.095], $p = .162$) when the measure of inflammatory burden is included

in the model. Using the Satorra-Bentler scaled Chi-square difference test, the finding also suggests that the model with a mediator of inflammatory burden provides a better fit to the data (TRd = 16.152, *df* =2, *p* = .0003).

To more stringently test the mediational model I examined the relative strength of the direct and indirect pathways from neighborhood disorder and self-reported health. Using the Delta approach outlined by MacKinnon et al. (2002), the indirect effect was significant (indirect effect = .011, *p* = .046) whereas the direct effect was not (direct effect = .038, *p* = .163). Twenty-two percent of the variance in self-reported health explained by neighborhood disorder was accounted for by the measure of inflammatory burden.

The Effect of G × E Interaction on Inflammatory Burden

As an initial step in the gene-environment interaction analyses, a multicollinearity test was performed. The VIF ranged from 1.01 to 1.34, indicating that none of the models suffers from the problems of multicollinearity. As can be seen in Table 3.3, the dominant model for the genotype is used in Model 1 through Model 2. Therefore, *rs2228145* SNP is coded as a binary variable with the value 1 if individuals are either heterozygous or homozygous for the minor allele C and 0 for those homogeneous for the major A allele. Controlling for the demographic and control measures, Model 1 shows that the main effects of neighborhood disorder and *rs2228145* are significantly associated with the inflammatory burden index, suggesting that women living in disordered neighborhoods and carrying at least one minor allele have elevated levels of inflammation.

I then examine the moderating effect of variation at *rs2228145* by entering the interaction of neighborhood disorder and this genotype on the regression equation. As hypothesized, the

results show that there is a significant interaction between neighborhood disorder and *rs2228145* in predicting the inflammatory burden index (b = .510, 95% CI [.252, .769], p = .001). Furthermore, analysis using the simple slope test (Aiken & West, 1991) indicated that the slope for respondents with at least one copy of the minor allele is significantly different from zero (b = .551, 95% CI [.348, .754], p = .001), whereas the slope is not significantly different from zero for those homozygous for the major allele (b = .041, 95% CI [-.073, .155], p = .484).

As can be seen in Figure 3.1, the fan-shaped patter is shown. The figure demonstrates that the effect of neighborhood disorder on the inflammation responses is steeper for individuals who carry at least one copy of the minor allele of *rs2228145* than for those who do not. Furthermore, based on the proposed proportion of interaction (PoI) index (Roisman et al., 2012; http://www.yourpersonality.net/interaction/ros.pl), the resulting value of .99 shows support for the diathesis-stress perspective that compare to individuals without the risk allele, those carrying the risk allele are at increased risk for health in response to environmental stressors.

To explicate whether the effects of gene-environment interaction is influenced by the types of coding SNPs, the additive model for the genotype is employed in Model 3 through Model 4. Unlikely the dominant model, the additive model counts the number of minor alleles for the SNP (i.e., 0, 1, and 2) (Lewis, 2002). As shown in Table 3.3, results from Model 3 through Model 4 are identical to results from Model 1 through Model 2. The findings suggest that the main effects of both neighborhood disorder and *rs2228145* SNP on the inflammatory responses are statistically significant. Model 4 then adds the multiplicative interaction term formed by multiplying neighborhood disorder by rs22228145. This interaction is significant. Using the simple slope test, the results show that the slopes for respondents with two copies (b = 1.005, p = .0002) or one copy of the minor allele of *rs2228145* are significantly different from

zero (b = .521, 95% CI [.358, .683], p = .0001), whereas the slope is not significantly different from zero for those homozygous for the major allele (b = .036, 95% CI [-.083, .155], p = .551).

Table 3.3.
Observer Ratings of Neighborhood Disorder and *rs2228145* as Predictors of the Inflammatory Burden

Dependent variable:	Model 1		Model 2		Model 3		Model 4	
Log-transformed Inflammatory burden	b	β	b	β	b	β	b	β
Environment and Genetic Variables								
Observer ratings of neighborhood disorder	.174 *	.119	.041	.028	.173 *	.119	.036	.025
	(.077)		(.058)		(.079)		(.061)	
rs2228145 (1 = C:C or A:C)	.811 **	.234	.808 **	.234				
	(.200)		(.188)					
rs2228145 (2 = C:C; 1 = A:C; 0 = A:A)					.706 **	.235	.685 **	.228
					(.222)		(.219)	
Gene-Environment Interaction								
Neighborhood disorder			.510 **	.182				
× *rs2228145* (1 = C:C or A:C)			(.132)					
Neighborhood disorder							.484 **	.193
× *rs2228145* (2 = C:C; 1 = A:C; 0 = A:A)							(.113)	
Control Variables								
SES	.121	.064	.138	.073	.115	.062	.134	.072
	(.084)		(.086)		(.085)		(.087)	
Age	.003	.017	.003	.017	.003	.015	.004	.020
	(.010)		(.011)		(.010)		(.010)	
South	-.410 *	-.131	-.404 *	-.130	-.387 *	-.124	-.388 *	-.124
	(.165)		(.165)		(.163)		(.163)	
Length of residence	.005	.030	.003	.021	.004	.023	.001	.008
	(.008)		(.007)		(.008)		(.007)	
Neighborhood cohesion	.006	.040	.010	.068	.006	.041	.011	.070
	(.009)		(.009)		(.009)		(.009)	
Concentrated disadvantage (CD)	-.129	-.091	-.135	-.095	-.128	-.090	-.131	-.093
	(.090)		(.089)		(.088)		(.087)	
Residential stability (RS)	.159	.108	.158	.108	.164 †	.112	.163 †	.111
	(.096)		(.095)		(.096)		(.095)	
Social isolation (CD × RS)	.039	.026	.057	.038	.041	.027	.060	.040
	(.082)		(.083)		(.082)		(.082)	
Constant	.001	.001	.019	.013	.003	.002	.036	.025
	(.156)		(.160)		(.162)		(.170)	
R-Square	.124		.147		.124		.151	
ΔR-Square			.023 **				.027 **	

Notes: Unstandardized and standardized coefficients are shown with robust standard errors in parentheses; SES, age, neighborhood cohesion, concentrated disadvantage, and residential stability are centered by mean subtraction; observer ratings of neighborhood disorder is standardized by z-transformation (mean = 0 and *SD* =1); n = 342.
† $p \leq .10$; * $p \leq .05$; ** $p \leq .01$ (two-tailed tests)

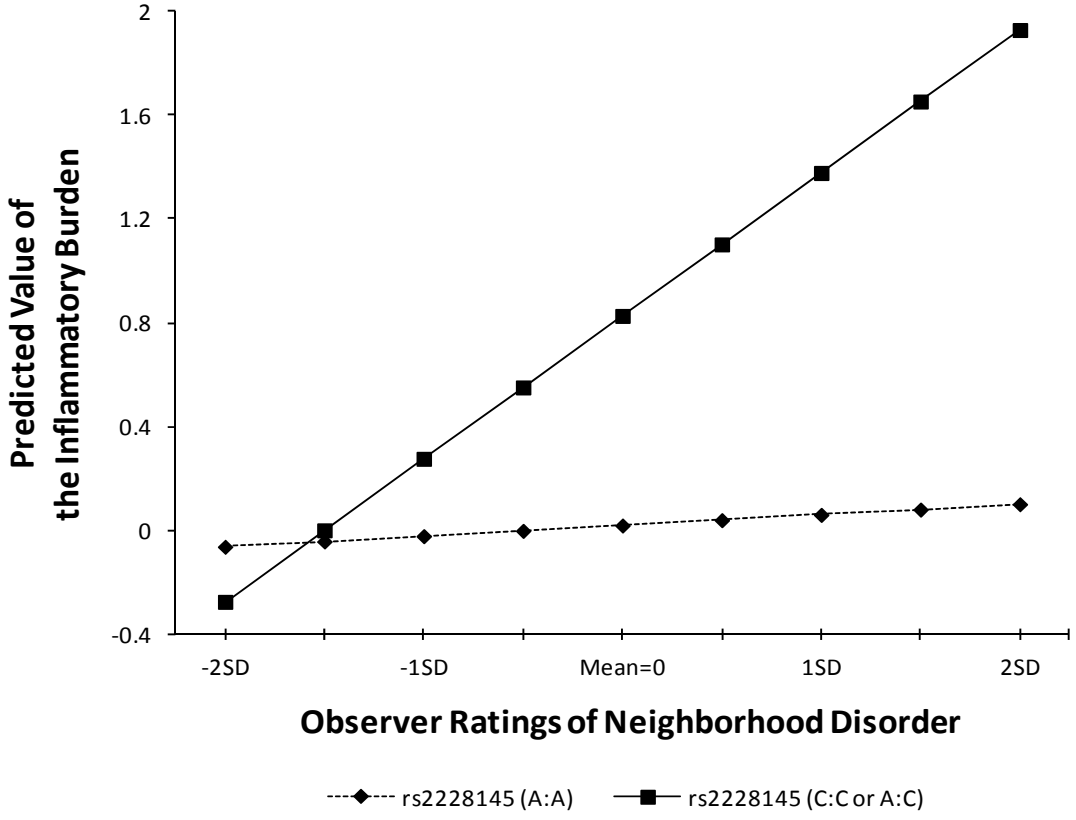

Figure 3.1.
The Effect of Observer Ratings of Neighborhood Disorder on the Inflammatory Burden by rs2228145.

As shown in Figure 3.2, the graph for this interaction in Model4 indicates a fan-shaped pattern identical to those depicted in Figure 3.1. Similar to the dominant model, the PoI value for the additive model is .98, indicating that the model is also consistent with the diathesis-stress perspective. In other words, both the dominant and additive coding schemes tell a similar story[1].

[1] I also tested the recessive model for this SNP, whereby individuals receive a score of 1 if they are homozygous for the minor allele and otherwise receive a zero 0. Unfortunately, I only had 8 (2.34%) participants with homozygous for the minor allele at this SNP. Thus, there was not enough statistical power to use the recessive model (Wang & Zhao, 2003).

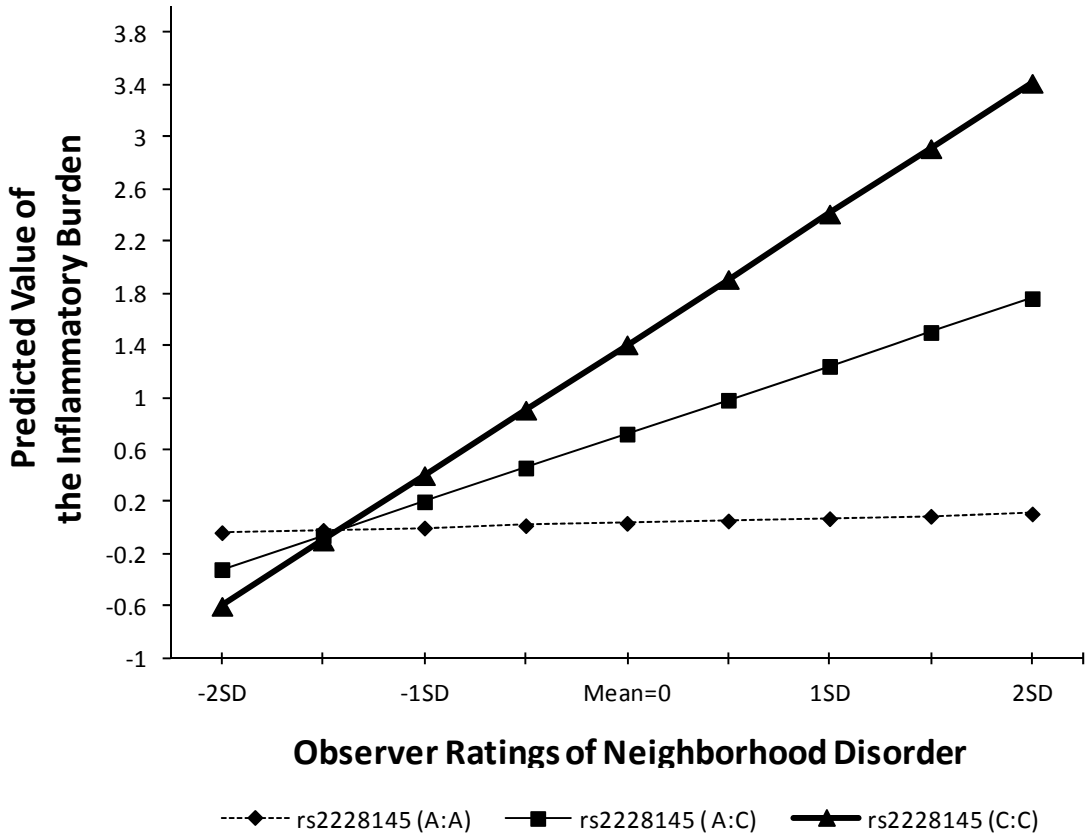

Figure 3.2.
The Effect of Observer Ratings of Neighborhood Disorder on the Inflammatory Burden by *rs2228145* (Additive Coding).

Mediating Effect of Inflammatory Burden

Furthermore, the mediated-moderation model (Preacher, Rucker, & Hayes, 2007) are tested to determine the extent to which the interaction of neighborhood disorder and genotype on self-reported health status is mediated by the inflammatory burden index. Using the Delta method, the results indicated that the indirect effect of G × E on self-reported health is significant [indirect effect = .035, 95% CI (.005, .064) for the dominant model and .038, 95% CI (.008, .068) for the additive model] and accounts for about 30% of the total variance. Consistent with my hypotheses, the combination effect of neighborhood disorder and *rs2228145* gene has a

significant indirect effect on self-reported health through the inflammatory burden as the composite physiological distress index.

Test of the Stress-Buffering Hypothesis

To test the hypothesis that marital status buffers the impact of neighborhood disorder and gene on physiological distress response, I examine the relationship in a three-way interaction regression. As can be seen in Table 3.4, Model 1 shows that both neighborhood disorder (b = .177, 95% CI [.028, .327], p = .020) and the dominant coding of *rs2228145* (b = .804, 95% CI [.408, 1.201], p = .000) are positively and significantly associated with the inflammatory burden index, whereas the direct effect of marital status on the inflammatory burden is nonsigificant (b = .172, 95% CI [-.157, .501], p = .305), indicating that marriage as a source of support does not directly reduce physiological distress. Model 2 enters the three-way interaction of neighborhood disorder × *rs2228145* × marital status in predicting the inflammatory burden index. The findings reveal that there is a significant three-way interaction effect (b = -.483, 95% CI [-.775, -.102], p = .026)[2].

To further examine these relationships, I graph the effect of neighborhood disorder, ranging from -2 to +2 standard deviations from the mean of neighborhood disorder, on the inflammatory burden index. As can be seen in Figure 3.3, when individuals live in disordered neighborhoods, those with at least one copy of the minor alleles on *rs2228145* show the highest level of inflammation, but such an effect is only evident for those in the sample who are not married. Based on a simple slope test, the results suggest that the regression line depicting the relationship between neighborhood disorder and the inflammatory burden is significantly steeper

[2] Although not presented for the purpose of brevity, the results using the additive coding are almost identical to the results using the dominant coding schemas. Detailed results are available upon request.

for individuals with at least one copy of the minor alleles on *rs2228145* and unmarried (b = .587, 95% CI [.356, .818], p = .001) than for married women or for women without this minor allele on *rs2228145*. Among the 342 respondents used in my analysis, 15.5% have at least one copy of the minor allele on *rs2228145* and are unmarried.

Finally, a simple slope difference test, presented in Table 3.4, is used to test which of the slopes statistically differ from each other (Dawson & Richter, 2006). As expected, the results show that unmarried women with the risk allele of *rs2228145* gene are more vulnerable to neighborhood contexts than others, whereas none of the simple slopes are significantly different among the three groups: married women without the risk allele, married women with the risk allele, and unmarried women without the risk allele.

Although not presented for the purpose of brevity, the results using the additive coding of *rs2228145* (2 = C/C; 1 = A/C; 0 = A/A) are almost identical to the results using the dominant coding schemas. Therefore, findings from the current study suggest that marriage is particularly helpful for individuals carrying genetic risk and living in disordered neighborhoods.

Finally, using the mediated-moderation model with the Delta method, the indirect effect of G × neighborhood × marital status on self-reported health status through the inflammatory burden is significant (indirect effect = -.64, p = .048) and accounts for 27.35% of the total variance.

Table 3.4.
Regression Analysis Examining the Relationships among Neighborhood Disorder, rs2228145, Marital Status, and Inflammatory burden

Dependent variable:	Model 1		Model 2	
Log-transformed Inflammatory burden	b	β	b	β
Environment and Genetic Variables				
Observer ratings of neighborhood disorder	.177*	.121	.059	.041
	(.076)		(.080)	
Marital status (1 = marriage)	.172	.054	.305	.095
	(.168)		(.195)	
rs2228145 (1 = C:C or A:C)	.804**	.232	.967**	.279
	(.202)		(.290)	
Gene-Environment Interaction				
Neighborhood disorder			.527**	.188
× *rs2228145* (1 = C:C or A:C)			(.159)	
Marital status			-.592	-.108
× *rs2228145* (1 = C:C or A:C)			(.379)	
Neighborhood disorder			-.065	-.021
× Marital status			(.139)	
Neighborhood disorder × Marital status			-.483*	-.059
× *rs2228145* (1 = C:C or A:C)			(.217)	
Control Variables				
SES	.092	.049	.092	.049
	(.085)		(.089)	
Age	.004	.019	.002	.013
	(.010)		(.010)	
South	-.406*	-.130	-.438*	-.141
	(.169)		(.171)	
Length of residence	.004	.027	.005	.030
	(.008)		(.008)	
Neighborhood cohesion	.006	.041	.009	.057
	(.008)		(.009)	
Concentrated disadvantage	-.125	-.088	-.133	-.094
	(.090)		(.089)	
Residential stability	.149	.102	.146	.099
	(.096)		(.094)	
Social isolation	.044	.030	.063	.042
	(.082)		(.083)	
Constant	-.046	-.031	-.059	-.040
	(.173)		(.192)	
R-Square	.127		.158	
ΔR-Square			.31**	

Notes: Unstandardized and standardized coefficients are shown with robust standard errors in parentheses; SES, age, neighborhood cohesion, concentrated disadvantage, and residential stability are centered by mean subtraction; observer ratings of neighborhood disorder is standardized by z-transformation (mean = 0 and *SD* =1); n = 342.
† $p \leq .10$; * $p \leq .05$; ** $p \leq .01$ (two-tailed tests)

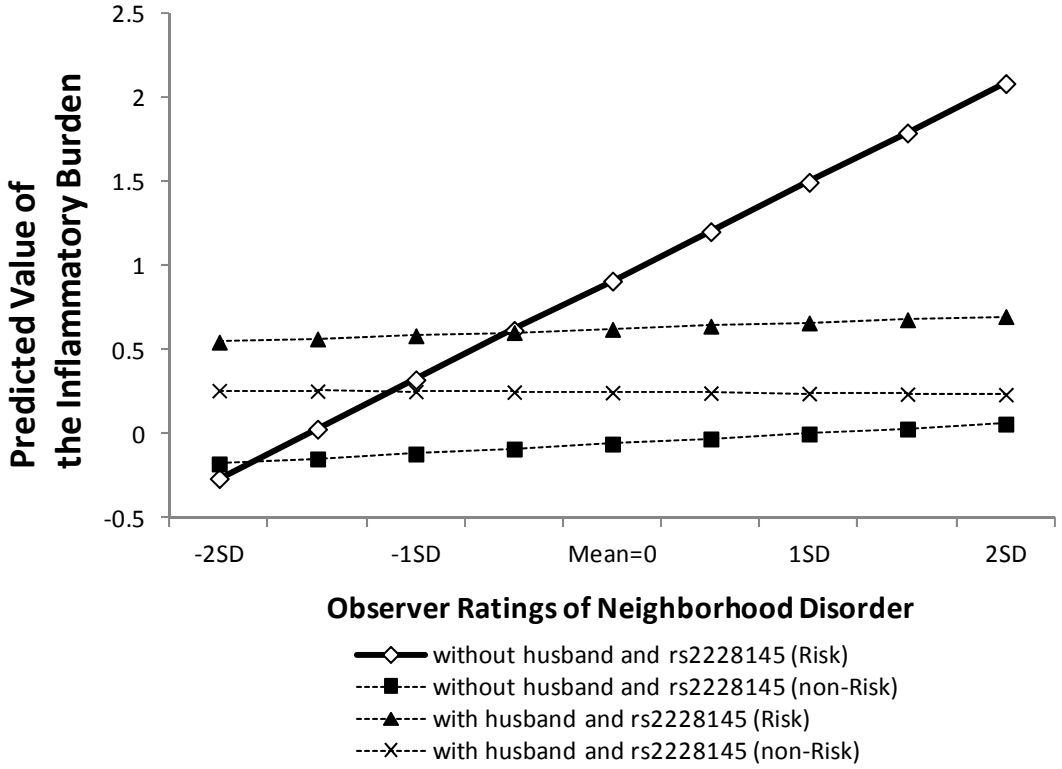

Figure 3.3.
The Effect of Observer Ratings of Neighborhood Disorder on the Inflammatory Burden by *rs2228145* and Marital Status.

DISCUSSION FOR STUDY 2

A number of studies have indicated that neighborhood context plays a crucial role in physical health (Cockerham, 2013; Diez-Roux & Mair, 2010). The stress process model along with classical neighborhood studies (Hill, Ross, & Amgel 2005; Pearlin, 1989; Turner, 2010) propose that prolonged exposure to chronic stress through the fight-or-flight response mechanism is associated with increases in chronic illnesses and suppression of immune function, and such stress is disproportionally represented in economically disadvantaged and disordered neighborhoods. According to this theoretical position, it is expected that an indirect pathway from neighborhood context is related to physiological distress which in turn increases risk of

chronic illnesses. While earlier studies have focused on the relationship between neighborhood characteristics and self-reported health studies, few studies involving objective measures of both neighborhood and physiological distress examine this association among adult African American women.

To understanding this stress process mechanism, the current study used inflammatory markers to assess biological dysregulation as a sign of physiological distress response. First of all, the findings are consistent with previous studies (Aneshensel & Sucoff, 1996; Cohen et al., 2003; Robert, 1998, 1999; Ross & Mirowsky, 2001) indicating that people who live in disordered neighborhoods are more likely to report poor physical health, and that this relationship is independent of demographic characteristics. This is important because the impact of neighborhood disorder on physical health has been found to be confounded with individual socioeconomic characteristics. By controlling for these confounding elements, results confirm that neighborhoods with the visible signs of disorder contribute uniquely to health outcomes.

This analysis also confirmed the stress process mechanism elaborating both the stress-process model and the broken window theory, indicating indirect influences of neighborhood disorder on self-reported health status through an elevated inflammatory response. These findings offer evidence for a potential mediating effect in the stress process model and indicate that, in general, residing in a disordered neighborhood context has deleterious effects on the functioning of biological stress regulatory systems and, in turn, on physical health (Aneshensel & Sucoff, 1996; Chen & Miller, 2013; Cohen et al., 2003; Hill, Ross, & Angel, 2005; Holmes & Marcelli, 2012; Nazmi et al., 2010; Ross & Mirowsky, 2001).

Although the stress process model provides insights in understanding the link between neighborhood context and physical health, this model cannot answer the question of why not all

people living in economically disadvantaged and disordered neighborhoods are distressed and in poor health. The current study extends the existing neighborhood literature to examine gene-environment interaction models (Brody et al., 2013; Caspi et al., 2010). The current results reveal that neighborhood disorder can trigger the inflammatory response, and allelic variation at the IL-6r gene (A>C, *rs2228145*) amplifies the chances that exposure to disordered neighborhood will increase inflammation. More importantly, findings from the mediated-moderation model indicate that neighborhood context and genotype interact to contribute to risk for physical health through the inflammatory burden as a physiological distress response. Therefore, the results provide important evidence that neighborhood effects may not be the same for all people. In fact, the effects of neighborhood and genetic variations are equally important in explaining physical health status.

In addition, in line with the stress-buffering hypothesis (Cohen, 1988; Lin & Ensel, 1989), they suggest that the impact of stress on physical health is reduced as levels of social and emotional support increases. In the long tradition of family sociology and public health (Anson, 1989; Hughes & Waite, 2009; Schwerdtfeger & Friedrich-Mai, 2009; Taylor, 2011), marriage represents a major source of social and emotional support that serves to buffer the effects of stressors on health. Consistent with this line of research, the results from the current study indicate that the effects of neighborhood disorder on the inflammatory stress response and health are not uniform but are most pronounced among unmarried women carrying the minor allele of the IL-6r gene. These results suggest that not all women who carry the genetic risk and live in disordered neighborhoods will experience dysregulated biological systems. It appears that high genetic-risk individuals who are exposed to disordered neighborhoods are better able to protect their physical health when they are married. This is consistent with the stress-buffering

hypothesis, suggesting that health differences can be explained by disparities in social and emotional support.

There are several notable strengths to the current study. First, previous studies have relied upon self-report data. The magnitude of the spurious inflation of relationships may be overestimated due to common method variance and the projection effect. To avoid the bias from using self-reported measures, the current study uses observers' ratings to assess neighborhood disorder and a blood draw to assess biomarkers of inflammation. Second, this study incorporates the gene-environment interaction model into neighborhood studies and employs a mediated-moderation model to simultaneously test mediation and moderation.

Although the present study offers important findings concerning the relationships between neighborhood context and physical health, this study is not without limitations. First, the sample in this study focuses upon African-American families living in Iowa and Georgia at the time of recruitment. This sample allows me to investigate how neighborhood context, genotype, and marriage combine to influence physical health among a racial group who often lives in disordered neighborhoods (Peterson & Krivo, 2010) and experience high mortality rates (The National Center for Health Statistics, 2011). While there is little reason to believe that the theoretical processes tested in the present study are specific to African Americans, it is clearly the case that these findings need to be replicated using more diverse samples. Second, the sample poses another and possibly problematic limitation. Given that the adults in the sample were selected because of their status as primary caregivers, virtually all of them were women. The lack of men in the sample prevented us from being able to assess the extent to which the current results might differ by gender. For instance, although marriage has beneficial effects on physical health, some studies claim that both women and men benefit from marriage (Horwitz et al.,

1996), whereas other studies indicate that marriage benefit is particularly important for men (Sbarra, 2009). Hopefully, future studies will strive to replicate the findings using samples that contain both males and females. Third, the current study focuses only on marital status rather than marital quality because the sample size precluded the use of an internal moderation model. Future studies can incorporate marital relationship measures by examining the extent to which marital quality and/or conflict moderate the probability that exposure to disordered neighborhoods will lead to physiological distress and poor health status. Finally, studies have argued that people select themselves into neighborhoods. This is a common and problematic confounder of general survey data (Leventhal et al., 2000; Sampson et al., 2012). Unfortunately, this possible selection bias is nearly impossible to rule out in non-experimental analysis. In my study, individual characteristics and neighborhood related measures have been controlled in all models in order to reduce neighborhood selection bias.

In conclusion, several lines of evidences indicate that health and well-being is a product of individual characteristics and the environment in which people live (Diez-Roux & Mair, 2010; Sampson, 2013; Wikström, 2004). Controlling for individual characteristics and possible confounding factors , these findings provide strong evidence that neighborhoods where people live play fundamentally distinct roles in physical health. More importantly, the theoretical implication of stress process mechanism is that the inflammatory burden is a plausible physiological pathway linking neighborhood disorder to physical health. Such findings suggest that the index of inflammatory burden as a signal of physiological distress provides a more precise understanding of how neighborhood context influence physical health. Finally, the current results show that neighborhood disorder and genotype interact to influence physiological distress response and health outcomes. In addition, a protective effect of marriage in

ameliorating such relationships is evident. Thus it appears that both genetic variation and marital status combine to influence the impact of neighborhood disorder on biological dysregulation and physical health. Based upon these results, biological and social scientists are not locked in a zero-sum game. In fact, both social and physiological factors are fundamental in understanding the mechanisms of the body.

CHAPTER 4

STUDY 3: NEIGHBORHOOD ENVIRONMENTS, STREET CODE, AND VIOLENCE

In the past two decades, culture has resurged as an important topic in neighborhood studies (Messner & Zimmerman, 2012; Steffensmeier et al., 2010; Wilson, 2009). Scholars have proposed that neighborhood effects should focus not only on structural contexts, but also on cultural processes because cultural values provide a frame for individuals to understand their lives. In *Code of the Street*, Elijah Anderson (1999) attempted to incorporate culture into neighborhood structural models and to answer the basic questions of how everyday life and behavior is shaped by neighborhood structural characteristics and how such a relationship between behavior and structure is connected by cultural adaptations. He proposed that street code represents an oppositional subculture and is used as a guide for handling interpersonal conflict in disadvantaged neighborhoods. Numerous empirical studies have supported Anderson's argument that commitment to the street code is a critical link between neighborhood characteristics and crime/violence (Baumer et al., 2003; Brezina et al., 2004; Matsuda et al., 2013; Nowacki, 2012; Oliver, 2006; Parker & Reckdenwald, 2008; Sampson, Morenoff, & Raudenbush, 2005; Simons et al., 2012; Stewart, Simons, & Conger, 2002, Stewart & Simons, 2006).

While a handful of existing studies support Anderson's ideas, few studies have considered his code-switching hypothesis. Unlike classical sub-culture theories which tend to assume that sub-cultural values and beliefs are relatively stable (Wolfgang & Ferracuti, 1967), Anderson (1999, 2011) identified "decent" and "street" code values and indicated that people can

code switch between these two values. Unfortunately, no study to date has examined the assumption of code-switching. Particularly, it has not been well-studied whether change in adopting the street code is linked to change in criminal and violent behavior. In fact, clarifying the temporal nature of street code is important because evidence of change would strategies for violence and/or crime prevention in disadvantaged areas.

Furthermore, Anderson (1999, 2011) emphasizes an interactive effect between street code and the social environment in that adherence to code values can be switched over time when social environments change. This occurs because adherence to codes arises from individual responses to social situations. Therefore, social factors might play a role in explaining changes in code adherence. It is important to examine whether changes in social environment are associated with change in street code adherence over time.

Using a longitudinal framework, the purpose of this study is to further elaborate the relationships among environmental contexts, street code, and violent behavior. To better understand how code-switching actually takes place, I use the longitudinal data from the Family and Community Health Study that was designed to assess the linkages among families, communities, peers, and well-being in an African American sample. Building on Anderson's arguments, I tests hypotheses regarding the factors that predict the extent of stability and change in street code adherence over time.

THE CODE OF STREET THESIS

Anderson's studies expanded on the classic sub-cultural theories (e.g. Cohen, 1965; Cloward & Ohlin, 1960) by incorporating a social disorganization theoretical framework. He suggested that neighborhood studies must consider both structural and cultural process

mechanisms. Based on four years of ethnographic research in the Philadelphia area, Anderson (1999) provided insight into how neighborhood characteristics were related to the development and adoption of a sub-cultural value code, and demonstrated how residents of disadvantaged neighborhoods adopt a subculture to handle their everyday routines. He observed that lack of educational opportunity, racism, crime, economic stress, and despair pervade disadvantaged areas. As a result, residents of these disadvantaged neighborhoods, which are often typified by racial and social isolation, feel alienated from mainstream society, do not trust the police or other formal control systems, and are very sensitive to signs of disrespect. Violence is the most readily available means for achieving or maintaining status on the streets. Young people living in these disadvantaged and criminogenic neighborhoods are more likely to use toughness and violence as a means of self-preservation and to avoid victimization.

Based upon these observations, Anderson proposed the concept of "street code." He indicated that the street code is a set of informal rules governing youths' daily lives and interpersonal public behavior. Elements of the street code as an oppositional culture include obtaining and maintaining respect through a reputation for violence, ruthlessness, and a keen understanding of swift justice for shows of disrespect. Residents who accept the street code acquire "juice" by performing their fearlessness in combat. Therefore, the heart of this code is to "campaign for respect" by establishing reputation for toughness and violence.

The street code does not exist in a vacuum but is influenced by social contexts (Anderson, 2002). In order to survive in disadvantaged neighborhoods, people living in those areas adhere to the street code in order to govern the application of violence for the purpose of resolving disputes and establishing a reputation for being violent. This is created by fostering a self-image based on "juice" as respect and power on the street. In other words, individuals'

adoption of street code is dependent upon the characteristics of where they live. Anderson attempted to link sub-cultural with the structural factors and hypothesized that street code is a means of acquiring respect in disadvantaged neighborhoods.

While the code of the street thesis is applicable to any racial, ethnic, or national context (Brookman et al., 2011; Jimerson & Oware, 2006), street code is particularly salient for African Americans (Cobbina, Miller, & Brunson, 2008; Matsueda, Drakulich, & Kubrin, 2006; Taylor, Esbensen, Brick, & Freng, 2010) because they are more likely to reside in the disadvantaged social settings described by Anderson. They are more likely to reside in neighborhoods with high crime rates and with high levels of social disorder (McNulty, 2001; Peterson & Krivo, 2010). In these neighborhoods, racial discrimination is common. Therefore, Anderson suggested that African Americans who experience racial discrimination may "pride themselves on knowing and being able to enact the code of the street" (Anderson, 1988, p.94). Based on his observations, street code is a major means for African Americans to gain social status and to self- protect in disadvantaged neighborhoods.

In the past decade, several studies support Anderson's argument that violence is shaped by an oppositional cultural orientation, and street code thrives in places lacking neighborhood structural resources. It is in these neighborhoods that respect becomes indispensable for self-preservation. For instance, Brezina et al. (2004), using three waves of data from the National Youth Survey, found that street code adoption is related to youth violence, and he indicated that Anderson's thesis can be generalized to all disadvantaged social settings. Some studies also found evidence that African Americans who resided in disadvantaged neighborhoods, who had been raised in street-oriented families, and who felt racial discrimination were likely to adopt street code, which in turn increased the likelihood of engaging in violent delinquency (Sampson,

Morenoff, & Raudenbush, 2005) or of suffering violent victimization (Stewart et al., 2006b). Stewart and Simons (2006) revealed that an individual-level adoption of street code could be seen as a cognitive schema that mediated some of the association between neighborhood characteristics, informal social control, and involvement in violent crime. Furthermore, Stewart and Simons (2010) indicated that the effect of adopting the street code on violence is increased when people live in a neighborhood where the street culture is highly prevalent. Unfortunately, although previous studies have provided evidence for the link among neighborhood characteristics, street code, and violent behavior, little is known about the extent of stability or the nature of change in street code over time.

ASSUMPTION OF CODE SWITCHING

In criminological studies, the issue of continuity and stability in crime/deviance from childhood into adolescence and adulthood has been received with considerable interest (Loeber, 2012; Sampson & Laub, 1997). There are two competiting approaches to explain the age-crime relationship (Nagin & Paternoster, 2000; Simons et al., 1998). One is the latent trait approach. This approach focuses on the manifestation of a latent propensity to explain the stability of antisocial behavior. For example, Gottfredson and Hirschi (1990) proposed the self-control theory and asserted the age-invariance hypothesis. They argued that crime/deviance is relatively stable after age ten because inept parenting in early childhood causes children to develop low self-control and that this self-control then remains low throughout the life course. However, the age-crime invariance hypothesis has not been supported by many longitudinal studies (e.g. Burt & Simons, 2006; Giordano et al., 2008; Hay & Forrest, 2006; Na & Paternoster, 2012; Sampson

& Laub, 2003; Turner & Piquero, 2002;). In fact, numerous empirical studies observe that there is a change in latent traits and crime/deviance over the life course.

Another approach is state dependence. Unlike the latent trait approach, the state dependence approach claims that patterns of offending across the life course are not the result of a stable latent trait but of social factors that change the developmental trajectories of criminal and deviant behaviors over time (Sampson & Laub, 1993). Recently, many studies have provided a social explanation of change in crime/deviance over the life course. For instance, Kreager et al. (2010) reported that the transition to motherhood resulted in desistance from criminal activities for women living in disadvantaged communities because it changed their daily activities and life experiences. Although there is a large body of longitudinal research on changes in crime/deviance, street code is rarely considered. It is unclear whether changes in adopting the street code occur over time, with these changes, in turn, fostering changes in crime.

While Anderson's work emphasized street code in disadvantaged neighborhoods, he did not assume that there is a single cultural value in those neighborhood contexts. In fact, he observed that not all people living in disadvantaged or disorderly neighborhoods have internalized street code, and he hypothesized that the two ideal types of code values coexist in disadvantage neighborhoods. Social behaviors in disadvantaged neighborhoods are characterized by both the street and decent codes (Anderson, 1999, pp. 37-53). Many residents follow the decent code, which reflects middle-class values and mainstream society activities. Decent residents believe that success is earned by working hard and maintaining a law-abiding lifestyle. For example, Parker and Reckdenwald (2008) examined Anderson's argument, and found that youth living in disadvantaged neighborhoods who adopt the decent code are more likely to

embrace mainstream culture and norms and emphasize self-reliance and hard work. They are also less likely to engage in violence.

In contrast to the decent code, street code adhering residents place less emphasis on work and education and are more devoted to the oppositional culture because of their deep distrust of the formal social structure (Anderson, 1994). Anderson claimed that youths living in "street families" are more likely to receive hostility and harsh punishments in their family and experience alienation from mainstream culture. The resulting weak social bonds cause these youth to adopt the street code.

More importantly, the street code as a subculture of informal rules is not fixed and unchangeable over time (Anderson, 1999, p. 36). Anderson (2011) emphasizes that "most people code switch from time to time, depending on how they read a particular [social] situation" (p. 189). Therefore, people can switch coded from decent to street, or street people can act decent. In particular, code values can be switched between decent and street over time depending on changes in the social situation. In essence, the basic assumption of code-switching is consistent with the state dependence approach. Unfortunately, previous research has been limited to cross-sectional analysis and therefore does not allow for an examination of stability and change over the lifespan in street code.

Although no study to date has examined the assumption of code-switching, several studies have examined values as cognitive schemas that are flexible and changeable over the life span (D'Andrade, 1995; Garro, 2000). According to neuroscientific research, for instance, Davidson and Begley (2012) found that human brains and cognitive schemas are not unchangeable. In contrast, these can change over time in response to social environments. Similarly, Agnew (2011) also reported that that code values change over the lifespan, shaping

how people react to social situations. In other words, core values, beliefs, and schemas are relatively dynamic cognitive characteristics and structures (Drummond, Bolland, & Harris, 2011; Walker & Underwood, 2003). These lines of research, suggest that the commitment to the code of the street might also change over time. The main goal of this study is to extend the existing studies of the code of the street thesis by using a longitudinal data to examine the dynamic nature of the street code. The present study hypothesizes that street code changes over the lifespan in response to changes in neighborhood conditions.

SOCIAL CONDITIONS AND CODE SWITCHING

When the assumptions of street code are supported, there is a question of what drives change in street code over time. Anderson proposed that variation in code values is related to various demoralizing and hostile neighborhood and family conditions because both the street and decent codes are not produced in a vacuum but are embedded in the social system. According to Anderson's observations and previous studies, the high rates of neighborhood violence, racial discrimination, hostile family environments, and deviant peers increase the likelihood of adopting the street code (Baron, Kennedy, & Forde, 2001; Baumer et al., 2003; Brezina et al., 2004; Haynie & Payne, 2006; Simons et al., 2003; Stewart & Simons, 2006).

First of all, Anderson argued that street code is constructed through the neighborhood in which people live. In disorderly neighborhoods, individuals adopt street code as a means of survival in a demoralizing environment without safe open spaces and monetary opportunity (Anderson, 1999; Baumer et al., 2003; Oliver, 2006). For example, Stewart and Simons (2006) found that people living in neighborhoods where violence is common are more likely to express fear of victimization and feelings of being unsafe. Thus, individuals who live in such dangerous

neighborhoods are more likely to use the street code as a strategy for the purpose of self-protection.

Furthermore, Anderson (1999) indicated that "a sense of discrimination justifies withdrawal of attributions of legitimacy from conventional rules of conduct" (p.212), thereby increasing the probability that street code and violence will be used to achieve status and gain respect. As a result, being a victim of racial discrimination results in some African Americans adopting the view that people are untrustworthy and exploitive and that African Americans have restricted opportunities for achieving status and obtaining respect in mainstream culture. Therefore, some African Americans protect themselves by adopting the street code as a necessary and effective strategy to fend off victimization (Bernard, 1990; Burt, Simons, & Gibbons, 2012; Simons et al., 2003, 2012; Stewart & Simons, 2006).

In addition to neighborhood violence and racial discrimination, Anderson (1999) emphasized the importance of family environments in the development of code values. According to his observation in disadvantaged African Americans neighborhoods, two types of families often coexist in the same neighborhood. One type is "decent" and another is "street". He indicated that most families are "decent" in which these parents tend to accept mainstream values and emphasis working hard, saving money, and hope for the future. In particular, parents in decent families tend to be more authoritative, less punitive in their parenting style, and discourage violence (Burton & Jarrett, 2000; Simons et al., 2012). By contrast, "street" families reject mainstream values and often show a lack of consideration for other people. Anderson (1994; 1999) reported that parents in street families often engage in ineffective parenting strategies and are immersed in the street code. They tend to emphasis violence and toughness and thus are more likely to use harsh parenting strategies as a means of teaching their children. As a

result, children who grow up in these families learn that violent behavior is a justified means to solve problems (Agnew, 1994; Heimer, 1997). Consistent with Anderson's argument, there is growing evidence that parenting styles could play a central role in understanding street code adoption (Baron, Kennedy, & Forde, 2001; Brezina et al., 2004; Nowacki, 2012; Simons et al., 2012; Stewart & Simons, 2006).

Finally, Anderson (1990) stressed that disadvantaged African Americans are more influenced by their friends than are high status blacks or other racial groups. Similar to social learning theory, street code is also learned through interaction with others within specific social contexts (Agnew, 1994). Therefore, affiliation with deviant peers is a strong risk factor for street code adoption. Numerous studies have provided evidence that adolescents learn street code through interaction with peers (Brezina et al., 2004; Matsuda et al., 2013; Simons & Burt, 2011). For instance, using data from 125 homeless male street youth, Baron, Kennedy, and Forde (2001) found that both harsh parenting and affiliations with deviant peers were positively associated with street code. In addition, street code mediated the effects of parenting and deviant peers on violent behavior. Matsuda and colleagues (2013), using the national representative data, also supported the link among gang membership, street code adoption, and violent behavior.

According to Anderson's thesis, street code is potentially malleable over the lifespan depending upon social contexts in which people live and interact with each other. Although much of the research suggests that individuals adopt code values and choose their behaviors based upon the social environment, research has neglected to examine how social environment factors help in understanding change in street code adherence over time. The current study focuses on four socially demoralizing factors identified by Anderson (1990; 1999), including neighborhood violence, hash/inept parenting, racial discrimination, and deviant peers. I expect

that change in the adoption of the street code over time can be explained by change in the measures of these social conditions and that these changes in street code, in turn, predict change in violent behavior.

DO GENDER DIFFERENCES EXIST?

One of the most consistent empirical findings in criminology is that women commit far fewer violent crimes (Belknap, 2007; Daly & Chesney-Lind-Lind, 1988) and adopt less hostile orientations (Lei et al., 2013; Messerschmidt, 1993; Schrock & Schwalbe, 2009) than men. More importantly, feminist criminological scholars have argued that there is "significant differences in the ways that women experience society compared with men" (Daly, 1998, p.98). Similar to feminist research, Anderson (1994, pp.91-92) observed that the ultimate form of respect through street code on the inner-city street is reserved for men and argued that adopting the street code is predominately male-oriented. Basically, women gain respect and maintain status through competition over boyfriends and through men's protection. In other words, Anderson assumed that there are gender differences in street code adoption, with men being more likely to the adopt street code than women.

Unfortunately, the results of previous studies regarding the street code thesis largely ignore gender or only use gender as a control variable (Cobbina, Miller, & Brunson, 2008; Miller & Mullins, 2009). To date, there are few studies taking into account gender differences, and this research has produced mixed results. Some studies (Nowacki, 2012; Taylor, Esbensen, Brick, & Freng, 2010) have supported Anderson's observation that code values are gendered such that while women who live in disadvantaged neighborhoods may have chances to adopt street code, men are more likely to embrace street code than women. However, other research has indicated

that females as well as males living in disadvantaged neighborhoods claimed to use violence as a means of maintaining respectable identities and reputations (Brunson & Stewart, 2006; Jones, 2010; Ness, 2004). Moreover, this research suggests that there are no gender differences in adopting the street code. Brunson and Stewart (2006), for example, found that both African American men and women living in a disadvantaged neighborhood embraced street code values and used violence to establish their social status and identity.

Given this mixed evidence, it remains unclear as to whether there are gender differences in adoption of the street code. Further, no study has examined potential gender differences in changes in the adoption of the street code over time. In the present study, I assess whether change in street code is more likely for men than women

RESEARCH DESIGN FOR STUDY 3

Sample

This study attempts to examine the dynamic hypothesis of street code. The hypotheses were tested using Waves 1 through 5 of the Family and Community Health Study (FACHS; Simons et al., 2011). FACHS was designed to identify neighborhood and family processes that contribute to the development of African Americans. The sample strategy was intentionally designed to generate families representing a range of socioeconomic statuses and neighborhood settings. Each family included a child who was in 5th grade at the time of recruitment.

In order to recruit households from neighborhoods that vary in demographic characteristics, researchers drew a probability sample of respondents through a multistage cluster sampling procedure. Thus, this data goes beyond urban-based studies and can effectively examine African Americans in different community situations. The first stage clusters, defined as

census Block Group Areas (BGAs) using 1990 census data, were selected to represent the diversity of communities where African Americans lived outside of densely populated inner cities. Rural, suburban, and modestly populated metropolitan areas were sampled, but the clusters excluded BGAs in Iowa and Georgia in African-American households that made up the lower 10 % of the population and the percentage of families with children living below the poverty line ranged from 10 percent to 100 percent.

In the second stage, recruitment strategies differed in Georgia and Iowa. In Iowa, families were recruited from 114 census BGAs through a sampling frame, which includes rosters of all African-American students in grades four to six in the public school system. Thus, the sampling criteria included children in households who: (a) studied in the public school system, (b) studied in grades four to six with an age range of 10 to 11, (c) were African American, and (d) were on the rosters of residents' addresses (excluded homeless or illegal residents' addresses). After that, researchers randomly selected households from these rosters and contacted them to determine their interest in participating. Candidates who declined were removed from the rosters, and other households were randomly selected until the required number of households from each BGA had been recruited. In Georgia, the sampling frame was derived from community liaisons. These community liaisons were compiled from rosters of children within 115 BGAs who met the above sampling criteria. Households were then randomly selected from these rosters and contacted to determine their interest in participating in the project. Finally, at the first wave, the FACHS sample consisted of 889 African American children. At the study's inception in 1997-1998, about half of the sample resided in Georgia and the other half in Iowa; all of the children were in the 5th grade and averaged ten years of age. Of the 889 respondents at Wave 1, 779 were re-interviewed at Wave 2, 767 at Wave 3, 714 at Wave 4, and 689 at Wave 5 (77.50% of the

original sample). Details regarding recruitment are described by Gibbons and colleagues (2004) and Simons and colleagues (2011). The second, third and fourth waves of data were collected from 1999 to 2000, 2001 to 2002, 2004 to 2005, and 2007 to 2008 to capture information when respondents were ages 12 to 13, 14 to 15, 17 to 18, and 20 to 21 years, respectively.

The current study involves both individual and neighborhood characteristics. The measures of neighborhood characteristics were created using the 2000 census Summary Tape File 3 (STF3A) which was geocoded with participant's residential addresses at Wave 1. Additional details regarding neighborhood data can be found in Simons and colleagues (2005). At Wave 1, FACHS included 155 census tracts: 88 in Iowa and 87 in Georgia. Of the 889 respondents, 19% of the respondents have primary caregivers with less than a high school education, 55% live in a single parent family, and 37% live below the poverty line. Median family income is $ 27,500. For the 155 census tracts based on the 2000 Census, 58% of the neighborhoods are urban areas, per capita income are 18226.7, and 33% have a population more than half of which is African American. The average poverty rate in 2000 is 15 percent (*SD* = .11).

Measures

Violence. Violence was assessed at Waves 1 using respondents' self-reports on the conduct disorder section of the Diagnostic Interview Schedule for Children, Version 4 (DISC-IV; Shaffer et al. 1993). The DISC was developed over a 15-year period of research on thousands of children and parents. Several studies show that the DISC-IV has acceptable levels of test-retest reliability and construct validity (Simons et al., 2012; Stewart & Simons, 2010). Respondents reported (1 = yes, 0 = no) whether they had engaged in 8 aggressive behaviors in

the past year such as cruelty to animals, damaging property, fighting with weapons, and hurting another. The maximum possible score of eight corresponds to a subject responding that they had engaged in all of the various acts. Cronbach's alpha for the summated items was approximately .90 at each wave.

I assessed violence at Wave 5 using five items adapted from Elliott's (Elliott, Huizinga, & Menard 1989) instrument. Respondents reported whether, in the past year, they had engaged in aggressive behaviors such as fighting with weapons, carrying a hidden weapon, shooting or stabbing someone, hurting someone, or pulling a knife on someone. The maximum possible score of five corresponded to subjects responding that they had engaged in all of the different acts. Cronbach's alpha for the summated items was .73.

Adopting the street code. At Waves 1 through 5, the respondents completed the seven-item Street Code scale developed by Stewart and Simons (2010). Of the seven items, only five items were available across all five waves of data. Respondents were asked to indicate the extent to which they agree (1 = strongly disagree and 4 = strongly agree) with statements such as: people will take advantage of you if you don't let them know how tough you are; people do not respect a person who is afraid to fight for his or her rights; sometimes you need to threaten people in order to get them to treat you fairly; it is important to show others that you cannot be intimated; and, people tend to respect a person who is tough and aggressive. Responses to the 5 items were summed to form a measure of adopting the street code. Higher scores indicated that the respondent's beliefs correspond with high levels of street code adoption. The coefficient alpha for the scale was at .62 at Wave 1, .67 at Wave 2, .70 at Wave 3, .70 at Wave 4, and .81 at Wave 5.

Racial discrimination was assessed at Waves 1 through 5 using the 13-item Schedule of Racist Events. This instrument has strong psychometric properties and has been used extensively in studies of African Americans of all ages (Landrine & Klonoff, 1996). The items focus on the extent (1 = never, 4 = several times) to which respondents experienced various discriminatory events during the preceding year (e.g., how often has someone yelled a racial slur or racial insult at you just because you are African American? How often have the police hassled you just because you are African American? How often has someone threatened you physically just because you are African American?). Cronbach's alpha for the scale was roughly .90 at each wave.

Deviant peers. At Waves 1 through 5, the target youth reported their affiliation with deviant peers using an instrument adapted from the National Youth Survey (Elliot, Huizinga, & Menard, 1989). They were asked how many of their close friends (1 = none, 2 = half, and 3 = all) had engaged in each of the 12 deviant behaviors in the past years. The items focus on acts such as using tobacco, to more serious violations, such as stealing something, attacking someone with a weapon with the idea of hurting them, and using crack or cocaine. Cronbach's alpha for this scale was .83 at Wave 1, .85 at Wave 2, .85 at Wave 3, .85 at Wave 4, and .83 at Wave 5.

Harsh/inept parenting. This construct consisted of 4 questions at Waves 1 through 5 regarding how often during the past year the primary caregiver engaged in verbal and physical hostilities (e.g. How often did your mom get angry at you? How often did your mom insult or swear at you? How often did your mother criticize you? How often did your mother get so mad at you that they broke or threw things?) when disciplining the respondent. The response format for items ranged from 1 (never) to 4 (always). Higher scores indicated a higher level of maternal

hostility. Research shows this scale has high validity and reliability (see Simons et al. 2007). Cronbach's alpha was .48, .55, .55, .63, and .65 for Waves 1, 2, 3, 4, and 5, respectively.

Neighborhood crime was measured at Waves 1 through 5 using a revised version of the Community deviance scale developed for the Project on Human Development I Chicago Neighborhoods (PHDCN; Sampson, Raudenbush, & Earls, 1997). The 3-item measure asks respondents to report how often (1 = never, 3 = often) behaviors such as fighting with weapons, gang fights, sexual assaults, or robberies occur within their neighborhood. The scores were summed to form a measure of neighborhood crime. Cronbach's alpha was .59, .52, .57, .65, and .64 for Waves 1, 2, 3, 4, and 5, respectively.

Hostile/demoralizing environments. To measure this construct, a structural equation modeling with multiple indicators was used. Figure 4.1 depicts a latent variable of hostile/demoralizing environments. Using confirmatory factor analysis, all factor loadings were significant and in the expected direction. For example, at Wave 1, the factor loadings were .63 for racial discrimination, .63 for deviant peers, .38 for harsh parenting, and .48 for neighborhood crime. I used the Nunnally (1978) reliability formula to assess the reliability of this composite measure. This technique uses information regarding the internal consistency of each scale being combined to determine the reliability of the new aggregate measure. With this procedure, the reliability of a composite measure of hostile/demoralizing environments was approximately .90 at each wave.

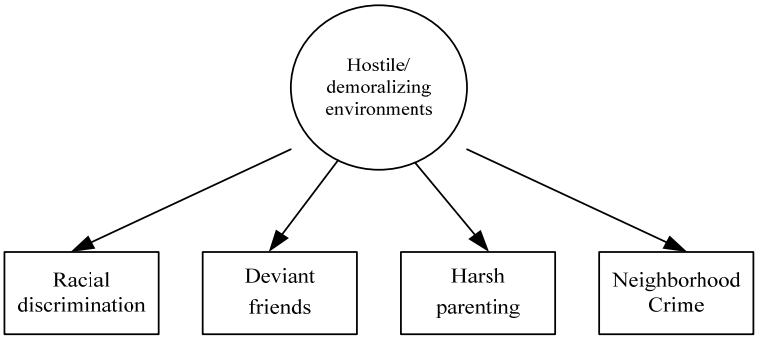

Figure 4.1.
Latent Variable of Hostile/Demoralizing Environments

Concentrated disadvantage was assessed with 2000 STF3A census tract data. Following previous studies (Sampson, Raudenbush, & Earls, 1997), the scale include five items: average per-capita income, the percentage of unemployment, the percentage of residents below the poverty threshold, the percentage of female-headed households, and the percentage of those receiving public assistance. To provide equal weight for each item, per capita income was reverse-coded, and I used factor scores obtained through principal-components analyses to form the scale. Factor loadings ranged from .74 for per capita income to .88 for the percentage of residents below the poverty threshold. Cronbach's alpha for the measure was .89.

Racial composition was assessed by using census data regarding the percentage of white residents in the respondent's census tract in 2000 (M = 57.72, *SD* = 28.37).

Control variables. To avoid overestimated results, the analyses controlled for family SES and single family status.

Analytic Strategy

All data analyses were performed with M*plus* 7.0 (Muthén & Muthén, 2012). To understand the growth trajectory of street code, I employed the unconditional Latent Growth Model (LGM) with individually-varying times of observation to examine whether there is a significant change in the mean level of adopting the street code over time and whether those shapes show linear or nonlinear growth (Curran & Bollen, 2001). Because age range varied across waves, individually varying times of observation were used, and age was centered at age 9. A different test using the log-likelihood was used to compare the model fit of the linear and non-linear models (M*plus*, http://www.statmodel.com/chidiff.shtml). Then, if the significant mean levels of the slope and the quadratic term are found, a peak time of growth curve is calculated using the following equation:

$$Time = -\frac{\pi_1}{2\pi_2}$$

where π_1 is the parameter of the slope term, and π_2 is the parameter of the quadratic term. In addition, I tested for differences between the models for males and females using multiple group analysis. I began by estimating a model that constrained the paths for males and females to be equal. Next, I estimated a model that freed the coefficients to vary by gender. The chi-square difference between the models was significant, indicating gender differences in growth rates of adopting the street code.

The LGM with time varying covariates (Preacher et al., 2008) is used to test the hypothesis: change in the adoption of the street code over time can be explained by change in the measures of socially demoralizing environments. Figure 4.2 shows a graphical illustration of this model. Next, I used a parallel LGM to simultaneously examine change in the adoption of the

street code and change in socially demoralizing environments. This model simultaneously includes two LGM models.

Finally, the mediating model examines growth rates of socially demoralizing environments as a mediator of growth rates of adopting the street code on change in violence. To assess the model fit in the mediation model, Steiger's Root Mean Square error of approximation (RMSEA) and a chi-square were used. A RMSEA indicates a close fit when it is smaller than .05 (Bentler, 1990). All direct and indirect effects were examined using M*plus* with bootstrap = 1000, which used bias corrected and accelerated bootstrap confidence intervals (95%) to assess statistically significant mediation (MacKinnon, 2007; Mallinckrodt et al., 2006). The bootstrapping method for assessing the indirect effects is superior to traditional approaches (e.g. Baron & Kenny 1986) as it estimates direct and indirect effects simultaneously, does not assume a standard normal distribution when calculating the p-value for the indirect effect, and repeatedly samples the data to estimate the indirect effect (Mackinnon, 2007; Preacher, Rucker, & Hayes 2007). The full maximum likelihood (FIML) method is used to handle all of the missing data in the current study. This method assumes that missing data are randomly distributed and are unrelated to the dependent variable (Graham, 2009). This assumption is met in the FACHS sample as missing data are derived from the random attrition associated with a longitudinal design (Simons et al., 2012).

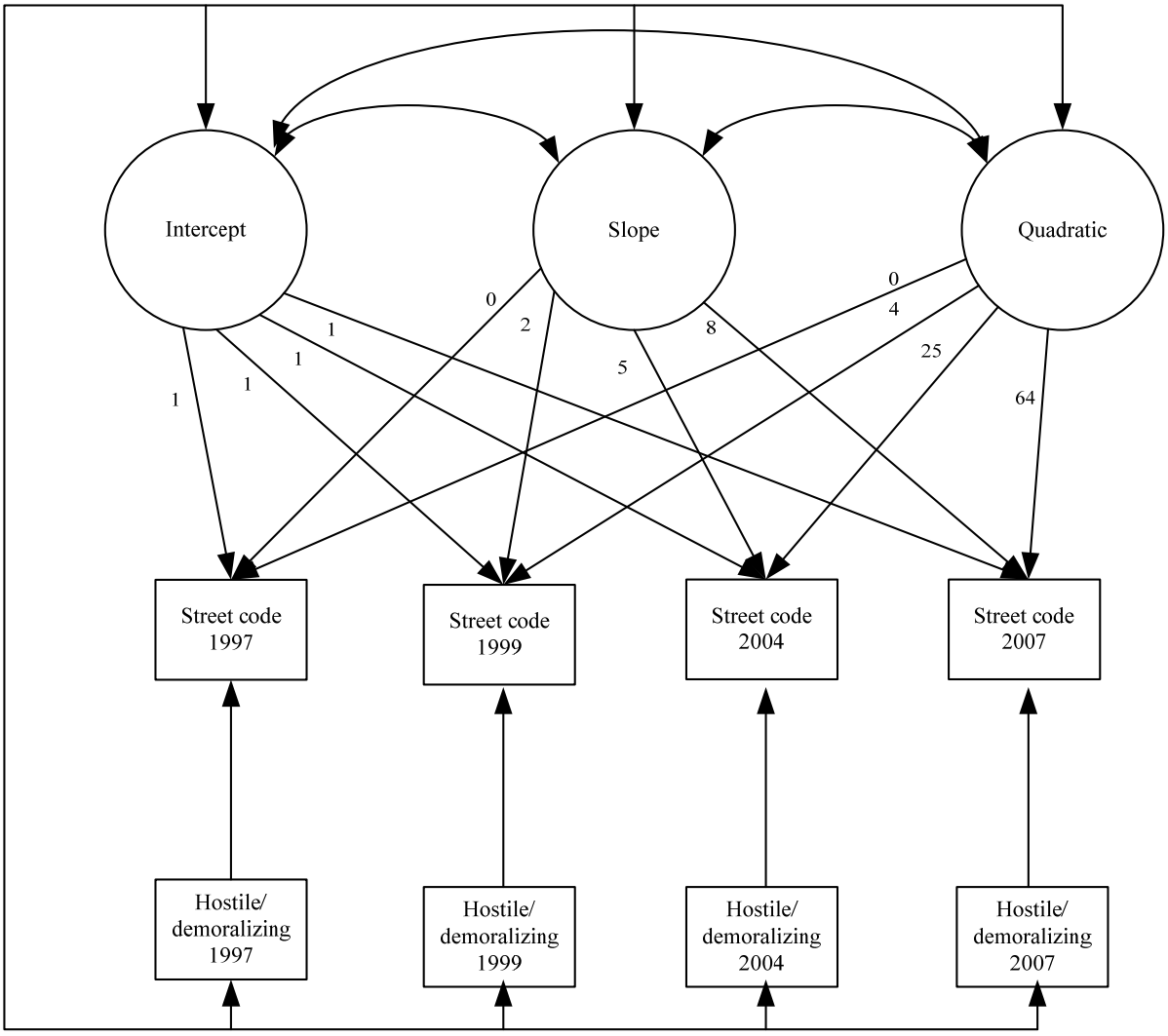

Figure 4.2.
The Latent Growth Model with Time-Varying Covariates.

RESULTS FOR STUDY 3

Initial Findings

Adopting the street code was assessed five times, around the ages of 10 to 11, 12 to 13, 14 to 15, 17 to 18, and 20 to 21 years. The average scores were 12.913 (*SD* = 2.784) at Time 1, 12.932 (*SD* = 2.661) at Time 2, 13.182 (*SD* = 2.741) at Time 3, 12.939 (*SD* = 2.531) at Time 4, and 12.646 (*SD* = 3.312) at Time 5, showing a non-linear growth pattern over the five time points. On the other hand, as shown in Table 4.1, the zero order correlations among the study variables at each time point indicate that the commitment to the street code is significantly associated with racial discrimination (a range of r = .13 to .27, $p < .01$), deviant friends (a range of r = .14 to .28, $p < .01$), harsh parenting (a range of r = .10 to .17, $p < .01$), neighborhood crime (a range of r = .13 to .27, $p < .01$), and violent behavior (a range of r = .10 to .18, $p < .01$). Violent behavior is also related to racial discrimination (a range of r = .13 to .21, $p < .01$), deviant friends (a range of r = .14 to .35, $p < .01$), harsh parenting (a range of r = .10 to .21, $p < .01$), and neighborhood crime (a range of r = .09 to .25, $p < .01$).

As expected, all variables of the hostile/demoralizing environments are related to one another in positive directions (a range of r = .17 to .38, $p < .01$). Using confirmatory factor analysis, all factor loadings are significant and in the expected direction, $\lambda > .50$.

Table 4.1.
Correlation Matrix for the Study Variables of Study 3.

	Street code adoption				
	W1	W2	W3	W4	W5
Racial discrimination W1	.272**	.199**	.116**	.049	.137**
Racial discrimination W2	.156**	.198**	.084*	.082*	.070†
Racial discrimination W3	.118**	.088*	.128**	.027	.057
Racial discrimination W4	.123**	.135**	.084*	.219**	.063
Racial discrimination W5	.138**	.182**	.162**	.103**	.195**
Deviant friends W1	.144**	.097**	.068†	.139**	.123**
Deviant friends W2	.158**	.213**	.164**	.159**	.116**
Deviant friends W3	.106**	.115**	.176**	.139**	.146**
Deviant friends W4	.122**	.158**	.108**	.280**	.004**
Deviant friends W5	.048	.121**	.077†	.075†	.174**
Harsh parenting W1	.144**	.050	-.006	.053	.020
Harsh parenting W2	.067†	.165**	.072†	.087*	.123**
Harsh parenting W3	.057	.083†	.148**	.084*	.024
Harsh parenting W4	.022	.093*	.091*	.195**	.092*
Harsh parenting W5	.014	.079†	.043	.123**	.173**
Neighborhood crime W1	.132**	.049	.090*	.059	.093*
Neighborhood crime W2	.081*	.131**	.121**	.089*	.130**
Neighborhood crime W3	.079*	.037	.106**	.160	.127**
Neighborhood crime W4	.026	.032	.042	.147**	.095*
Neighborhood crime W5	-.015	.117**	.150**	.110**	.167**
Violent behavior W1	.098**	.093*	.097**	.137**	.018
Violent behavior W2	.149**	.153**	.101**	.103**	.088*
Violent behavior W3	.058	.072†	.091*	.135**	.082*
Violent behavior W4	.090*	.089*	.063	.144**	.080*
Violent behavior W5	.033	.102*	.160**	.081*	.181**

Note. W1 = Wave 1; W2 = Wave 2; W3 = Wave3; W4 = Wave4; W5 = Wave 5.

† $p \leq .10$; * $p \leq .05$; ** $p \leq .01$ (two-tailed tests)

Unconditional Latent Growth Modeling of Adopting the Street Code

The unconditional LGM for adopting the street code is shown in Table 4.2. Because individually-varying times of measurement are specified, Satorra-Bentler scales chi-square difference testing is used to determine whether there is a linear or nonlinear growth trajectory. The result shows that the chi-square difference between a linear and non-linear model is statistical significance (Δ chi-square = 16.70, $df = 4$, $p < .01$), suggesting that the non-linear growth model provides a better fit with the data.

Table 4.2.
Summary of Model Fit Indexes and Parameter Estimates for Unconditional Linear Growth Models (N = 889)

	Intercept		**Slope**		**Quadratic**		**Chi-square difference testing**		
	Mean	*Var.*	*Mean*	*Var.*	*Mean*	*Var.*	$\Delta\chi^2$	*df*	*p-value*
Linear model	13.019 **	2.453 **	-.012	.035 **					
Non-linear model	12.637 **	2.762 *	.155 **	.074	-.012 **	.000	16.70	4	.002

Note: Chi-square difference testing using the log-likelihood (http://www.statmodel.com/chidiff.shtml).
**$p \leq .01$; *$p \leq .05$ (two-tailed tests)

This model is presented in Figure 4.3. The average value of adopting the street code at age 10 is 12.637, which is a statistically significant difference from zero. Both the slope and the quadratic growth parameters are also significantly different from zero, indicating that the average rate of adopting the street code are estimated to rise with age, 10 to 15.46 [peaking at age = 9 + .155/(2×.012) = 15.46], and then, to fall from ages 15.46 to 21. On the other hand, the analysis shows non-significant variations in the slope and the quadratic term, implying no individual differences in the trajectories of the street code. As expected, the results support that the levels of commitment to the street code can change over time within individuals.

Figure 4.3.
Unconditional Latent Growth Non-Linear Model of Adopting the Street Code.

Gender Difference in Adopting the Street Code

The model comparison procedure is used to test the statistical significance of growth parameters between the LGM for males versus females. This approach simultaneously includes models for males and females and tests for differences in the chi-square between models that constrain relationships between the parameters to be identical for the two groups versus models that free them to differ. The results are shown in Table 4.3, indicating that there are no gender differences in the intercept ($\Delta\chi^2 = 2.113$, $df = 1$, $p = .146$), slope ($\Delta\chi^2 = 1.377$, $df = 1$, $p = .241$), and quadratic terms ($\Delta\chi^2 = 2.806$, $df = 1$, $p = .094$) of the LGM equation. In other words, the initial status and developmental trajectory of adopting the street code do not differ substantially between males and females.

Table 4.3.
Comparison of the Coefficients for Males and Females

		Coefficients		Chi-square difference testing		
	Model	*Males*	*Females*	$\Delta\chi^2$	*df*	*p-value*
Intercept	constrained coefficient for males and females to be equal	12.650 **	12.650 **	2.113	1	.146
	coefficient for males and females free to differ	12.850 **	12.476 **			
Slope	constrained coefficient for males and females to be equal	.157 **	.157 **	1.377	1	.241
	coefficient for males and females free to differ	.099	.199 **			
Quadratic	constrained coefficient for males and females to be equal	-.013 **	-.013 **	2.806	1	.094
	coefficient for males and females free to differ	-.007	-.017 **			

Note: Chi-square difference testing using the log-likelihood (http://www.statmodel.com/chidiff.shtml).
**$p \leq .01$; *$p \leq .05$ (two-tailed tests)

The Latent Growth Model with Time-Varying Covariates

Previous studies have indicated that racial discrimination, deviant friends, harsh parenting, and neighborhood crime may be important predictors of commitment to the street code (Simons et al., 2012). Table 4.4 presents a non-linear growth model with time-varying covariates controlling for gender, family poverty, single family status, neighborhood disadvantage, and racial composition. The results show that racial discrimination, deviant friends, harsh parenting, and neighborhood crime are associated with commitment to the street code at ages 10 to 21. More importantly, after these time-varying covariates are included in the latent growth model, the slope and quadratic terms of the street code are no longer significant. The results suggest that the dynamic pattern of the adoption of the street code is explained by racial discrimination, deviant friends, harsh parenting, and neighborhood crime.

Based upon these findings, I treated these four variables — racial discrimination, deviant friends, harsh parenting, and neighborhood crime — as indictors of the latent construct hostile/demoralizing environments. Factor loadings for this latent construct showed good construct validity, and fit indices indicated that the model fits the data well. All factor loading were significant and in the expected direction, $\lambda > .5$. As expected, a comprehensive measure of hostile/demoralizing environments is associated with adopting the street code at ages 10 to 21. In addition, both slope and quadratic coefficient were not significant when a time-varying variable of the street code was added.

As a next step, I ran the models separately for males and females. These findings were a virtually identical to those obtained in Table 4.4. In other words, change in street code adoption over time can be explained by the pattern of change in socially demoralizing environments over time, as characterized by dimensions of racial discrimination, deviant friends, harsh parenting, and neighborhood crime. This is true for both males and females

Table 4.4.
Summary of Model Fit Indexes and Parameter Estimates for Non-Linear Growth Models with Time-Varying Covariates (N = 889)

						I	S	Q			
	Street code					Mean	Mean	Mean	Model fit		
With time-varying	W1	W2	W3	W4	W5	(Variance)	(Variance)	(Variance)	Log-likelihood	AIC	BIC
Raical discrimination	.090 **	.074 **	.072 **	.068 **	.079 **	10.802 ** (2.148)	.017 (.063)	-.005 (.000)	-21166.63	42441.25	42699.92
Deviant friends	.139 **	.128 **	.128 **	.125 **	.115 **	11.023 ** (2.825)	-.108 (.082)	.001 (.000)	-19137.96	38383.93	38624.59
Harsh parenting	.262 **	.220 **	.228 **	.223 **	.237 **	11.288 ** (2.576)	-.015 (.033)	-.003 (.000)	-16417.52	32943.03	33201.70
Neighborhood crime	.242 **	.188 **	.216 **	.212 **	.255 **	11.856 ** (2.721)	.010 (.084)	-.005 (.000)	-15263.73	30635.45	30894.12
Hostile/demoralizing environments	3.716 **	3.467 **	3.296 **	3.253 **	3.410 **	9.170 ** (2.487)	-.117 (.097)	-.001 (.000)	-46382.372	92918.54	93287.38

Note: Gender, family poverty, family structure, neighborhood disadvantage, and racial composition are controlled in these analyses.
**$p \leq .01$; *$p \leq .05$ (two-tailed tests)

Two Parallel Growth Models

The result of the multiple indicator latent growth analysis of hostile/demoralizing environments is very similar to the non-linear trajectory of the street code. Both the slope (mean = .030, $p < .001$) and the quadratic growth (mean = -.003, $p < .001$) parameters are significantly different from zero, suggesting a rise in the rate of hostile/demoralizing environments between 10 and 14 [peaking at age = 9 + .030/(2×.003) = 14] and, then, a fall between ages 14 and 21. Next, I estimated two parallel growth models with individually-varying times of observation to determine whether changes in hostile/demoralizing environments are associated with changes in commitment to the street code.

As shown in Figure 4.4, the initial level of hostile/demoralizing environments is significantly associated with the intercept of adopting the street code (b = 2.602, $p < .001$). Both the slope and quadratic parameters of hostile/demoralizing environments are also positively related to the slope (b = 1.549, $p < .001$) and quadratic (b = 1.538, $p < .001$) factors of commitment to the street code. Consistent with the results of the latent growth model with time-varying covariates, these findings provide further evidence that change in the adoption of the street code over time can be explained by the pattern of change in socially demoralizing environments over time.

The Mediating Effect of Adopting the Street Code

Finally, SEM was used to examine the extent to which changes in commitment to the street code mediate the effect of changes in hostile/demoralizing environments on violent behavior. As shown in Figure 4.5, the fit indexes show a relatively good fit for the mediation model (Chi-square = 20.177, $df = 12$, $p = .064$; RMSEA=.028). Controlling for gender, family

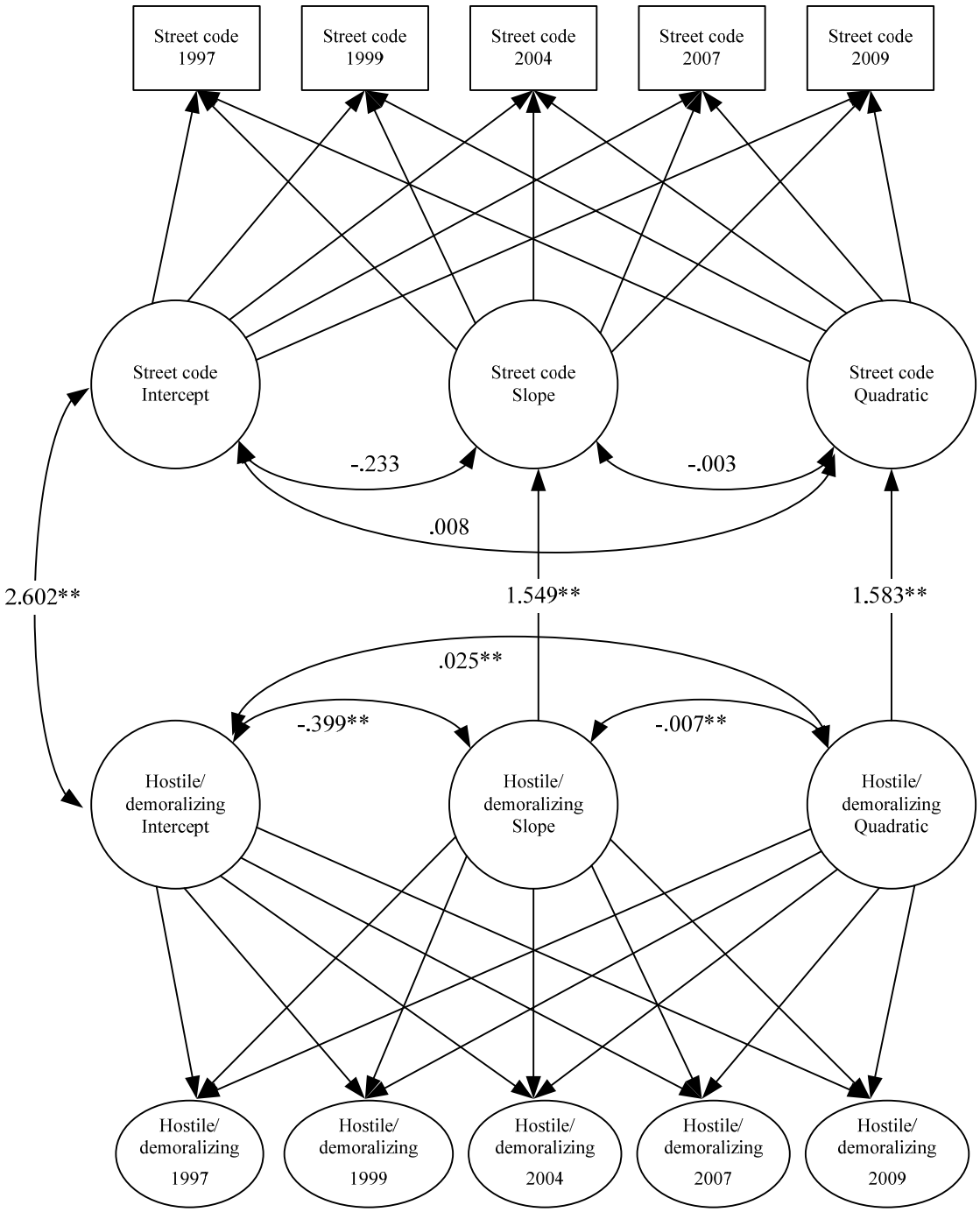

Figure 4.4.
Two Parallel Latent Growth Model Showing the Association between Hostile/Demoralizing Environments and Adopting the Street Code.

Note: Individually-varying times of observation are specified; the measure of hostile/demoralizing is a multiple indicator growth model. **$p \leq .01$; *$p \leq .05$ (two-tailed tests). $N = 889$.

poverty, single family status, neighborhood disadvantage, and racial composition, the growth rate of hostile/demoralizing environments is related to the growth rate of adopting the street code (β = .103, p = .002), which, in turn, is related to violence (β = .086, p = .020). Using the bootstrap methods with 1000 replications (Preacher & Hayes, 2008), the indirect effect was significant (indirect effect = .064, 95% CI [.012, .160], p <.05), whereas the direct effect was not (direct effect = -.191, 95% CI [-.765, .357], p = .507). Ten percent of the variance in violence explained by socially demoralizing environments is accounted for by adopting the street code.

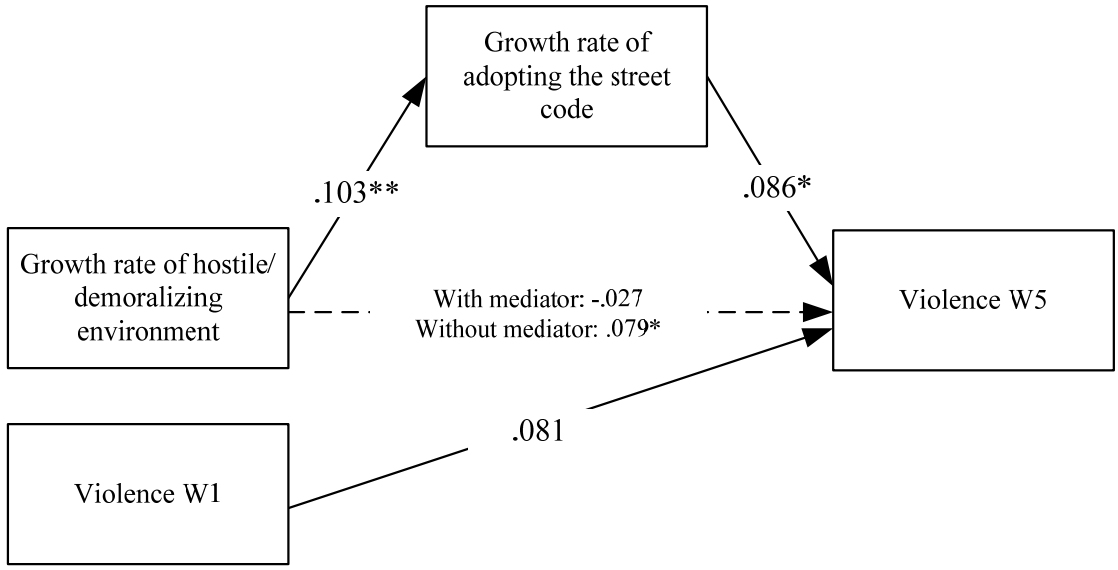

Figure 4.5.
Adopting the Street Code as Mediator of the Effect of Change in Hostile/Demoralizing Environments on Violent Behavior.
Note: χ^2 = 20.177, *df* = 12, p = .064. RMSEA = .028. Values are standardized parameter estimate. Using bootstrap methods with 1,000 replications, bold lines indicate that the test of the indirect effect of the interaction term is significant (indirect effect = .064, 25.10% portion of the total variance, 95% bias-corrected confidence intervals [.012, .160). Gender, family poverty, family structure, neighborhood disadvantage, and racial composition are controlled in these analyses. N = 889.
**$p \leq .01$; *$p \leq .05$ (two-tailed tests).

DISCUSSION FOR STUDY 3

The mediating relationship of adopting the street code between adverse social environments and violence have been shown previously (Simons et al., 2012; Stewart & Simons, 2006), supporting the idea that the "street" code is a cognitive schema that offers a guide for interpersonal public behavior. Although Anderson does not directly state that adopting the street code changes over time, he (2011, p.189) emphasizes that "most people code switch from time to time, depending on how they read a particular situation." In addition, several neurobiological scholars (D'Andrade, 1995; Davidson & Begley, 2012; Garro, 2000) have identified code values as cognitive schemas, which are changeable over the life span. Unfortunately, previous studies focused only on the stabile nature of the street code. Therefore, an unresolved question in the literature is whether commitment to the street code changes over time and if so why. I extend this line of research by investigating the dynamic nature of the street code.

Taken together, the results support my hypotheses. First, the results show that street code adoption changes over time, showing a steady increase from age 10 to 15 years and, then, a drop from age 15 to 21 years. This bell-shaped trajectory of the street code is consistent with the age-graded trajectory of crime/deviance. In addition, these findings are consistent with Anderson's code-switching hypothesis and neurologists' emphasis on brain plasticity, which maintain that street code adoption as a cognitive schema is more likely to change over time rather than remain stable. Second, consistent with previous studies (Brunson & Stewart, 2006), the current findings reveal that females as well as males living in socially demoralizing environments claim to use the street code as a means of maintaining respectable identities and reputations. I found is no gender difference in the trajectory patterns of street code adoption. Third, socially demoralizing environments, as characterized by dimensions of racial

discrimination, deviant friends, harsh parenting, and neighborhood crime, are related to changes in street code adherence from adolescent to young adulthood. Therefore, street code adoption appears to change in response to changes in exposure to adverse social environments.

The results also confirm that the growth pattern of commitment to the street code mediates the impact of the growth pattern of socially demoralizing environments on changes in violent behavior. Therefore, the findings provide evidence that changes in exposure to demoralizing environmental conditions fosters changes in commitment to the street code, with level of street code commitment, in turn, influencing level of violence. This pattern of findings provides a tentative understanding of the initiation, persistence, and decline in street code commitment over time.

Strengths of this study include the use of longitudinal data and latent growth modeling with time-varying covariates. However, several limitations of this study should be mentioned. First, the measure of concentrated disadvantage is measured by census data. It is relatively stable over time, because few cases relocate every year. Thus, the current study only uses neighborhood concentrated disadvantage as a control variable and uses perceived neighborhood crime to predict street code adoption. Future research should investigate whether there is a "moving" effect such that residential relocations operate as a series of turning points that may relate to the code switching. Second, the main purpose of the current study was to examine the dynamic role of the street code and in doing so I assumed that all individuals have the same pattern of development. Future studies should consider identifying subpopulations with latent trajectories of street code adoption across time using growth mixture modeling. Finally, the theoretical models tested in the present study utilized an African- American sample. Use of such a sample is consistent with Anderson's arguments that African Americans are more likely than many other

ethnic groups to experience the adverse social environments that give rise to the street code. But, future studies need to examine whether the same pattern of findings is evident among other racial/ethnic groups.

Despite these limitations, the current study extends the street code literature and indicates that street code adoption can change over time within individuals, and that these changes in street code commitment mediate the effect of socially hostile/demoralizing environments on violent crime. These findings might be seen as somewhat encouraging in regard to the development of intervention and prevention program designed to reduce commitment to the street code and involvement violent behavior. They suggest that programs and policies that reduce poverty, racial discrimination, community crime, and poor parenting are likely to decrease adoption of the street code and in turn violence. Given the present political climate, however, it seems unlikely that the federal, state or local governments will be willing to invest in the serious pursuit of such goals.

In conclusion, while classical subculture theory (Wolfgang & Ferracuti, 1967) tends to assume that subcultural values and beliefs are relatively stable, the current findings support the hypotheses that street code adoption is a dynamic process rather than a fixed and stable trait or schema. Furthermore, changes in street code adoption mediate the effects of social hostile/demoralizing environments on violent behavior. Understanding the dynamic nature of the street code that can mediate the association between adverse social environments and violence is important for providing insight into the mechanism by which social environments affect violence and crime.

CHAPTER 5

CONCLUSION AND DISCUSSION

"The city is not a spatial entity with sociological consequences, but a sociological entity that is formed spatially" (Georg Simmel, [1903]1997, p. 143)

Since the Chicago School's tradition of community ecology (e.g., Parker & Burgress, 1924; Shaw & McKay, 1942 [1969]), sociologists and criminologists have sought to understand how neighborhood contexts affect individuals. Shaw and McKay's social disorganization model is perhaps the most recognized theory in this literature. They argued that neighborhoods are an important context in which people live and interact. They contend that different types of neighborhood structures contribute to different levels of social disorganization because neighborhood structures are highly correlated with the core social institutions of society such as family stability, personal relations, and social norms. Thus, rapid urbanization and industrialization cause social disorganization by disrupting the control systems, which in turn leads to higher crime rates.

During the 1960s and 1980s, classical social disorganization theory was criticized for its lack of an operational definition of disorganization and the mechanisms that link neighborhoods and crime (Bursik, 1988). First of all, classical social disorganization theory has ignored the formal and informal relational networks of association that pertain to the public sphere of social control and shape neighborhood residents' activities. Second, while Shaw and McKay ([1942]

1969) proposed the cultural transmission approach to explain how delinquent values are transmitted across generations in disadvantaged neighborhoods, traditional neighborhood scholars (e.g., Kornhauser, 1978) deemphasized the importance of the cultural assumption in social disorganization models and instead focused on the control assumptions of structural disorganization. Third, feminist scholars (Belknap, 2007; Zahn & Browne, 2009) have argued that classical neighborhood studies were presumed to be gender neutral, focused disproportionately on males, ignored women's experience, and simply used gender as a control variable.

In the past two decades, sociologists and criminologists have shown a renewed interest in studying neighborhood effects. For example, Robert Bursik and Harold Grasmick (1993) propose the systemic model of crime that extends the classical social disorganization model by including networks of social relations. They assume neighborhood relational networks are a means of social control that links neighborhood structure with crime/deviance. Furthermore, Robert Sampson and his colleagues (1997) introduced the concept of collective efficacy. They explained neighborhoods' informal social control through social cohesion and shared expectations. In neighborhoods with high collective efficacy, residents are more likely to trust their neighbors and are willing to intervene to help their neighbors to reach collective goals. In addition to the control assumption of structural disorganization, Elijah Anderson's street code thesis (1999) linked classical sub-cultural theories into a classical social disorganization model and indicated that residents in disadvantaged neighborhoods are more likely to adopt street code values and to employ violence as a means of showing others that they are strong and powerful.

On the other hand, feminist criminologists (Belknap, 2007; Chesney-Lind & Pasko, 2013; Cobbina, Miller, & Brunson, 2008; Miller & White, 2006;) have claimed that neighborhood

context is a gender-stratified environment. Women and men have unique experiences in their neighborhood contexts which influence gendered behavior. For example, several studies have reported that fear of sexual violence or crime is a powerful mechanism of social control for girls living in disadvantaged neighborhoods (Campbell, 2005; Cobbina, Miller, & Brunson, 2008). Thus, feminist scholars argued that classical neighborhood models constructed by men and for men may not be generalizable to women.

While past studies have yielded valuable contributions, they have often left an unanswered and important question: Why is there so much heterogeneity in the well-being of individuals residing in neighborhoods? For instance, Mayer and Jencks' article published in *Science* in 1989 indicated that not all people living in disadvantaged neighborhoods are violent /deviant and contended that neighborhood effects are small in magnitude because such effects are always confounded with individual characteristics, socioeconomic status and race. Recently, Chen and Miller (2012) also found that some people who grow up in disadvantaged neighborhoods maintain a low risk for chronic disease and illness over the long term.

Furthermore, while a lot of research in the neighborhood studies tradition has focused on crime/deviance, medical sociologists and public health scientists have provided theoretical frameworks to link neighborhood contexts and health-related issues. Unfortunately, substantial work in the past decade on physical health was done using self-reports, and these results were mixed. On the one hand, some studies (e.g., Aneshensel & Sucoff, 1996; Cohen et al., 2003; Diez-Roux et al., 2001; Franzini et al., 2005; Holmes & Marcelli, 2012; Lee & Cubbin, 2003; Morenoff, 2003; Robert, 1998, 1999; Ross & Mirowsky, 2001) found that neighborhood factors influence one's physical health, even after controlling for individual socioeconomic status. On the other hand, several studies have indicated that the effects of neighborhood context on health

outcomes were no longer significant after controlling for individual SES (Bowling & Stafford, 2007; Gallo et al., 2012; Petersen et al., 2008). Given these mixed findings, whether living in disadvantaged neighborhoods has a unique effect on health outcomes is unknown.

Finally, while Anderson's street code thesis has been tested by means of quantitative (e.g., Baumer, Horney, Felson, & Lauritsen, 2003; Stewart & Simons, 2010) and qualitative (e.g., Brookman, Copes, & Hochstetler, 2011; Jimerson & Oware, 2006) methods, no studies to date have examined whether street code adoption can change over time. Further, if street code adoption is dynamic, the mechanisms inherent to these changes are also unknown.

In summary, the link between neighborhood contexts and individual well-being is robust (e.g., Browning, Cagney, & Iveniuk, 2012; Hill, Ross, & Angel, 2005; Sampson, Rausdenbush, & Earls, 1997). According to these lines of research, individuals engage in crime/deviance and have poor physical health depending upon where they live. However, little is known about how individual characteristics affect the way in which people respond to neighborhood conditions and how these individual differences impact on deviance and health. This dissertation, using panel data from the Family and Community Health Study (FACHS), addressed many of the limitations of past studies by examining four important questions that have received little attention in the neighborhood literature. First, can findings of prior neighborhood studies be replicated using a sample of adult African American females and does gender matter in street code adoption? Second, why is there so much heterogeneity in the behavior of individuals residing in the same neighborhood? Third, how do neighborhood effects influence physical health? And, fourth, does street code change over time and if so why? The main contributions and limitations of the current study are summarized and discussed below.

WOMEN AND NEIGHBORHOOD CONTEXT

The generalizability problem of traditional criminological theories is an important issue in feminist criminology (Daly & Chesney-Lind, 1988). Research on feminist studies have argued that the classical social disorganization model has been tested using male-only samples and was developed by male criminologists to explain crimes committed by males (Belknap, 2007; Chesney-Lind & Pasko, 2013). Nevertheless, male-centered theory is presumed to explain both female and male offending and to promulgate the belief that crime is gender-neutral. Therefore, feminist criminology attempts to answer whether traditional criminological theories like social disorganization theory apply to women as well as men.

Using a sample of adult women, the results of my first and second studies indicated that neighborhood characteristics are significantly associated with increased involvement in antisocial behavior and the risk of adverse health outcomes. Furthermore, given that neighborhood processes link neighborhood structure and crime/deviance, Study 1 examined the extent to which the relationship between neighborhood disadvantage and antisocial behavior can be explained by neighborhood social ties. Consonant with the findings from prior research, neighborhood social ties is a mediator of concentrated disadvantage on antisocial behavior. These findings suggest that social disorganization models can also help explain the antisocial behavior and physical health of adult women.

Although these results replicate the existing neighborhood literature, the adults in the sample were selected because of their status as primary caregivers. Predictably, all of them were women. The lack of men in the sample barred us from being able to assess the extent to which the results might differ by gender. Future studies should replicate the results with samples that include both women and men and examine whether neighborhood effects vary by gender.

Moreover, based on the traditional criminological literature, neighborhood characteristics in the current study were measured by an absolute socio-economic index, such as the percentage of residents in a census-tract that were living below the federal poverty threshold. It should be noted that some studies have found that levels of *relative* inequality within neighborhoods could effectively predict gender differences in criminal behavior. Lei and his colleagues (2014), for example, found that gender differences in violent behavior are wide within gender-inegalitarian neighborhoods, whereas these differences decrease within gender-egalitarian neighborhoods. This occurs because boys' rates of violence decrease while girls' rates remain relatively low across neighborhoods. Additionally, the relationship between neighborhood gender equality and violence is mediated by expressions of toughness, which supports the importance of hegemonic masculinity in criminological studies. Thus, they concluded that girls and boys have different experiences in their neighborhood contexts due to gender equality levels. Future studies should pay more attention to the relationship between neighborhood gender equality as a relative index and individual well-being.

On the other hand, previous research has shown that women adopt less hostile orientations than men (Messerschmidt, 1993; Schrock & Schwalbe, 2009). Anderson (1994, pp. 91-92) also indicated that adoption of street code is predominately a male-oriented practice. Unfortunately, the results of these studies are mixed. Some studies have found that women as well as men living in disadvantaged neighborhoods adopt street code values (Brunson & Stewart, 2006; Jones, 2010; Ness, 2004), whereas other studies (Nowacki, 2012; Taylor, Esbensen, Brick, & Freng, 2010), have found gender differences. According to the findings of Study 3, there is no gender difference in the trajectory patterns of street code adoption. In addition, I found that adopting the street code can be explained by neighborhood crime and socially demoralizing

environments for both males and females. It should be noted that, although this study did not find gender differences in dynamic patterns of street code adoption, future studies should consider identifying latent trajectories of street code adoption across time and examine whether gender differences exist among various types of trajectories.

GENOTYPES AND NEIGHBORHOOD CONTEXT

Most neighborhood studies have assumed that a disadvantaged neighborhood structure is a relatively stable and external object, and its effect influences people in similar ways. Thus, past studies tend to focus on the effect of neighborhoods per se. For example, Sampson (2012, p. 356) emphasizes that "what is truly American is not so much the individual but neighborhood inequality." Regardless of individual differences and variation, a neighborhood is a fundamental context explaining crime/deviance and different aspects of well-being. It is interesting to note, however, that about only five percent of the variance in individual behaviors can be explained by neighborhood context (Leventhal & Brooks-Gunn, 2000). All of this unexplained variance may be because of individual differences in their neighborhood experiences (Chen & Miller, 2012; Mayer & Jencks, 1989).

In the past decade, advances in biosocial studies (e.g., Caspi et al., 2003; Guo, Roettger, & Cai, 2008; Simons et al., 2011) have provided compelling reasons to examine the ways in which social factors and genotypes combine to influence well-being across the life course. Not surprisingly, research on neighborhood effects has fit into this emerging paradigm, because it can answer the question of why some individuals are genetically more susceptible to neighborhood influences than others. Unfortunately, many studies have indicated that crime/antisocial behavior is affected by neighborhood context, but few studies have investigated whether these effects vary

by genetic variations. The aim of the first study was to investigate whether plasticity genes modify the associations between neighborhood factors and antisocial behavior.

Consistent with prior molecular studies (Caspi et al., 2003; Moffitt, Caspi, & Rutter, 2006), there was no genetic main effect of the serotonin transporter gene on the antisocial behavior of adult women. As hypothesized, this gene moderated the association of both concentrated disadvantage and social ties on increases in antisocial behavior. In addition, my findings confirm previous research (Bursik & Grasmick, 1993; Sampson, Raudenbush, & Earls, 1997) suggesting that neighborhood social ties are mediators that explain the effect of concentrated disadvantage on criminal or antisocial behavior. Given these findings, I ran hierarchical multilevel regression models and found that the interaction between genes and concentrated disadvantage were spuriously related to antisocial behavior; controlling for the gene-neighborhood social ties interaction. In other words, as expected, the moderating effect of genetic variation on the relationship between concentrated disadvantage and adult women's antisocial behavior was explained by the interaction of genetic variation with social ties. These findings suggest that variation in the serotonin transporter gene is an individual difference that accounts, at least in part, for dissimilarities in the way that people respond to neighborhood influences.

Furthermore, there are two perspectives that explain gene-environment interactions (Belsky, Bakermans-Kranenburg, & von IJzendoorn, 2007; Belsky & Pluess, 2009). The diathesis-stress perspective asserts that individuals with the genetic risk alleles are more vulnerable to negative environmental influences than those without these risk alleles, whereas the differential susceptibility perspective assumes that individuals carrying specific genotypes differ in their susceptibility to environmental influence in a 'for better and for worse' manner.

However, studies have indicated that the statistical power to test the differential susceptibility is often limited by range restrictions in environmental measurements (Dick, 2011; Duncan & Keller, 2011). Given the disproportionately high percentage of African Americans who live in disadvantaged neighborhoods (McNulty, 2001; McNulty & Bellair, 2003; Peterson & Krivo, 2010; Sampson, Morenoff, & Raudenbush, 2005), I used Widaman and collogues' (2012) method to determine whether the diathesis-stress model or the differential susceptibility model of gene-environment interaction best explains the moderating effect of the serotonin transporter gene on neighborhood influences.

The results of Study 1 supported the differential susceptibility perspective and indicated that women with the S-allele of *5-HTTLPR* showed poorer adjustment than other genotypes when the neighborhood environment was adverse but better adjustment than other genotypes when the neighborhood environment was favorable. In other words, this dissertation provided evidence that individuals who carry specific genotypes are not only most vulnerable to adverse neighborhood environments but also benefit most from favorable neighborhood environments.

Methodologically, differentiating between the diathesis-stress and differential susceptibility models requires data representing the full range of values on the environmental variable of interest, from adverse to favorable. Such a range is necessary to determine whether there is the cross-over effect predicted by the differential susceptibility model where individuals with the focal allele show poorer adjustment than other genotypes when the environment is adverse but better adjustment than other genotypes when the environment is favorable. Using traditional post-hoc tests of interaction effects, I was able to determine from my analysis that individuals with the S-allele show higher rates of antisocial behavior than other genotypes when the neighborhood environment is adverse. Therefore, this study provided evidence that the

overrepresentation of African American families in disadvantaged neighborhoods found in this sample is consonant with that reported in several other studies (e.g., McNulty, 2001; McNulty & Bellair, 2003; Peterson & Krivo, 2010; Sampson, Morenoff, & Raudenbush, 2005).

In summary, the results of this dissertation strongly support that individual well-being is "always dependent on who is in what setting" (Wikström, 2004, p. 19). Therefore, my study is important, because the results demonstrate that individual well-being is determined by a combination of neighborhood context and individual characteristics. Based on my findings, future studies should focus on the moderating roles of neighborhood and individual characteristics on well-being. Whether the effects of neighborhood disadvantage on individual well-being are moderated by individual factors such as parenting, affiliation with deviant peers, low self-control, school engagement, and religiosity remains a central question.

Future studies will also need to seek to replicate these findings using other plasticity genes, including the *DRD4*, *DRD2*, *GABRA1*, *GABRG2*, and *OXTR* genes. For example, Belsky and his colleagues (Belsky & Pluess, 2009) assumed that the more plasticity alleles one carries, the more susceptible one will be to environmental influence. Furthermore, Simons et al. (2011) revealed that individuals with multiple plasticity alleles scored higher than others on violent behavior when the neighborhood environment was adverse, whereas persons with this genotype scored lower than others on violent behavior when the neighborhood environment was favorable. Future studies will need to incorporate a measure of cumulative genetic plasticity to examine gene-environment interactions. Finally, my findings provide substantial evidence that genetic effects do not occur in a vacuum but are conditioned by their neighborhood and social contexts. Therefore, individual well-beings are not determined by either genetics or environmental variations alone. Without knowledge of the gene-environment relationship, the importance of

genetic effects and neighborhood effects in the lives of individuals might go unrecognized. As noted recently by David Farrington (2012, p. XX), future research should focus on "longitudinal studies to measure biological and neighborhood/community influences on offending" and well-being.

HEALTH AND NEIGHBORHOOD CONTEXT

There is a long history of research that explains the link between neighborhood context and health (Aneshensel & Sucoff, 1996; Browning & Cagney, 2003; Cohen et al., 2003; Diez-Roux et al., 2001; Ross & Mirowsky, 2001; Turner, 2010). However, studies have indicated that social disorganization theory does not explain health outcomes because the relationship between a lack of informal social control and health has been hard to establish. So, what accounts for the link between neighborhood context and physical health? In the medical sociological tradition, Leonard Pearlin (1989) proposed the stress process model and claimed that neighborhood context, as a constellation of stressors, impact physical and mental health because there is an unequal distribution of resources and opportunities across neighborhoods. For example, residing in disordered neighborhoods is stressful, and relates to reduced occupational and educational opportunities, housing instability, high crime rates, and employment-related stress for adults. Therefore, a growing number of studies have indicated that neighborhood disorder, defined as neighborhoods that present signs of physical and social deterioration, is a stressful condition that adversely influences physical health (Diez-Roux, 2001; Hill, Ross, & Angel, 2005; Ross & Mirowsky, 2001).

Despite evidence that people residing in disordered neighborhoods have a higher risk of chronic disease and mortality rates, some scholars (Bowling & Stafford, 2007; Gallo et al., 2012;

Petersen et al., 2008) have argued that the relationship between neighborhood context and health might be spurious because such a relationship may be confounded by individual socioeconomic status and health-related life styles. In addition, evidence for neighborhood effects on physical health mainly relies on self-reported neighborhood disorder, distress responses, and physical health status (e.g., Browning & Cagney, 2003; Hill, Ross, & Angel, 2005; Ross & Mirowsky, 2009). Methodologically, self-reported measures are biased by projection and "common method variance" effects (Podsakoff et al., 2003). As a result, the neighborhood-health relationship may be inflated by self-reported measures. Indeed, it is unclear whether neighborhood disorder really affects physical health. Given these limitations, this dissertation controlled for health-related individual characteristics and used the systemic social observation of neighborhoods and inflammation biomarkers from blood samples as objective neighborhood disorder and health-related distress indicators, so as not to overestimate the effects of neighborhood on health. Theoretically, I examined the stress process mechanism that integrates a sociological approach that focuses on neighborhood structures with a neuroscience approach that emphasizes physiological and immunological systems, both of which are associated with physical health status.

As hypothesized, after controlling for neighborhood and demographic measures, the results of Study 2 confirmed that women living in disordered neighborhoods have significantly higher levels of inflammatory burden as well as self-reported poor health status. I also revealed that inflammatory burden mediated the effect of neighborhood disorder on self-reported health. Study 2 provided strong evidence for the stress process mechanism of neighborhood disorder on physical health. Residents of disordered neighborhoods are exposed to multiple environmental

stressors and are more likely to provoke a chronic inflammatory response, which in turn influence health and disease.

As I have noted, there is wide variability in how individuals respond to neighborhood contexts. Similar to Study 1, I incorporated genetic information into my theoretical models and examined why some people who live in disordered neighborhoods do not experience adverse health effects. Expanding the neighborhood-health relationship literature, the findings from Study 2 supported the idea that genetic variations are the moderating effect of neighborhood disorder on health-related inflammatory burden.

It should be noted that there are two dramatically different perspectives on the gene-environment interaction effects. Unlike Study 1, the results of Study 2 supported the diathesis-stress perspective, whereby individuals with the risk-allele of the IL-6r gene show higher rates of inflammatory burden than those without the risk-allele when the neighborhood environment is adverse. To explain the different results between Study 1 and Study 2, it is quite possible that, unlike cognitive and behavioral outcomes, health-related outcomes are more difficult to reverse even when the environment is favorable. In addition, low health index scores are not always positive for health. For example, low and high blood pressure or body mass index (BMI) are both related to poor health outcomes.

Further, research concerning family sociology has shown that married adults compared to unmarried adults are more likely to have lower rates of mortality and morbidity and to report better health (Hughes & Waite, 2009; Johnson, Backlund, Sorlie, & Loveless, 2000; Williams & Umberson, 2004). Following this line of research, medical sociologists have proposed the stress-buffering hypothesis (Cohen, 1988; Diez-Roux & Mair, 2010; Hughes & Waite 2009; Lin &

Ensel, 1989; Phelan, Link, & Tehranifar, 2010) which suggests that marriage is a source of social and emotional support which buffers the relationship between neighborhood disorder and health.

This dissertation extended this line of research by investigating how neighborhood disorder, genotype, and marriage combine to influence physical health. The results suggested that the effects of neighborhood disorder on the inflammatory stress response and health are not uniform but are most pronounced among unmarried women carrying the risk allele of the IL-6r gene. Thus, physiological response to disordered neighborhoods varies not only by genetic variations, but also by marital status.

While Study 2 implied that marital status serves as an important protection for physical health, a recent study (Donoho, Crimmins, & Seeman, 2013) has found that marital quality is also an important factor for women's inflammatory responses and physical health. In other words, the stress process mechanism may vary by gender, and it is not only marital status which is related to physical health, but also marital quality. Unfortunately, this study did not include measures of marital quality because the sample size precluded the use of an internal moderation model. Furthermore, given that the adults in the sample were selected because of their status as primary caregivers, almost all of them were women. Future studies will need to examine how the quality of marriage influences the relationship between neighborhood characteristics and physical health, and whether gender differences exist in the stress process mechanism.

In addition to the importance of marital quality, Edith Chen and Gregory Miller (2012, 2013) proposed the shift-and-persist model to examine why some people do not experience chronic disease despite ongoing experiences with severe adversity. They argue that some low-SES individuals have significant others (family members and caretakers) who teach them to trust others, to better regulate their emotions, and to focus on future goals. Therefore, these

individuals respond to adversity by reframing the events more positively and by shifting their focus to future goals. To date, few studies have investigated this approach. In this dissertation, I have provided evidence that people living in disadvantaged neighborhoods present a great deal of heterogeneity. Based on the shift-and-persist model, future studies should further address the extent to which family or peer variables buffer neighborhood effects on individual well-being.

Finally, the sample in Study 2 is limited to adult women. A number of studies have examined that childhood exposure to economic hardship would forecast physical health during young adulthood (Fagundes, Glaser, & Kiecolt-Glaser, 2012; Miller, Chen, & Parker, 2011). Future efforts to collect more detailed objective measures on health and neighborhood for youth panel samples would allow for a test of long-term effects.

VIOLENCE ORIENTATION AND NEIGHBORHOOD CONTEXT

Elijah Anderson's studies (1999, 2011) expanded the classic subcultural theories and proposed the concept of street code as one of the key neighborhood processes linking neighborhood context to crime. Recently, several studies have taken the street code theory and melded it with social disorganization theoretical frameworks (e.g. Matsuda et al., 2013; Nowacki, 2012; Oliver, 2006; Parker & Reckdenwald, 2008; Simons et al., 2012; Stewart & Simons, 2006), and have found that neighborhood structures are sets of mutually sustaining cultural schemas that empower and constrain social action and that tend to be reproduced by that social action. They found that adopting the street code as a type of cognitive schemas can be used to understand how individuals employ violence to resolve conflict while others do not, when they reside in socially demoralizing environments. Despite numerous studies examining the relationships among environment contexts, street code value, and violence, explanations of

the dynamic role of street code remain unresolved. Anderson (1999, pp. 98-106) has demonstrated that the code can change between decent and street orientations depending on the situation. Although Anderson did not mention that the street code can be changed over time, neurobiological scholars (D'Andrade, 1995; Davidson and Begley, 2012; Garro, 2000) have identified code values as cognitive schemas, which are not stable traits or fixed over time. Following this approach, Study 3 extended prior cross-sectional research by investigating the dynamic role of street code adoption among African Americans.

First, I hypothesized that street code can change over time within individuals. Using a Latent Growth Model the results of Study 3 showed that the probability of adopting the code of the street increase rapidly between the ages of 10 and 11, and then gradually decrease from ages 16 to 21. The graph of street code adherence indicated a pattern virtually identical to the age-graded trajectory of crime/violence. Thus, the findings provide evidence that, similar to criminal and violent behavior, adopting the street code is a dynamic process rather than a fixed and stable trait.

The question then is what are the mechanisms that explain the dynamic nature of street code adoption? Anderson (1999) claimed that the street code values, as informal rules, guide how people interact with one another and interpret their neighborhood environments. Thus, people who resided in disadvantaged neighborhoods are likely to adopt the street code, because these neighborhoods are likely to contain a constellation of sociodemographic risk factors such as racial discrimination and harassment, harsh parenting, affiliation with deviant peers, and criminal victimization. For instance, past studies (Simons et al., 2012; Stewart & Simons, 2006) focused on possible between-individual differences in street code adoption and indicated that

racial discrimination, affiliation with deviant friends, parenting, and neighborhood crime are related to street code adoption.

However, no studies to date have examined Anderson's street code thesis by comparing within-individual changes in socially demoralizing environments over time with within-individual changes in street code adoption over time. After controlling for neighborhood disadvantages and demographic measures, the results of Study 3 strongly support that changes in socially demoralizing environments, as characterized by dimensions of racial discrimination, deviant friends, harsh parenting, and neighborhood crime, are followed by changes in street code adoption. As a consequence, street code adoption is not fixed over time, but continuously shaped by the social environment.

Furthermore, in socially demoralizing environments, individuals who embrace the values of the street code are more likely to use violence as a means of achieving their reputation (Anderson, 1999; 2011). Stewart & Simons (2006) conducted quantitative research using African Americans samples. They found that street code adoption mediates the effects of neighborhood crime, racial discrimination, and parenting on violent behavior. Following this line of research, this dissertation extended the street code literature and addressed the mediating role of street code adoption while focus on the dynamic development of this code. I found that changes in street code adoption mediate the change effects of socially demoralizing environments on violent behavior. Taken together, street code adoption can change over time within individuals, and this code mediates the effects of socially hostile/demoralizing environments on violent crime.

Although Study 3 provided strong evidence for stability and change in street code adoption in relation to social environments, the sample was limited in examining relocation effects because this sample tended to stay in their Wave 1 neighborhoods. Therefore, the

relationship between changes in neighborhood disadvantage and adopting the street code over time is unclear. In the current study, I found that change in perceived neighborhood crime was related to change in street code adoption over time. Based on life course perspectives (Sampson & Laub, 2003), future studies will need to examine relocation effects on street code adherence and to answer whether relocation from disadvantaged to advantaged neighborhoods reduce street code adherence.

Finally, the main purpose of Study 3 was to examine the dynamic role of street code adoption and argue that all individuals have the same pattern of development. Future studies should consider identifying subpopulations with latent trajectories of street code adoption across time using growth mixture modeling and should test why some people persist or desist in street code adherence. These hypotheses might be explored in subsequent research.

COMMON LIMITATIONS

Although this dissertation offered a number of insights into the impact of neighborhood characteristics on individual well-being, including antisocial behavior, physical health, and violence, some common limitations must be noted.

First, the sample in this dissertation focused upon African-American families living in Iowa and Georgia. One of the most consistent findings in the neighborhood literature is that individuals who reside in disadvantaged neighborhoods are more likely to engage in crime/deviance and to have poor health status than those who reside in advantaged neighborhoods (Diez-Roux & Mair, 2010; Sampson, Morenoff, Gannon-Rowley, 2002), and that African Americans commit much more crime/deviance and have worse health-related outcomes than other ethnic groups (Wakefield & Uggen, 2010; The National Center for Health Statistics,

2011). As a result, the relationships between neighborhood context and well-being are especially salient for African Americans who are disproportionally likely to reside in extremely disadvantaged neighborhoods (Krivo, Peterson, and Kuhl, 2009; McNulty, 2001; Peterson and Krivo, 2010). In some ways, this dissertation was an advantage given the overrepresentation of African Americans in disadvantaged communities. But, it is clearly the case that the findings need to be replicated using more diverse samples.

Second, a neighborhood is a small physical area that has some traditional identity and personal network (Bursik & Grasmick, 1993). Traditionally, neighborhoods have been measured using census-defined areas, systematic social observation, and a self-reported survey (Diez-Roux & Mair, 2010; Sampson, 2012). This dissertation used these three methods to measure aspects of neighborhoods. It should be noted that these three methods have strengths and limitations.

In accordance with social disorganization theory, concentrated disadvantage was measured using the 2000 census data which was geocoded with participant's residential addresses. The advantages of census data are its reliability and its representativeness. Although geographic boundaries are generally measured by census tract groups, officially designated units are meaningless to some residents (Sampson, 2008; 2012). For example, rural areas might be overestimated due to their low population, and neighborhood effects might be confounded by people living close to boundaries. In addition, no information is provided regarding a wide variety of indicators of physical and social disorder including graffiti, trash, abandoned cars and buildings, and vandalism. More importantly, census data is collected every ten years. There is the time lag between the collected date of census and a survey data. It is very hard to examine the dynamic effects of neighborhood.

Study 2 attempted to examine the relationship between neighborhood disorder and health-related outcomes. As I have noted above, neighborhood disorder cannot be measured by the census data because this data do not provide detailed information about physical and social incivilities in neighborhoods (Sampson, Morenoff, & Gannon-Rowley, 2002). Thus, in Study 2, neighborhood disorder was assessed using the observers' ratings of the participants' neighborhoods. Unlike a self-reported neighborhood disorder, the systematic social observation of neighborhood as an objective measure can reduce the likelihood of "common method variance" biases (Browning et al., 2013; Podsakoff et al., 2003; Sampsons & Raudenbush, 1999). While the social observation method can overcome the limitations of census and self-reported data set, the systematic social observation of neighborhood is not available for all of the waves in the FACHS dataset because a considerable amount of time and money is required to collect the observers' data. As a consequence, both the census data and the systematic social observation cannot be used to examine the relationship between changes in neighborhood characteristics and adopting the street code over time. Therefore, Study 3 used self-reported neighborhood crime to assess the extent to which change in adoption of the street code over time can be explained by change in neighborhood crime and socially demoralizing environments.

In summary, based on data limitations and theoretical reasons, this dissertation used different methods to measure neighborhood characteristics in the three studies presented. Thus, the findings of this dissertation need to be replicated using different data set.

Third, some researchers have argued the relationship between neighborhood and well-being is confounded by a self-selection bias. Nevertheless, Sampson (2012, p.29) has claimed that "selection is not a bias but rather part and parcel of a dynamic social process — another

form of neighborhood effect." In addition, using experimental data from the MTO program[3], work by Sampson and colleagues (Sampson, Morenoff, & Raudenbush, 2005; Sampson, 2008; Sampson & Sharkey, 2008; Sharkey & Sampson, 2010) has provided strong evidence that neighborhood effects are robust even when controlling for self-selection effects. Because selection effects are nearly impossible to completely rule out in non-experimental studies, individual and family demographic variables were included as controls to reduce such effects in the current study.

CONCLUSION

Research in sociology and criminology has shown a strong link between neighborhood characteristics and individual well-being (e.g., Browning, Cagney, & Iveniuk, 2012; Martin et al., 2011; McNulty, Bellair, & Watts, 2013; Ross & Mirowsky, 2009; Sampson, Morenoff, & Raudenbush, 2005). Consistent with this line of research, findings from all of my three studies showed the significant impact of neighborhood context on individual well-being, including antisocial behavior, physical health, and violent orientation. People who live in socially and economically disadvantaged neighborhoods have a higher risk of committing criminal and violent behaviors and getting sick. As a consequence, these results inform policy makers of the need to take into account neighborhood inequality in economic and social resources because such inequality appears to substantially affect individual well-being.

Although the link between neighborhood and well-being provides useful insights, they cannot answer the question: why is there so much variation in the well-being of individuals

[3] The Moving to Opportunity (MTO) program utilized a form of Section 8 vouchers to help relocate a randomly-selected group of program applicants living in disadvantaged public housing projects in five US cities into neighborhoods in which fewer than 10% of their neighbors were peers.

residing in disadvantaged neighborhoods? Thus, I incorporated genetic effects into neighborhood influence models to answer this question.

In *The Bell Curve*, Richard Herrnstein and Charles Murray (1994) argued that intelligence quotient (IQ) can explain racial disparities in individual well-being and that IQ is substantially genetically determined. Therefore, they claimed that social policy should seek to eliminate cognitive inequalities rather than to remove environmental and neighborhood disadvantages and suggested developing remedial education programs for disadvantaged population. In contrast, results from this dissertation showed that variation in genetic polymorphism does not show a direct effect on antisocial behavior and health; rather, they exert their influence by moderating the effects of neighborhood context.

Based upon my results, social and neighborhood factors are fundamental to understanding the mechanisms of genetic factors on individual well-being. As a result, social and biological social scientists are not locked in a zero-sum game. In fact, social scientists are key experts who can help researchers in other disciplines explain heterogeneity among people with similar genetic structures, provide accurate measurement of social contexts, and interpret the meaning of research involving social factors.

In particular, distinguishing between the diathesis-stress and differential susceptibility models is important as they suggest very different intervention and policy approaches. Whereas the diathesis-stress model supports the argument that individuals with certain genetic risk-alleles have difficulty given their genetic tendency to be hyper-responsive to adversity, the differential susceptibility model supports claims that their environmental sensitivity makes them good candidates for intervention. They are more likely than those with differing genotypes to learn the lessons being taught by a new, more favorable neighborhood environment.

Furthermore, findings from this dissertation supported the argument that physiological distress and health do not take place in a vacuum, but are influenced by neighborhood and social contexts. Also, I indicated that both genetic makeup and marital status moderate the association between neighborhood context and health. Taken together, my findings indicated that neighborhood and individual characteristics combine to influence well-being. Just as Zimmerman and Messner (2012, p. 75) noted, "Persons and contexts do not exist in isolation," but are interdependent. According to my results, neighborhood intervention programs must take into account differential effectiveness with different population groups.

Finally, the current study has produced new insights into the dynamic nature and social determinants of street code adoption. Because there are significant relationships between changes in socially hostile/demoralizing environments and street code adoption over time, strategies for crime and/or violence prevention in dangerous social environments are possible. As a result, future prevention research should design neighborhood or family programs which address street code adoption. For example, neighborhood prevention programs should be considered to reduce racial discrimination in education, employment, and every day activities. Family prevention programs are also needed. These should be designed to prevent affiliation with delinquent peers and to promote some aspect of effective parenting.

In summation, while neighborhood research traditionally relies on social disorganization theory, the findings of this dissertation indicate that neighborhood contexts are link to individual well-being through complex mechanisms. Studies should consider the biosocial approach, the broken window theory, the stress process model, the street code theory, and social disorganization theory to shed light into the nature of neighborhood effects.

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APPENDIX A

RE-PARAMETERIZED MULTILEVEL POISSON MODEL.

Because the multilevel Poisson models are used in the current study, the re-parameterized equation is calculated by generalized nonlinear mixed models (PROC NLMIXED in SAS) (SAS Institute, Cary, NC). I show the syntax in the following:

```
PROC NLMIXED DATA=mydata;
 PARMS logsig 0  B0 = 0 B1 = 0  C = 0 B3 = 0 V1=0;
    eta = B0 + B1*(IDV - C) + B3*((IDV - C)*MODERATOR) + V1*CONTROL + e;
    lambda = exp(eta);
    MODEL DV ~ poisson(lambda) ;
    RANDOM e ~ normal(0,exp(2*logsig)) subject=FIPST;
RUN;
```