Does Integration Produce Equity?:
A Longitudinal Study of Neighborhood Conditions and Racial Health Inequality

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ABSTRACT

Research identifies racial residential segregation as a fundamental determinant of racial health disparities, but whether neighborhood economic conditions and neighborhood racial composition operate through similar or unique mechanisms to affect health remains unknown. Using hierarchical modeling strategies and longitudinal data, this study examines how the economic and racial characteristics of neighborhoods relate to markers of physiological functioning as individuals age. Results indicate that, after adjusting for neighborhood economic conditions, the associations between neighborhood racial composition and physical functioning vary by race and level of segregation. Whereas increased levels of neighborhood racial diversity are associated with better outcomes for Whites, neighborhood racial heterogeneity is associated with worse outcomes for Blacks. Conversely, net of economic conditions, high levels of residential segregation are associated with higher levels physiological dysregulation for Whites but lower levels of dysregulation for Blacks. Additional analyses suggest that psychosocial factors—including exposure to racial discrimination—may mediate these associations.

INTRODUCTION

Understanding how where individuals live influences how they live has been the focus of social scientific inquiry for more than 70 years. Since the publication of Shaw and McKay's (1942) *Juvenile delinquency and urban areas*, research on the effects of neighborhood conditions on individual well-being has surged, with a wide body of research now linking neighborhood context to a number of individual outcomes, including child development (Aneshenesel & Sucoff 1996; Brooks-Gunn et al. 1993; Ge et al. 2002; Hoffman 2002; Kowaleski-Jones 2000), educational achievement and attainment (Ainsworth 2002; Chase-Lansdale et al. 1997; Duncan 1994; Entwisle, Alexander, & Olson 1994; Halpern-Felsher et al. 1997), and social mobility (Sharkey 2012). Along this line of research, a number of studies have examined the role of neighborhood contexts in producing racial health inequality, with the extant literature now identifying racial residential segregation as a fundamental cause of racial health disparities (Williams & Collins 2001). Studies across disciplines link the conditions in racially segregated neighborhoods to disparities across a number of health outcomes, including low birthweight, depression, and cardiovascular disease (Diez-Roux et al. 1997; Williams & Jackson 2005).

While research examining the effects of racial residential segregation on health has boomed in recent years, critical gaps in the literature remain. First, whether neighborhood economic conditions and neighborhood racial composition operate through similar or unique mechanisms to affect health remains unknown, as many studies of the effects of racial residential segregation on health confound these distinct—yet related—characteristics of neighborhood context. Decades of racially discriminatory policies in practices in lending, real estate, educational, employment, and judiciary institutions have resulted in striking levels of racial residential segregation in the United States, as well as a concentration of poverty and economic

deprivation in segregated communities of color (Charles 2003; Massey & Denton 1993). The association between neighborhood racial composition and neighborhood economic conditions has led some studies to conflate the effects of these neighborhood characteristics on health, which may be problematic for two reasons. First, recent research documents a rise in "ethnoburbs," or middle-class or affluent suburban neighborhoods with high proportions of residents of color (Logan 2004; Wen, Lauderdale, & Kandula 2009). Given a documented variation in the correlation between neighborhood racial composition and economic conditions over time and across space, the confounding of neighborhood economic conditions and neighborhood racial composition may bias estimates of the effects of segregation on health. Second, conflating neighborhood economic conditions and neighborhood racial composition masks variation in how these dimensions of neighborhoods relate to markers of health and disease risk and restricts understanding of the mechanisms underlying the association between residential segregation and health. On the one hand, racial residential segregation may contribute to disease risk by concentrating poverty, reducing socioeconomic mobility, and producing unhealthy physical environments in minority majority neighborhoods (Williams & Collins 2001). On the other hand, racial segregation may be protective against poor health for people of color in particular, as individuals living in racially homogenous communities develop robust social networks and support systems that both reduce exposure to racial discrimination and buffer against stress and physiological dysregulation associated with racism (Bécares, Nazroo, & Stafford 2009). Nevertheless, it merits further investigation whether neighborhood socioeconomic deprivation and racial composition are independent or interrelated indicators of neighborhood context that uniquely contribute to individual health and well-being.

Second, while research links racial residential segregation to a host of adverse health outcomes, the biophysiological and psychosocial mechanisms underlying the association between neighborhood conditions and health are not well specified or tested (Daniel, Moore, & Kestens 2008; Hull et al. 2008; Wickrama & Bryant 2003). First, most studies of neighborhood effects on health examine the associations between neighborhood conditions and disease outcomes, which leaves questions about how neighborhood conditions "get under the skin" to affect well-being unanswered. As a result, concerns about biological plausibility remain in much of the literature on neighborhood effects on health (Daniel, Moore, & Kestens 2008). Further, much of the research on neighborhood effects on health focuses on how structural conditions—such as the availability of healthy food sources and the presence green space for physical activity—affect individual health outcomes. However, neighborhood conditions may also affect health through psychosocial mechanisms by reducing or increasing physiological stress response (Daniel, Moore, & Kestens 2008). Because so few studies specify and test the biophysiological and psychosocial underlying the association between structural conditions and individual outcomes, it remains to be better understood how neighborhood conditions contribute to disease risk through the conditioning of individual behavioral, psychosocial, and regulatory responses.

Finally, while a wide body of literature establishes a cross-sectional link between neighborhood context at a single point in time and health, few studies have examined how neighborhood conditions experienced at different life stages may jointly or uniquely affect health, which raises several concerns. For one, neighborhoods are not static, but rather they change dynamically over time (Sampson, Morenoff, & Gannon-Rowley 2002). As a result, individuals may experience a variety of neighborhood contexts over the life course, even if they never move. Second, there is evidence that the documented association between current

neighborhood characteristics and health may be subsuming the effects of earlier-life neighborhood conditions. For example, Wheaton & Clarke (2003) find a lagged effect of childhood neighborhood conditions on later life mental health that explains the apparent cross-sectional association between current neighborhood conditions on health. Further, life course epidemiologists suggest that events and conditions experienced during critical or sensitive experiences such as childhood or adolescence may become biologically embedded and have a greater influence on health outcomes and disease risk than conditions experienced later in the life course (Richardson et al. 2012). It remains unknown, for example, whether neighborhood conditions experienced during critical or sensitive periods such as childhood or adolescence have stronger associations with future disease risk than neighborhood conditions experienced later in life. Finally, the use of cross-sectional data also restricts researchers' ability to draw causal inferences about the impacts of neighborhood conditions on physiological regulation and disease risk.

This study fills these gaps in the literature by using three waves of longitudinal data from the National Study of Adolescent Health (Add Health) to examine how neighborhood economic conditions and racial composition uniquely relate to markers of physical functioning over time. I utilize longitudinal hierarchical modeling strategies to examine how neighborhood conditions affect health through both psychosocial and biophysiological mechanisms. By examining the economic, psychological, and physiological pathways connecting neighborhoods to health, this study improves understanding of the links between race, place, and health.

THEORETICAL MODEL

Neighborhoods and Health

Figure 1 displays the theoretical model guiding this study. This study is grounded in the ecological development perspective, which views individuals as developing and aging within a set of embedded social contexts that shape individual knowledge and skills, access to resources, exposure to risks, psychological well-being, and health (Bronfenbrenner 1986; Leventhal & Brooks-Gunn 2000; Wickrama & Bryant 2003). As indicated by Box 1 of Figure 1, the key social context of interest to this study is the neighborhood. While most studies in the ecological development perspective focus on the family as the central social construct shaping health and development, research indicates that extra-familial social contexts, such as neighborhoods, also influence individual behaviors and outcomes (Aneshenesel & Sucoff 1996; Brooks-Gunn et al. 1993; Wickrama & Bryant 2003).

[Figure 1 about here]

Research suggests that neighborhoods may directly influence physiological functioning and health (Path 1A), as the structural characteristics of neighborhoods may function as individual risk or protective factors (Hull et al. 2008; Wickrama & Bryant 2003). Neighborhood economic conditions can affect health by either promoting or restricting access to health-promoting material resources and increasing or reducing exposure to daily stress (Aneshenesel & Sucoff 1996; Ross, Reynolds, & Karlyn 2000). Individuals living in poor neighborhoods have less access than those in economically advantaged neighborhoods to health-promoting resources such as grocery stores and green space, and they also experience increased exposure to hazards such as toxins and violence (Williams and Collins 2001). Further, the daily stress of living in economically deprived or racially isolated areas may produce feelings of fear, hopelessness, frustration, distress, and loneliness (Aneshenesel & Sucoff 1996; Ross, Reynolds, & Karlyn 2000; Wickrama & Bryant 2003), which may further physiological dysregulation. In response to

stressors such as infections or perceived threats, the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) respond by secreting hormones to up-regulate physiological functioning (Hawkley et al. 2006; Christou et al. 2005; McEwen 1998; Selye 1978). While up-regulation immune, neuroendicine, metabolic, and cardiovascular changes in response to acute threats and infections is necessary for health, long-term activation of these systems in response to chronic stressors can harm health by promoting physiological dysregulation and increasing disease risk (Cohen et al. 2012; Kietcolt-Glaser et al. 2005; McEwen and Stellar 1993; McEwen 1998). This study seeks to investigate whether neighborhood conditions—including neighborhood economic deprivation and racial composition—directly influence health by up-regulating physiological stress response and increasing physiological dysregulation, as proposed by Path 1A in Figure 1.

Mediating Mechanisms

Social Isolation

In addition to the direct effects of neighborhoods on physiological functioning, the model presented in Figure 1 proposes that neighborhood conditions also exert an influence on individuals through two proximal psychosocial mechanisms. First, as indicated by Path 1B in Figure 1, neighborhood conditions may affect individual health by promoting or restricting the connectedness of residents (Box 2). In the literature, social integration refers to the structural dimension of social relationships and reflects the quantity and nature of ties in an individual's social network (Thoits 2011). Measures of social integration include relationship status (Hu & Goldman 1990; Litwak et al. 1989), number of network members (Berkman & Syme 1979) and frequency of contact with friends or family (Brummett et al. 2001). A wide body of research links social isolation—or a lack of social integration—to a range of diseases and outcomes,

including cardiovascular disease (Berkman et al. 1993; Orth-Gomér et al. 1993; Eng et al. 2002), cancer (Penwell & Larkin 2010), and depression (George et al. 1989; Heikkienen & Kauppinen 2004), as well as biomarkers of physiological dysregulation such as inflammation (Kiecolt-Glaser et al. 2010; Yang et al. 2013) and infection (Cohen et al. 1997). Social isolation has also been linked to higher rates of mortality (Berkman & Syme 1979; Thoits 1995; Yang at al. 2013). Studies suggest that social isolation influences physical functioning (Path 2A) through both stress inducing and behavioral mechanisms (Thoits 2011).

Neighborhood conditions may affect individual levels of social isolation (Path 1B) in two ways. First, because of social disorganization, research indicates that individuals living in economically disadvantaged communities may be less likely to form relationships with other residents (Kowaleski-Jones 2000) and less likely to participate in formal community organizations (Ross, Mirowsky, & Pribesh 2001; Sampson 2001) than individuals living in less disadvantaged neighborhoods. Second, neighborhood racial composition may also have an effect on individual social isolation. On the one hand, neighborhood racial heterogeneity may hinder the formation of social relationships, as diversity of languages, religions, and cultural practices may restrict communication and interaction between residents (Wickrama & Bryant 2003). On the other hand, research indicates that high levels of racial homogeneity may be conducive to the creation and maintenance of social relationships. In particular, non-Whites living in highly segregated neighborhoods may have more opportunities to form and maintain social relationships than they would in racially diverse neighborhoods (Bécares, Nazroo, & Stafford 2009; Wickrama & Bryant 2003).

Racial Discrimination

Neighborhood racial composition may also affect physiological functioning and disease risk by increasing or decreasing individual exposure to racial discrimination (Box 3). A wide body of research documents a link between racial discrimination and health (Path 3A), with the stress associated with racial discrimination being linked to depression, elevated blood pressure, chronic inflammation, and increased cardiovascular disease risk (Williams & Mohammed 2009; Williams, Neighbors, & Jackson 2003). Neighborhood racial composition may affect individual exposure to racial discrimination (Path 1C) by increasing or decreasing opportunities for inter-racial interaction and discrimination. Increased levels of neighborhood racial diversity may result in increased reports of racial discrimination by non-Whites, as increased interaction between racial groups increases opportunities for racial discrimination. Similarly, in highly segregated neighborhoods, non-Whites may experience less racial discrimination than in more diverse community contexts because of decreased inter-racial contact (Halpern & Nazroo 1999; Bécares, Nazroo, & Stafford 2009).

Variation across the life course

As indicated by the arrow labeled "Age" in Figure 1, this study is also guided by the life course perspective, which argues that human development and aging are life-long processes and that the antecedents and consequences of events and conditions for health can vary according to their timing in one's life (Pavalko & Willson 2011). Given the temporal dynamics of neighborhoods and findings suggesting that the health effects of neighborhoods vary by their life course timing, the model proposed here seeks to examine life course variation in the association between neighborhoods and health.

Research questions

This studies aims to address four key research questions:

1. Do neighborhood economic conditions and neighborhood racial composition have unique associations with markers of physiological well-being?
2. Does the association between neighborhood racial composition and physiological well-being vary by race?
3. Is there age variation in the associations between neighborhood characteristics and physiological well-being?
4. Do psychosocial factors such as social isolation and perceived discrimination mediate the associations between neighborhood characteristics and health?

DATA AND ANALYTIC METHODS

Data

Data for this study come from the National Longitudinal Study of Adolescent Health (Add Health), which is a nationally representative, longitudinal study of U.S. adolescents. Using a school-based complex cluster sampling frame, Add Health began in 1994-95 with an in-school questionnaire administered to a nationally-representative sample of students in grades 7-12. Following the in-school questionnaire, a gender- and grade-stratified random sample of 20,745 adolescents (79% response rate) was selected for in-home interviews at Wave I. The study then followed up with a series of in-home interviews conducted in 1996 (Wave II; 88% response rate), 2001-02 (Wave III; 77% response rate), and 2007-08 (Wave IV; 80% response rate). Add Health is a particularly rich source of data for studying life course trajectories of health and well-being because the study followed young people from their teen years through their transition to adulthood, allowing researchers to gain new insights into how young people's social contexts affect their health and well-being as they age.

In addition to the interviews, Add Health also collected biological specimens from study participants at Wave IV. The collection of physical measurements, saliva samples, and dried blood spots allows researchers to better understand the linkages between respondents' social lives and their cardiovascular, metabolic, and immunologic functioning. At Wave IV, Add Health interviewers collected a number of cardiovascular and anthropometric measures, including systolic blood pressure, diastolic blood pressure, height, weight, and waist circumference. The study also collected blood spots from respondents for a lipid panel and assays of glucose. For detailed information about biomarker collection procedures and protocols, see Entzel et al. (2009) and Whitsel et al. (2012).

At present, data for this study come from the in-home interviews and at Waves I and IV and the biomarker indicators at Wave IV. I also utilize Census tract-level data linked to respondents' residences at Wave I and IV. Future analyses will also include interview and Census tract-level data from Wave III.

Measures

Dependent Variables

The outcomes of interest include two measures of physical functioning that represent important risk factors for a future morbidity and mortality: metabolic dysregulation and body mass index (BMI). All outcomes are measured at Wave IV, when respondents were aged 24-32 years.

I construct an index of *metabolic dysregulation*, which indicates overall levels of metabolic burden using clinical markers of metabolic syndrome: waist circumference, serum triglycerides, HDL cholesterol, serum glucose, systolic blood pressure, and diastolic blood pressure. For each individual measure, I construct a dummy measure where 1 indicates high risk.

Cut points for high risk were defined by clinical practice or empirically defined as the top quintile (bottom quintile was used for HDL cholesterol). I then summed the scores from each of the markers to construct the index of overall metabolic burden, which is modeled as a continuous measure ranging from 0 (low metabolic dysregulation) to 6 (high metabolic dysregulation).

Body mass index (BMI) is included as a continuous measure. Research indicates that BMI is predictive of health and longevity, with overweight (BMI≥25kg/m^2) and obese (BMI≥30kg/m^2) individuals being at increased risk of cardiovascular disease, cancer, disability, and premature mortality (Kopelman 2007).

Key Explanatory Variables

Neighborhood Conditions

I measure *neighborhood economic deprivation* at Waves I and IV using a composite index of four measures of neighborhood economic conditions using Census tract-level data: proportion of residents who are unemployed, proportion of residents over the age of 25 years without a high school degree, proportion of families living in poverty, and proportion of families receiving public assistance. For each measure, I created a dummy variable indicating the top quartile of all Census tracts (e.g., the Census tracts in the top quartile for unemployment). To create the index of neighborhood economic deprivation, I summed the four measures, producing an index ranging from 0 (low deprivation) to 4 (high deprivation).

To measure neighborhood racial composition, I create a measure of *neighborhood racial density* at Waves I and IV. This measure indicates the percentage of neighborhood residents who are the same race as the respondent (White, Black or Hispanic). In order to capture non-linearities in the relationship between neighborhood racial heterogeneity, I also include a measure of *neighborhood racial density*^{2}, which captures high levels of racial homogeneity.

Psychosocial Mediators and Moderators

As shown in Figure 1, I examine two possible mechanisms through which neighborhood conditions may get "under the skin" to affect physical well-being: social isolation (Box 2) and perceived discrimination (Box 3), both of which are measured at Wave IV using data from the in-home interviews. Measures included in the index of *social isolation* include relationship status, number of close friends, frequency of religious attendance, and frequency of volunteering. To construct the index of social isolation, I create dummy variables for each measure, with 1 indicating the top quartile of all respondents (with the exception of relationship status, where 1=married or cohabiting at Wave IV). The index ranged from 0 (highest isolation) to 4 (lowest isolation). In preliminary models, I include social isolation as a dummy variable where "1" indicates an isolation index score of 0 or 1.

Perceived discrimination is constructed using data from the in-home interview at Wave IV. Add Health asked respondents, "In your day-to-day life, how often do you feel you have been treated with less respect or courtesy than other people?" Perceived discrimination is included as a categorical measure, where 0=never, 1=rarely, 2=sometimes, and 3=often.

Covariates

Race is included as a categorical measure, where 1=White, 2=Black, and 3=Hispanic. All analyses adjust for *gender* (1=female), *age*, and *SES*. At both Waves I and IV, I include SES as a composite measure, where SES is calculated as the mean of standardized (z-score) measures of economic well-being. At Wave I, the measures included in the composite SES measure include parental education and household income. At Wave IV, SES reflects the respondent's level of education attainment and household income. For both SES measures, positive values represent higher levels of SES.

Analytic Sample

I run analyses for metabolic dysregulation and BMI separately, with each outcome corresponding to a different analytic sample size. The samples include respondents who have complete data on the variables included in the analyses as well as valid survey sampling weights. The metabolic dysregulation models include 5,256 respondents, and the BMI sample includes 5,221 respondents.

Analytic Methods

I use multilevel regression models to examine the influence of individual and neighborhood-level predictors on metabolic dysregulation and BMI. The use of multi-level models allows to me account for the nested nature of the data, where individuals are clustered within neighborhoods. In the multilevel models, individual outcomes are predicted by individual-level factors, neighborhood-level characteristics, and interactions between individual and neighborhood factors. The multilevel models include error terms at both the individual and neighborhood levels.

I estimate models for metabolic dysregulation and BMI separately. For each outcome, I begin with Model 1, which regresses the outcome measures on neighborhood and individual characteristics at during adolescence (Wave I). Model 2 regresses the outcomes on neighborhood and individual characteristics experienced during young adulthood (Wave IV). Comparing coefficient estimates and model fit statistics across Models 1 and 2 provides insights into whether neighborhood characteristics experienced during different life course stages have differential effects on biomarkers of health during young adulthood. For each outcome, Models 3 and 4 build off of Model 2 and integrate measure of perceived discrimination and social isolation, respectively, into the multilevel models.

All analyses were performed in Stata 13.0 and use sample weights to ensure the representativeness of the respondents.

PRELIMINARY RESULTS

Descriptive Statistics

Descriptive statistics for all the variables used in the analyses are presented in Table 1. Table 2 presents the means for the outcome measures and neighborhood characteristics by race. Table 2 reveals racial differences in mean BMI, with Blacks and Hispanics having higher BMIs than White respondents ($p<0.001$). Further, descriptive statistics reveal stark racial disparities in neighborhood characteristics during both adolescence and the transition to adulthood. On average, Blacks and Latinos experience greater levels of neighborhood economic deprivation than Whites during both adolescence and the transition to adulthood ($p<0.001$). However, Whites live in neighborhoods characterized by higher levels of racial segregation than Blacks and Hispanics, as indicated by Whites' higher mean neighborhood racial density during both adolescence and young adulthood ($p<0.001$).

[Table 1 about here]

[Table 2 about here]

Multilevel Models

Metabolic Dysregulation

Table 3 displays the results of the multilevel regression analyses for metabolic dysregulation, where I regress metabolic dysregulation (measured during young adulthood) on neighborhood characteristics, individual characteristics, and psychosocial factors. For all models, Table 3 presents the coefficients estimates and standard errors.

[Table 3 about here]

As shown in Model 1 of Table 3, neighborhood context experienced during adolescence is not associated with metabolic dysregulation, net of individual characteristics. While neighborhood characteristics during adolescence are not associated with metabolic dysregulation, individual and family factors during adolescence predict metabolic dysregulation in young adulthood. Family SES during adolescence is protective against metabolic dysregulation, such that increases in SES reduce metabolic risk (β=-0.168, p<0.001).

Model 2 regresses metabolic dysregulation on neighborhood and individual factors during young adulthood. Net of individual characteristics, neighborhood economic deprivation during young adulthood is not significantly associated with metabolic risk. Neighborhood racial composition is significantly associated with metabolic dysregulation, but the direction of the association varies by race and level of segregation. Increasing levels of neighborhood racial heterogeneity (as indicated by the coefficient for neighborhood racial density) are associated with reduced levels of metabolic dysfunction for Whites (β=-0.020, p<0.05), whereas increased levels of neighborhood racial heterogeneity are associated with higher levels of metabolic dysregulation for Blacks (β=0.022, p<0.1). However, high levels of neighborhood racial homogeneity (as indicated by the coefficient for neighborhood racial density^2) increase levels of metabolic dysregulation for Whites (β=0.017, p<0.05), but reduce levels of metabolic dysregulation for Blacks (β=-0.017, p<0.1). In addition to neighborhood factors, individual SES is also associated with metabolic risk, with increases in individual SES protecting against metabolic dysregulation (β=-0.131, p<0.001). It is also worth noting that, after adjusting for neighborhood characteristics and individual SES, the Black-White disparity in metabolic dysregulation is no longer significant.

Models 3 and 4 of Table 3 integrate measures of perceived discrimination and social isolation, respectively. While perceived discrimination is not significantly associated with metabolic dysregulation, social isolation is predictive of metabolic risk. Compared to more socially integrated individuals, those with high levels of isolation have higher levels of metabolic dysregulation (β=0.103, p<0.05).

BMI

Table 4 presents the results of the multilevel regression analyses for BMI, where BMI in young adulthood is predicted by neighborhood characteristics, individual characteristics, and psychosocial factors. Table 4 displays the coefficients estimates and standard errors for all models.

[Table 4 about here]

Consistent with model estimates for metabolic dysregulation, Model 1 of Table 4 indicates that neighborhood context experienced during adolescence is not associated with BMI at young adulthood, net of individual characteristics. While neighborhood characteristics during adolescence are not associated with BMI, individual and family factors during adolescence predict BMI. In particular, family SES during adolescence is protective against BMI, such that increases in SES are associated with decreases in BMI (β=-0.975, p<0.001).

Model 2 regresses BMI on neighborhood and individual factors during young adulthood. Net of individual characteristics, including SES, neighborhood economic deprivation during young adulthood is positively associated with BMI (β=0.566, p<0.001). That is, even after controlling for individual SES, individuals living in areas of increased neighborhood economic disadvantage have higher BMIs than individuals living in less disadvantaged neighborhoods. In addition to neighborhood SES, neighborhood racial composition is also significantly associated

with BMI. Similar to the model estimates for metabolic dysregulation, I find that the direction of the association between neighborhood racial composition and BMI varies by race and level of racial segregation. Whereas increases in neighborhood racial heterogeneity are associated with lower BMI for Whites (β=-0.188, $p<0.1$), increased levels of neighborhood racial heterogeneity are associated with higher BMI for Blacks (β=0.244, $p<0.01$). Conversely, high levels of White neighborhood segregation (as indicated by the coefficient for neighborhood racial density^2) are associated with increases in BMI for Whites (β=0.080, $p<0.1$), but high levels of Black segregation are associated with lower BMI for Blacks (β=-0.210, $p<0.01$). In addition to neighborhood factors, individual SES is also negatively associated with BMI, with increases in individual SES resulting in lower BMI (β=-0.566, $p<0.05$).

Models 3 and 4 of Table 4 introduce measures of perceived discrimination and social isolation, respectively. I find a positive association between perceived discrimination and BMI. Compared to individuals who do not report perceptions of discrimination, individuals who report that they perceive discrimination have higher BMIs (β=2.631, $p<0.001$). Social isolation is not significantly associated with BMI.

DISCUSSION & NEXT STEPS

While research widely cites racial residential segregation as fundamental determinant of racial health inequality, critical gaps in the literature remain. In particular, it remains unknown whether neighborhood economic conditions and neighborhood racial composition have similar or unique associations with health. The present study aims to improve understanding of the links between race, place, and health by examining how neighborhood conditions—including neighborhood economic deprivation and racial composition—"get under the skin" to affect health and well-being as individuals age.

Descriptive analyses presented in Table 2 reveal stark racial disparities in neighborhood context and markers of physical functioning. Compared to Whites, Blacks and Hispanics have higher BMIs and experience higher levels of neighborhood economic deprivation. On the other hand, Whites are more likely than Blacks and Hispanics to live in areas of high racial density. On average, Whites reside in overwhelmingly White spaces, with the average White adolescents living in a neighborhood where 9 in 10 residents are White.

Preliminary results from the multilevel regression analyses in Tables 3 and 4 suggest that neighborhood conditions play a fundamental role in individual disease risk, though in nuanced ways that vary by age, race, and outcome. While I found no association between neighborhood economic deprivation and metabolic dysregulation, results document that living in an economically deprived neighborhood in young adulthood was associated with higher BMI, net of individual SES. That is, contextual disadvantages associated with neighborhood economic deprivation confer health risks to residents, above and beyond the health risks associated with low individual or household SES. These findings offer further support for previous research that suggests that neighborhood economic conditions can act as protective or risk factors by either promoting or restricting access to health-promoting material resources and increasing or reducing exposure to daily stress (Aneshenesel & Sucoff 1996; Ross, Reynolds, & Karlyn 2000).

Further, preliminary analyses suggest that neighborhood economic conditions and neighborhood racial composition have unique effects on health. Models 2, 3, and 4 of Tables 3 and 4 indicate that neighborhood racial composition is significantly related to physical functioning, but the direction of the association varies by race and level of segregation. Results from both the metabolic dysregulation and BMI models indicate that, after adjusting for neighborhood economic conditions, Whites receive protective benefits from living in areas of

increasing racial heterogeneity. For Whites, increased levels of neighborhood racial diversity are negatively associated with metabolic dysregulation and BMI. However, high levels of White segregation are associated with worse outcomes for Whites. Compared with Whites, the association between neighborhood racial composition and physical functioning is in the opposite direction for Blacks. Whereas Whites have better outcomes with increasing neighborhood racial diversity, Blacks have worse outcomes in areas of increased racial heterogeneity. Conversely, whereas high levels of White segregation prove detrimental for Whites, high levels of Black segregation offer protective benefits for Blacks. Net of neighborhood economic deprivation, Blacks living in predominately Black neighborhoods have lower levels of metabolic dysregulation and BMI, compared to Blacks living in racially diverse neighborhoods. These findings are particularly pronounced in the BMI results presented in Table 4. These results speak to the importance of considering both neighborhood economic conditions and neighborhood racial composition in studies of health inequality, as these characteristics of neighborhoods confer different health effects to residents.

Earlier research has hypothesized that high levels of non-White residential segregation, in particular, may be protective for people of color because of the increased opportunities for social integration and decreased exposure to racial discrimination that result from living in a segregated neighborhood (Wickrama & Bryant 2003). Results from Models 3 and 4 of Tables 3 and 4 offer some preliminary evidence that these psychosocial mechanisms may, in fact, mediate the association between neighborhood racial composition and health. I find that social isolation is associated with increased risk of metabolic dysregulation and that perceptions of discrimination are associated with higher BMI. These findings offer preliminary evidence that the protective benefits Blacks receive from living in hyper-segregated communities may be because of the

increased opportunities for social integration and the decreased exposure to racial discrimination in these racially homogenous communities, which may buffer them against biophysiological stress response. The analyses presented here are preliminary, however, and do not formally test for mediation. Future analyses will formally test whether social isolation and perceived discrimination mediate the associations between neighborhood contexts and biophysiological well-being using Sobel-Goodman tests.

This study also documents significant age variation in the associations between neighborhood characteristics and physical functioning. Previous research has documented that the neighborhood conditions earlier in the life course may be particularly important for later life health (Wheaton & Clarke 2003), offering support for the critical or sensitive period hypothesis (Richardson et al. 2012). However, the results presented in Tables 3 and 4 do not lend support for this hypothesis. In fact, I find no associations between neighborhood characteristics experienced during adolescence and markers of physical functioning in young adulthood. Instead, I find that neighborhood context experienced during young adulthood—at the same time as the measurement of the outcome—is significantly associated with markers of physical functioning. The present study utilized data from Waves I and IV only. Future analyses will build multi-level longitudinal models using data from Waves I, III, and IV to examine the cumulative effects of neighborhood conditions on biomarkers of health. Further, I will also examine the joint effects of neighborhood conditions experienced at different life stages on health by simultaneously entering neighborhood conditions across the life course into analytic models.

Finally, all studies of neighborhood effects on health are subject to concerns about selection. The present study does not account for selection, though future analyses will attempt to adjust for the role of neighborhood selection using inverse probability of treatment models.

By examining the psychosocial and biophysiological mechanisms linking neighborhoods to health, this study improves understanding of the contextual and structural determinants of racial health inequality. In particular, this study emphasizes the primary role of place in contributing to the social stratification of health and offers a more nuanced story of the role of residential segregation in the production of racial health inequality than has been previously documented. While racial residential segregation is often implicated in the literature as a key determinant of racial health disparities (Williams & Collins 2001; Williams & Jackson 2005), questions remain as to why and how spaces "get under the skin" to affect disease risk and longevity. The findings presented here suggest that it is not the mere patterning of individuals of different races across space that contributes to health inequality, but it is the flow of socioeconomic resources and power that fall along racial and spatial lines that contributes to the unequal distribution of disease risk. People of color in the United States are more likely than Whites to live in economically disadvantaged households and neighborhoods, which, as shown in the present study, contributes to disparities in physiological dysregulation and disease risk. However, this study finds that, after adjusting for differences in neighborhood economic conditions, living in racially segregated spaces may confer psychosocial benefits to Blacks, in particular, that may buffer against the physiological dysregulation associated with chronic stressors. By no means does this study offer support for continued racial segregation, but it rather suggests that the mere mixing of individuals of different races will not eliminate racial health inequality. In fact, the findings presented here suggest that promoting neighborhood racial

heterogeneity without understanding of how racism contributes to health inequality through both psychosocial and socioeconomic means may actually exacerbate health inequality.

REFERENCES

Ainsworth, J.W.. 2002. Why Does It Take a Village? The Mediation of Neighborhood Effects on Educational Achievement. *Social Forces* 81(1):117-52.

Aneshensel, C. S. & C.A. Sucoff. 1996. The neighborhood context of adolescent mental health. *Journal of Health and Social Behavior* 37:293-310.

Berkman, L.F., & L. Syme. 1979. Social Networks, Host Resistance, and Mortality: A Nine Year Follow-up Study of Alameda County Residents. *American Journal of Epidemiology* 109(2):186-204.

Bécaresa, L., J. Nazroob, & M. Stafforda. 2009. The buffering effects of ethnic density on experienced racism and health. *Health & Place* 15(3):700-08.

Berkman, L.F., V. Vaccarino, & T. Seeman. 1993. Gender Differences in Cardiovascular Morbidity and Mortality: The Contributions of Social Networks and Support. *Annals of Behavioral Medicine* 15(2-3):112-118.

Bronfenbrenner, U. 1986. Ecology of the family as a context for human development: Research perspectives. *Developmental Psychology* 22: 723-42.

Brooks-Gunn, J., G.J. Duncan, P.K. Klebanov, & N. Sealand. 1993. Do neighborhoods influence child and adolescent development? *American Journal of Sociology* 99(2):353-95.

Brummett, B.H., J.C. Barefoot, I.C. Siegler, N.E. Clapp-Channing, B.L. Lytle, H.B. Bosworth,

R.B. Williams, B. Redford, & D.B. Mark. 2001. Characteristics of socially isolated patients with coronary artery disease who are at elevated risk for mortality. *Psychosomatic Medicine, 63*(2), 267-272.

Charles, C. Z. 2003. The dynamics of racial residential segregation. *Annual Review of Sociology* 167-207.

Christou, D. D., P.P. Jones, J. Jordan, A. Diedrich, D. Robertson, & R.D. Seals. 2005. Women have lower tonic autonomic support of arterial blood pressure and less effective baroreflex buffering than men. *Circulation* 111:494– 98.

Clarke, P., J. Morenoff, M. Debbink, E. Golberstein, M.R. Elliot, & P.M. Lantz. 2004. Cumulative Exposure to Neighborhood Context: Consequences for Health Transitions Over the Adult Life Course. *Research on Aging* 36(1):115-42.

Cohen, S.D., J. William, D.P. Skoner, B.S. Rabin, & J.M. Gwaltney Jr. 1997. Social ties and susceptibility to the common cold. *JAMA* 277(24):1940-44.

Daniel, M., S. Moore, & Y. Kestens. 2008. Framing the biosocial pathways underlying associations between place and cardiometabolic disease. *Health & Place* 14(2):117-32.

Diez-Roux, Ana V., et al. 1997. Neighborhood environments and coronary heart disease: a multilevel analysis. *American Journal of Epidemiology* 146(1): 48-63.

Duncan, G. J. 1994. Families and neighbors as sources of disadvantage in the schooling decisions of White and Black adolescents. *American Journal of Education* 103:20-53.

Eng, P.M.., Rimm, E.B.., Fitzmaurice, G., & Kawachi, I. 2002. Social Ties and Change in Social Ties in Relation to Subsequent Total and Cause-Specific Mortality and Coronary Heart Disease in Men. *American Journal of Epidemiology* 155(8):700-09.

Entwisle, D. R., Alexander, K. L., & Olson, L. S. 1994. The gender gap in math: Its possible origins in neighborhood effects. *American Sociological Review* 59:822– 838.

Entzel, P., E. Whitsel, A. Richardson, J. Tabor, S. Hallquist, J. Hussey, C. Halpern, and K. Mullan Harris. 2009. "Add Health Wave IV Documentation: Cardiovascular and Anthropmetric Measures." Retrieved January 2, 2014 from http://www.cpc.unc.edu/projects/addhealth/data/guides/

Ge, X., Gene, B., Conger, R. D., Simons, R., & Murry, V. M. 2002. Contextual amplification of pubertal transition effects on deviant peer affiliation and externalizing behavior among African American children. *Developmental Psychology* 28:42-54.

George, L.K., Blazer, D.G., Hughes, D.C., & Fowler, N. 1989. Social support and the outcome of major depression. *The British Journal of Psychiatry* 154(4): 478-85.

Glaser, R. and J. K. Kiecolt-Glaser. 2005. Stress-induced immune dysfunction: implications for health. *National Review of Immunology* 5(3): 243-251.

Halpern, D. and Nazroo, J.Y. 1999. The ethnic density effect: results from a national community survey of England and Wales. *International Journal of Social Psychiatry* 46:34–46.

Halpern-Felsher, B., Connell, J. P., Spencer, M. B., Aber, J. L., Duncan, G. J., Clifford, E., Crichlow, W., Usinger, P., & Cole, S. S. 1997. Neighborhood and family factors predicting educational risk and attainment in African American and White children and adolescents. In J.Brooks-Gunn, G. J.Duncan, & J. L.Aber (Eds.), *Neighborhood poverty: Vol. 1*. Context and consequences for children (pp. 146– 173). New York: Russell Sage Foundation.

Hawkley, L.C., Masi, C.M., Berry, J.D., & Cacioppo, J.T. 2006. Loneliness Is a Unique

Predictor of Age-Related Differences in Systolic Blood Pressure. *Psychology and Aging* 21(1):152-64.

Heikkinen, R.L,, & Kauppinen, M. 2004. Depressive symptoms in late life: a 10-year follow up. *Archives of gerontology and geriatrics, 38*(3), 239-250.

Hoffman, J. P. 2002. The community context of family structure and adolescent drug use. *Journal of Marriage and Family* 64:314-330.

Hu, Y. & Goldman, N. 1990. Mortality differentials by marital status: an international comparison. *Demography*, 27(2), 233-250.

Hull, P., Kilbourne, B., Reece, M., & Husaini, B. 2008. Community involvement and adolescent mental health: Moderating effects of race/ethnicity and neighborhood disadvantage. *Journal of Community psychology* 36(4):534-551.

Kopelman, P. 2007. Health risks associated with overweight and obesity. *Obesity Reviews* 8(S1):13-17.

Kowaleski-Jones, L. 2000. Staying out of trouble: Community resource and problem behavior among high-risk adolescents. *Journal of Marriage and the Family* 62:449-464.

Leventhal, T. and J. Brooks-Gunn. 2000. The neighborhoods they live in: The effects of neighborhood residence on child and adolescent outcomes. *Psychological Bulletin* 126:309-337.

Litwak, E., Messeri, P., Wolfe, S., Gorman, S., Silverstein, M., &, Guilarte, M. 1989. Organizational theory, social supports, and mortality rates: A theoretical convergence. *American Sociological Review 54*: 49-66.

Logan, J. 2004. "The New Ethnic Enclaves in America's suburbs." A Report by The Lewis Mumford Center for Comparative Urban and Regional Research. Available at: http://

mumford1.dyndns.org/cen2000/suburban/SuburbanReport/page1.html.

Massey, D. S. and N.A. Denton. 1993. *American apartheid: Segregation and the making of the underclass*. Harvard University Press.

McDade, T.W., L.C. Hawkley, and J.T. Cacioppo. 2006. Psychosocial and behavioral predictors of inflammation in middle-aged and older adults: the Chicago health, aging, and social relations study. *Psychosomatic Medicine* 68(3): 376-81.

McEwen, B.S. 1998. Stress, Adaptation, and Disease - Allostasis and Allostatic Load. *Annals of the New York Academy of Sciences* 840(1): 33-44.

Orth-Gomér, K., Rosengren, A., & Wilhelmsen, L. 1993. Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. *Psychosomatic Medicine* 55(1):37-43.

Pavalko, E. & A. Wilson. 2011. Life Course Approaches to Health, Illness, and Healing. In B. A. Pescolido (Ed.) *Handbook of the Sociology of Health, Illness, and Healing*. Springer.

Penwell, L.M., & Larkin, K.T. 2010. Social Support and Risk for Cardiovascular Disease and Cancer: A Qualitative Review Examining the Role of Inflammatory Processes. *Health Psychology Review 4*(1):42-55.

Richardson, L.J., J.M. Hussey, & K.L. Strutz. 2012. A life course perspective on maternal and child health. In: Kotch, Jonathan B. *Maternal and Child Health: Programs, Problems, and Policy in Public Health, 3rd ed,* pp. 65-85. Burlington, MA: Jones & Bartlett

Ross, C.E., J. Mirowsky, and K. Goldsteen. 2001. Powerlessness and the amplification of threat: Neighborhood disadvantage, disorder, and mistrust. *American Sociological Review* 66:568-91.

Ross, C.E., J.R. Reynolds, and L.G. Karlyn. 2000. The contingent meaning of neighborhood

stability for residents' psychological well-being. *American Sociological Review* 65: 581-97.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. 1999. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation* 99:2192-2217.

Sampson, R. 2001. How do communities undergird or undermine human development? Relevant contexts and social mechanisms. Pp. 3-30 in A. Booth and A.C. Crouter (eds.) *Does it take a village? Community effects on children, adolescents, and families*. Mahwah, NJ: Lawrence Erlbaum Associates.

Sampson, R., J. Morenoff, & DT. Gannon-Rowley. 2002. Assessing "neighborhood effects": Social processes and new directions in research. *Annual Review of Sociology* 28:443-78.

Sesso HD, Stampfer MJ, Rosner B, Hennekens CH, Gaziano JM, Manson JE, and Glynn RJ. 2000. Systolic and diastolic blood pressure, pulse pressure, and mean arterial pressure as predictors of cardiovascular disease risk in Men. *Hypertension* 36(5):801-7.

Selye, H. 1974. *Stress Without Distress*. Philadelphia: J.B. Lippincott.

Sharkey, P. 2012. An Alternative Approach to Addressing Selection Into and Out of Social Settings: Neighborhood Change and African American Children's Economic Outcomes. *Sociological Methods Research* 41(2):251-293.

Shaw, C.R. and H.D. McKay. 1942. *Juvenile delinquency and urban areas*. Chicago, IL: University of Chicago Press.

Thoits, P.A. 2011. Mechanisms linking social ties and support to physical and mental health. *Journal of Health and Social Behavior, 52*(2), 145-161.

Van Uum, S.H.M., Lender, J.W.M., & Hermus, A.R.M.M. 2004. Cortisol, 11β-hydroxysteroid

dehydrogenases, and hypertension. *Seminars in Vascular Medicine* 4(2):121– 28.

Wen, M., D.S. Lauderdale, & N.R. Kandula. 2009. Ethnic Neighborhoods in Multi-Ethnic America, 1990-2000: Resurgent Ethnicity in the Ethnoburbs? *Social Forces* 88(1):425-60.

Wheaton, B. & P. Clarke. 2003. Space meets time: Integrating temporal and contextual influences on mental health in early adulthood. *American Sociological Review* 68:680-706.

Whitsel, E., C. Cuthbertson, J. Tabor, A. Potter, M. Wener, L. Killeya-Jones, and K. Mullan Harris. 2012. "Add Health Wave IV Documentation: Measures of Inflammation and Immune Function." Retrieved January 2, 2014 from http://www.cpc.unc.edu/projects/addhealth/data/guides/

Whitworth, J. A., Brown, M. A., Kelly, J. J., & Williamson, P. M. 1995. Mechanisms of cortisol induced hypertension in humans. *Steroids* 60:76– 80.

Wickrama, K. and C. Bryant. 2003. Community Context of Social Resources and Adolescent Mental Health. *Journal of Marriage and Family* 65(4): 850-66.

Williams, D.R., and C. Collins. 2001. Racial residential segregation: a fundamental cause of racial disparities in health. *Public health reports* 116(5): 404.

Williams, D.R. and P.B. Jackson. 2005. Social sources of racial disparities in health. *Health Affairs* 24(2):325-334.

Williams, D.R. and S.A. Mohammed. 2009. Discrimination and racial disparities in health: evidence and needed research. *Journal of Behavioral Medicine* 32(1):20-47.

Williams, D.R., H.W. Neighbors, and J.S. Jackson. 2003. Racial/ethnic discrimination and

health: findings from community studies. *American Journal of Public Health* 93(2):200-208.

Yang, Y.C., McClintock, M.K., Kozloski, M., & Li, T. 2013. Social isolation and adult mortality: the role of chronic inflammation and sex differences. *Journal of Health and Social Behavior* 54(2):183-203.

Yang, Y., & Kozloski, M. 2012. Change of Sex Gaps in Total and Cause-Specific Mortality Over the Life Span in the United States. *Annals of Epidemiology* 22(2):94-103.

Table 1. Sample Descriptive Statistics, Weighted (N=5,256)

	Mean or proportion	(SD)
Outcome measures[a]		
Metabolic dysregulation	2.58	(1.25)
BMI	28.77	(7.35)
Neighborhood characteristics		
…during adolescence[b]		
Neighborhood economic deprivation	0.86	(1.48)
Neighborhood racial density	79.64	(30.18)
…during young adulthood[a]		
Neighborhood economic deprivation	0.69	(1.04)
Neighborhood racial density	72.12	(29.43)
Individual characteristics		
Race		
White	0.75	-
Black	0.16	-
Hispanic	0.09	-
Gender (1=female)		
Age[b]	14.68	(1.81)
Family SES during adolescence[b]	0.04	(0.75)
SES during young adulthood[a]	0.03	(0.81)
Psychosocial factors[a]		
Perceived discrimination		
Never	0.30	-
Rarely	0.48	-
Sometimes	0.19	-
Often	0.04	-
Social isolation (1=isolated)	0.46	-

a: Measured during young adulthood (Wave IV)

b: Measured during adolescence (Wave I)

Table 2. Outcome and Neighborhood Measures, by Race

	Whites	Blacks	Hispanics	p-value
Outcome measures[a]				
Metabolic dysregulation	2.57	2.67	2.51	0.160
BMI	28.32	30.61	29.24	<0.001
Neighborhood characteristics				
…during adolescence[b]				
Neighborhood economic deprivation	0.53	2.14	1.36	<0.001
Neighborhood racial density	91.43	53.86	28.02	<0.001
…during young adulthood[a]				
Neighborhood economic deprivation	0.52	1.35	0.92	<0.001
Neighborhood racial density	82.91	44.72	31.43	<0.001

a: Measured during young adulthood (Wave IV)

b: Measured during adolescence (Wave I)

c: p-value of chi-square test of difference between in means between racial groups

Note: statistics are survey design adjusted and weighted

Table 3. Neighborhood Characteristics and Health: Multilevel Regression Models for Metabolic Dysregulation, 1994-2008

	Model 1 Coeff (SE)	**Model 2** Coeff (SE)	**Model 3** Coeff (SE)	**Model 4** Coeff (SE)
Neighborhood characteristics				
…during adolescence (Wave I)				
Neighborhood economic deprivation	0.012			
	(0.021)			
Neighborhood racial density	0.021			
	(0.014)			
Black x neighborhood racial density	-0.025			
	(0.017)			
Hispanic x neighborhood racial density	-0.022			
	(0.015)			
Neighborhood racial density^2	-0.012			
	(0.009)			
Black x neighborhood racial density^2	0.018			
	(0.013)			
Hispanic x neighborhood racial density^2	0.010			
	(0.011)			
…during young adulthood (Wave IV)				
Neighborhood economic deprivation		0.004	0.004	0.007
		(0.027)	(0.028)	(0.027)
Neighborhood racial density		-0.020*	-0.020*	-0.020*
		(0.009)	(0.009)	(0.009)
Black x neighborhood racial density		0.022*	0.022*	0.021*
		(0.011)	(0.011)	(0.011)
Hispanic x neighborhood racial density		0.014	0.013	0.014
		(0.015)	(0.015)	(0.015)
Neighborhood racial density^2		0.017*	0.017**	0.017*

		(0.007)	(0.007)	(0.007)
Black x neighborhood racial density^2		-0.017†	-0.017†	-0.016†
		(0.009)	(0.009)	(0.009)
Hispanic x neighborhood racial density^2		-0.009	-0.009	-0.009
		(0.013)	(0.013)	(0.013)
Individual characteristics				
Race (white is reference)				
Black	0.899†	-0.477	-0.476	-0.472
	(0.531)	(0.333)	(0.331)	(0.331)
Hispanic	0.793	-0.400	-0.398	-0.400
	(0.500)	(0.412)	(0.413)	(0.413)
Gender (1=female)	-0.315***	-0.296***	-0.294***	-0.292***
	(0.055)	(0.059)	(0.056)	(0.056)
Age	0.059***	0.061***	0.061***	0.063***
	(0.014)	(0.014)	(0.014)	(0.014)
Family SES during adolescence	-0.168***			
	(0.031)			
SES during young adulthood		-0.131***	-0.129***	-0.114***
		(0.037)	(0.034)	(0.038)
Psychosocial factors				
Perceived discrimination (never is reference)				
Rarely			0.039	
			(0.053)	
Sometimes			0.023	
			(0.089)	
Often			0.062	
			(0.217)	
Social isolation (1=isolated)				0.103*
				(0.044)
Intercept	1.014†	2.255***	2.227***	2.157***

	(0.549)	(0.448)	(0.442)	(0.449)
Model Fit Statisics				
AIC	3095328	3093732	3093497	3093616
BIC	3095427	3093830	3092026	3092131
N (unweighted)	5,256	5,256	5,256	5,256

***p<0.001, **p<0.01, *p<0.05, †p<0.1

Note: all models are survey design adjusted and weighted

Table 4. Neighborhood Characteristics and Health: Multilevel Regression Models for BMI, 1994-2008

	Model 1 Coeff (SE)	**Model 2** Coeff (SE)	**Model 3** Coeff (SE)	**Model 4** Coeff (SE)
Neighborhood characteristics				
…during adolescence (Wave I)				
Neighborhood economic deprivation	0.104			
	(0.124)			
Neighborhood racial density	0.034			
	(0.061)			
Black x neighborhood racial density	-0.025			
	(0.084)			
Hispanic x neighborhood racial density	0.002			
	(0.077)			
Neighborhood racial density^2	-0.031			
	(0.041)			
Black x neighborhood racial density^2	0.007			
	(0.076)			
Hispanic x neighborhood racial density^2	-0.047			
	(0.067)			
…during young adulthood (Wave IV)				
Neighborhood economic deprivation		0.566***	0.517***	0.560***
		(0.158)	(0.163)	(0.159)
Neighborhood racial density		-0.118†	-0.120†	-0.118†
		(0.062)	(0.062)	(0.063)
Black x neighborhood racial density		0.244**	0.248***	0.245***
		(0.093)	(0.093)	(0.094)
Hispanic x neighborhood racial density		0.129	0.131	0.129
		(0.101)	(0.101)	(0.101)
Neighborhood racial density^2		0.080†	0.081†	0.080†

		(0.048)	(0.047)	(0.048)
Black x neighborhood racial density^2		-0.210**	-0.210***	-0.210***
		(0.075)	(0.074)	(0.075)
Hispanic x neighborhood racial density^2		-0.116	-0.114	-0.115
		(0.092)	(0.092)	(0.093)
Individual characteristics				
Race (white is reference)				
Black	2.718	-4.337	-4.498†	-4.339
	(2.360)	(2.680)	(2.685)	(2.696)
Hispanic	1.296	-2.866	-2.969	-2.861
	(2.294)	(2.289)	(2.291)	(2.307)
Gender (1=female)	-0.200	-0.088	-0.079	-0.098
	(0.291)	(0.303)	(0.306)	(0.302)
Age	0.236**	0.251**	0.252***	0.247***
	(0.082)	(0.080)	(0.079)	(0.078)
Family SES during adolescence	-0.975***			
	(0.220)			
SES during young adulthood		-0.556*	-0.457†	-0.593*
		(0.267)	(0.263)	(0.270)
Psychosocial factors				
Perceived discrimination (never is reference)				
Rarely			0.208	
			(0.321)	
Sometimes			0.626	
			(0.514)	
Often			2.631***	
			(0.961)	
Social isolation (1=isolated)				-0.227
				(0.283)
Intercept	24.53***	28.47***	28.28***	28.68***

	(2.477)	(2.232)	(2.243)	(2.161)
Model Fit Statisics				
AIC	6574452	6567823	6562622	6567592
BIC	6574551	6567921	6562740	6567697
N (unweighted)	5,221	5,221	5,221	5,221

***p<0.001, **p<0.01, *p<0.05, †p<0.1

Note: all models are survey design adjusted and weighted

Figure 1. Theoretical Model