RACE, PLACE, AND HEALTH: HOW DOES NEIGHBORHOOD RACIAL/ETHNIC CONTEXT AFFECT HEALTH BEHAVIOR AND RISK FACTORS

by

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ABSTRACT

This dissertation builds upon current debates on the detrimental versus protective effects of racial/ethnic residential isolation and immigrant concentration on health in the United States (US), and empirically examines: (1) whether neighborhood-level ethnic density, racial diversity, and immigrant concentration are negatively or positively associated with health risks; (2) for whom and under what conditions are these residential patterns health-detrimental or health-protective; (3) what are the structural and psychosocial pathways underlying the detrimental or protective effects of these residential patterns; (4) whether neighborhood influences are susceptible to sample selection bias. Individual-level data from the 2003-2008 National Health and Nutrition Examination Survey and the 2006 and 2008 Southeastern Pennsylvania Household Health Survey were merged with census-tract profiles obtained from the 2005-2009 American Community Survey estimates, the 2000 Decennial Census and Geographic Information System-based built-environment data. Multilevel analysis, mediation analysis, and Propensity Score Matching method were performed to answer these research questions. Results largely confirmed the salient impact of neighborhood racial/ethnic context on individual health risks, while selection bias was also evident. Neighborhood racial isolation and ethnic concentration showed detrimental health effects, whereas racial diversity showed positive effects. The observed effect of immigrant concentration was likely due to neighborhood selection bias. Effect modification and

underlying pathways were complex and were dependent on the specific neighborhood contextual predictors.

I dedicate this dissertation to my parents.

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CHAPTER 1

INTRODUCTION

Health disparities and ecological research on population health are among the most critical fields of scholarship that is needed in the United States (US) given the manifested and debated issues of polarization by social groups, racial/ethnic relations, and the burden of healthcare. The annual healthcare cost constituted 18% of the total US economy in 2011 and is expected to reach over 20% within the next ten years (Center for Medicare & Medicaid Services). Despite its high expenditures on healthcare, the US has been questioned on why many of its health indicators at the population level, such as life expectancy, lag behind other developed countries or even pooper countries. It is believed that social polarization, such as racial segregation and income inequality, is playing a salient role as the US currently leads the developed world in terms of such issues. In particular, health disparities by race/ethnicity are of great concern. Studies have shown that many minority populations have poorer health-related outcomes compared to whites, yet such a seemingly evident argument is sometimes challenged by the observation of some groups' advantages in certain health indicators, with examples of the better health status of Mexican immigrants and lower mortality rates of Asian Americans. This attracts researchers to examine why inequalities in other social and economic domains easily transfer to people's life-long health trajectories, and how multilevel mechanisms

contribute to health disparities. Among them, neighborhood-level racial/ethnic context has attracted burgeoning academic attention.

Residential racial context has been particularly driven by race-based segregation patterns in the US. Residential segregation is usually referred to as the degree to which groups reside apart from each other in the urban environment (Massey and Denton 1988). Racial/ethnic residential segregation in the US is deeply rooted in its historical background. On the one hand, racial residential segregation as a manifestation of social polarization "has long been identified as the central determinant of the creation and perpetuation of racial inequalities in America" (Williams and Collins 2001:405). Blacks in general have experienced the highest segregation among all ethnic groups and the black-white segregation is usually referred to as hyper-segregation that reached to its highest level around the middle 20th century (Charles 2003). Since the civil rights movement, particularly the Civil Rights Act of 1968 that made housing and other institutional discrimination illegal, black-white segregation has been declining to some extent, but many of the most segregated regions have seen little or no change (Charles 2003; Iceland 2004).

On the other hand, influx of new immigrants since the 1960s has prompted more dynamic and distinctive ethnic settlement patterns (Logan, Wenquan, and Alba 2002). Along with this trend is the increasing concentration or isolation among Hispanics and Asians (Charles 2003; Iceland 2004; Logan, Stults, and Farley 2004), with the example of immigrant enclaves. Compared to the back-white segregation that is rooted more institutionally, some scholars see immigrant concentration as a result of social and cultural preferences, or "self-isolation" (Logan, Wenquan, and Alba 2002). Upon arrival, many of the new immigrants are constrained by social, cultural, and financial barriers,

and tend to cluster in co-ethnic or mixed minority neighborhoods in order to seek affordable housing, familiar culture, and social networks. Such dynamics may suggest divergent or even paradoxical consequences of residential patterns in terms of socioeconomic outcome and wellbeing in general.

The combination of the above issues has raised questions on whether and how living in places with one certain racial/ethnic group, with primarily co-ethnics, or of mixed racial/ethnic environment, may impact our health behaviors and health outcomes. This is the central inquiry of this dissertation project. The relationship between place context and health is an emerging and burgeoning topic in the fields of medical sociology and social epidemiology. The main argument in this literature is that where we live would affect our health status and health behaviors over and above individual risk factors through dynamic and complex mechanisms (Kawachi and Berkman 2003). This perspective on place context has been largely addressed by examining contextual-level (e.g., neighborhood-level) variations in socioeconomic characteristics such as percentage of residents with a college degree, percentage of female-headed household, or poverty concentration. Past research has also looked at whether and how racial segregation is associated with health in the traditional black-white discourse. Following this line of research documenting the detrimental effects of black-white segregation, Williams and Collins (2001) argue that racial residential segregation is a fundamental cause of racial disparities in health, because social and physical separations create institutional racism that operates through a wide array of channels. In recent years, however, another theoretical argument emerges in response to the protective effect of ethnic concentration on health, the so called "ethnic density effect" (Becares et al. 2012). The underlying assumption within this framework is that residing with co-ethnics may foster better social

cohesion, provide more health-promoting resources, and protect minorities or immigrants from racial discrimination and migration-related stress or language barriers.

Indeed, by looking at structural and psychosocial elements underlying residential patterns, we may better understand the mechanisms associated with multilevel determinants of individual health over and above individual risk factors in order to design appropriate public health interventions and to achieve healthy people goals. Moreover, it is important to look more into whether the observed contextual effects on health differ by social groups so that we can target high-risk populations.

Neighborhood Influences on Health

Place variations in health indicators are consistently observed across human societies. Scholars have been searching for explanations that can explicate why such spatial variations exist and persist and what can be done to reduce them and further prevent health risks for the general public. Macintyre et al. (2002) proposed three types of the place effect: compositional, contextual, and collective. *Compositional* effect is based on characteristics of individuals concentrated in particular places; that is, the observed spatial variations are merely due to aggregated characteristics of individuals. *Contextual* effect derives from opportunity structures in the local physical and social environment; it emphasizes variations of structural resources that are health-promoting or health-detrimental. *Collective* effect refers to socio-cultural and historical features of communities; it focuses on the importance of shared norms, values, traditions, and interests. One example Macintyre et al. (2002) offered is that that certain areas may witness poorer health than others because these areas consisted of more individuals whose personal characteristics, such as low individual SES, predispose them to smoking

(compositional). It may also be a result of more cigarette retail outlets, advertisements for cigarettes, and low-price cigarettes in these areas (contextual), or because local norms and attitudes are relatively pro-smoking (collective).

Neighborhoods constitute an important dimension of our living environment. The explanations of Macintyre et al. suggest that various physical, social, and cultural environments within residential neighborhoods can exert their influences independently, interactively, or jointly on individual health. A neighborhood approach is consistent with the concern about health inequalities because health risks and health outcomes not only often vary across neighborhoods, but neighborhoods are also strongly patterned by race/ethnicity and social position (Diez Roux and Mair 2010). To particularly address disparities in health-related behavioral and risk factors such as obesity, physical activity, and biomarkers, there is a growing interest in the neighborhood approach to apply ecological model to study impacts of physical environment (e.g., food environment, availability of recreational resources) and social environment (e.g., neighborhood SES, social cohesion) with the place of residence (Bird et al. 2010; Carroll-Scott et al. 2013; Durand et al. 2011; Long et al. 2010). Specific to health inequalities by race/ethnicity, examining the place effect provides the opportunity to investigate how multilevel mechanisms contribute to such disparities and what we can do to reduce them.

Despite an exponentially growing interest in studying racial residential segregation and ethnic density effect on health (Becares et al. 2012; Kramer and Hogue 2009), several limitations remain in the current literature. First, we do not know why neighborhood racial/ethnic context would impact individual behavior and health risk factors. Although a series of structural and psychosocial factors have been offered to explain this association, few studies have empirically tested these potential mediators.

Second, we do not know how individual and neighborhood characteristics may interact with the neighborhood racial/ethnic context in influencing individual health. Third, we do not know whether contextual effects of neighborhood on health found in observational studies are simply a result of the compositional effect. That is, existing evidence based on hierarchical linear modeling of cross-sectional data is still limited in disentangling the causation effect from the selection effect. There is need to utilize alternative analytical techniques in observational data such as propensity score matching to draw causal inference.

Theoretical Perspectives on Residential Patterns in Multi-ethnic America

To better understand consequences of a social phenomenon, we need to first understand its causes or antecedents. There may be divergent consequences of the same phenomenon (i.e., neighborhood racial/ethnic composition), according to different forces that help shape neighborhoods into segregated or integrated ones. Below I review three major theoretical perspectives regarding these forces.

Place Stratification

One major theory in the formation and perpetuation of racial residential segregation is the place stratification model, which contends racial residential segregation persists even when institutional racism is made illegal and when members of the minority groups achieve better socioeconomic status. Within this framework, race as an ascribed social status places primacy in sorting their members into residential neighborhoods attached to their groups' social standing in society, and restraining them from moving

into places comparable to whites (Alba and Logan 1993). According to the place stratification model, both individual and institutional prejudice and discrimination play central roles in constraining residential mobility options of the minorities and in maintaining racially separated neighborhoods (Charles 2003).

Evidence to date seems to suggest that the place stratification model is better at explaining the enduring black-white segregation in the US, which many thought had been eliminated after the Civil Rights Act of 1968. For example, by looking at black segregation levels by income in 30 metropolitan areas, Massey and Denton (1993:88) state that "the residential segregation of African Americans cannot be attributed in any meaningful way to the socioeconomic disadvantages they experience." Logan et al. (2004) analyze US census data to examine the changing segregation trend from 1980 to 2000, and show that although blacks in general have seen increasing income, communities with high percentages of blacks are still more segregated compared to others, and that those places see smaller deceases in segregation. Such evidence suggests that socioeconomic status as an explanatory variable indeed has limited power in black-white segregation. "On the relationship between racial residential segregation and socioeconomic status, simply put, there is no relationship between the two for black Americans" (Charles 2001:279).

Rejecting that it is the socioeconomic differences among racial groups that result in residential segregation, the place stratification model emphasizes race-based prejudice and discrimination, both individually and institutionally, beyond other salient factors. On the other way round, physical separation between racial groups inevitably perpetuates racial prejudice and discrimination. Such a vicious cycle not only results in deteriorated socioeconomic outcomes among the disadvantaged, but also serves as what Williams and

Collins (2001) call the fundamental cause of racial residential segregation on health outcomes. Because the place stratification model also puts emphasis on concentrated disadvantages in segregated neighborhoods, researchers largely draw on this model to examine how concentrated poverty and psychosocial distress of living in physically and socially disordered communities contribute to the detrimental effects of residential segregation. Past literature has mainly examined blacks in the US because of their unique history of residential experiences compared to other minority groups.

Spatial Assimilation

Spatial assimilation is a competing model that draws primarily on group differences in socioeconomic achievement in explaining racial residential segregation (Massey and Denton 1985). Within this framework, racial/ethnic minorities in the US, especially blacks and Latinos, have lower socioeconomic achievement measured by income level, educational attainment, and occupational status. Because of their relatively lower standing in the social hierarchy than whites, the minority populations are inevitably constrained in disadvantaged communities where many of their neighbors are also racial/ethnic minorities or their co-ethnics.

Contrary to the place stratification model that sees race as an ascribed status constraining the opportunities for residential mobility, spatial assimilation contends that as lower-status minorities gradually make socioeconomic achievement and become more assimilated into the mainstream US society, they are able to move up the social ladder and move out of the segregated and disadvantaged neighborhoods. Thus the spatial assimilation model allows the opportunities for residential mobility among racial and ethnic minorities. This opportunity for residential mobility, or *assimilation* process, not

only applies to native minorities, but also to the vast number of immigrants arriving in the US every year. Many immigrants are similar to US-born minority populations in that they have relatively few socioeconomic resources and therefore reside in neighborhoods that are usually considered segregated and disadvantaged. Moreover, when applying spatial assimilation theory to immigrants' residential mobility, researchers are not only concerned with the sole indicator of socioeconomic achievement, but also with *acculturation*, operationalized as accumulative duration of residence and English proficiency (Berry 1997; Charles 2003), because cultural aspects are also crucial factors to determine whether an immigrant lives in an integrated neighborhood or in her/his ethnic enclaves.

The segregation literature shows that the spatial assimilation model is better applied to Latinos and Asians in the US as substantial residential gains are achieved by improved socioeconomic status for these two groups (Alba et al. 1999; Iceland and Scopilliti 2008; Logan, Alba, and Leung 1996; Wahl, Breckenridge, and Gunkel 2007). In particular, when taking into account the effects of education and income, there is ample evidence to show that native-born Latinos and Asians are very likely to live in communities that are comparable to those of whites (Charles 2003). However among blacks, although a positive association between socioeconomic status and residential outcomes is also discernible (Logan et al. 2004), such residential achievement is counterbalanced by living with whites of lower socioeconomic status than their own (Alba, Logan, and Stults 2000).

Resurgent Ethnicity

The spatial assimilation model is limited in that, similar to the classical assimilation theory in general, it treats assimilation as a linear or one-directional process--assimilation to whites--and ignores the increasing sociocultural diversity in the US. The conventional indicators of assimilation in the US society, such as English proficiency and moving into suburban and whiter areas, have also been shown to have weakening effects. After analyzing the suburbanization patterns of eleven racial/ethnic groups including those growing rapidly through immigration, Alba and colleagues (1999:458) conclude that there is "a decline in the significance of suburbanization as a distinct stage in a larger process of residential assimilation" as well as "the weakening of the correspondence between suburbanization and linguistic assimilation." Thus there may be emerging residential patterns that are not predicted by the spatial assimilation model given the complexities and dynamics of this multi-ethnic society.

A new theoretical framework has been proposed in response to the recent patterns of minority concentration, the so-called resurgent ethnicity (Walton 2012; Wen, Lauderdale, and Kandula 2009). Different from the place stratification model contending that racism perpetuates segregation, or the spatial assimilation model contending that ethnic enclaves are only the initial stage before moving out of such communities, the resurgent ethnicity model asserts that ethnic groups willingly choose to live with their co-ethnics as self-preference or self-segregation, even after they become financially secured and can afford moving into "white neighborhood." Logan and colleagues (2002) use the term *ethnic communities* to refer to this pattern, as distinct from the traditional *ethnic enclaves*, suggesting that ethnic communities are "grounded in motives associated more with taste and preference than with economic necessity, or even with the ambition to

create neighborhoods that will symbolize and sustain ethnic identity" (Logan et al. 2002:300).

Research on residential choices and residential patterns among recent immigrants has provided supporting evidence for the resurgent ethnicity model. For example, in the spatial assimilation model, moving into suburban areas with more whites has been considered as an indicator of residential mobility, particularly for minorities living in inner cities. However, rather than a suburbanization process with eliminated segregation, it is shown that ethnic groups have been establishing suburban communities of ethnic concentration (Alba et al. 1999). This emerging pattern is most visible among middle-class Asian communities, but is also discernible, albeit to a lesser extent, among Latinos and blacks (Wen et al. 2009). Because socioeconomic structures and characteristics of such ethnic communities are very distinct from traditional ethnic enclaves, their health implications for residents living in such communities may considerably differ as well. A small but growing literature has started to pay attention to this issue (Becares et al. 2012; Kramer and Hogue 2009; White and Borrell 2011).

Divergent Consequences of the Place Context

The above dynamic forces driving racial/ethnic residential patterns thus imply divergent consequences for residents living in those communities, including their implications on individual health. Place stratification model would predict detrimental health effects of residential segregation on residents, whereas ethnic resurgence model might suggest protective effects of living with co-ethnics. As for spatial assimilation model, it could have mixed predictions depending on where an individual or family is in the assimilation continuum. Overall, one should expect to see different impacts of

residential racial/ethnic contexts, and what matters is which angle of the place context we look into.

Study Aims

This dissertation will empirically investigate: (1) whether neighborhood-level racial/ethnic density or diversity and immigrant concentration are negatively or positively associated with health-related outcomes; (2) for whom and under what conditions are these residential patterns health-detrimental or health-protective; in other words, whether there are differential effects for different groups by age, sex, race/ethnicity, urban status, and neighborhood SES; (3) what are the structural and psychosocial factors that serve as mechanisms or pathways underlying the detrimental or protective effects of these residential patterns; (4) whether neighborhood influences are susceptible to sample selection bias. I will combine these research questions by specifically examining two health-related outcomes among US adults in three empirical analyses: obesity and biological risk profiles including metabolic syndrome.

Organization of This Dissertation

Three empirical analyses are presented in Chapter 2, Chapter 3, and Chapter 4, respectively. Theoretical frameworks and literature review on health consequences of neighborhood racial/ethnic context specific to each research question are discussed in each of these three empirical chapters.

Chapter 2 presents the first empirical analysis using data from the 2006 and 2008 Southeastern Pennsylvania (SEPA) Household Health Survey to study the association between black concentration and obesity among blacks and whites. Overweight and

obesity are risk factors for a wide array of health problems such as diabetes, cardiovascular diseases, and cancer. The prevalence of obesity has been rising among major segments of the US population for a long period of time over the past decades (Mokdad 2003) and racial disparities in obesity persist (Wang and Beydoun 2007). The specific aim of this study is to apply the ecological model to examine whether neighborhood social and built-environmental attributes (i.e., social cohesion, neighborhood SES, street connectivity, and park accessibility) serve as pathways in the link between black concentration and obesity risks.

In Chapter 3, I investigate the association between neighborhood racial/ethnic diversity and health risks, specifically metabolic syndrome, among US adults. The application of biomarker data to the study of social determinants of health is one of the crosscutting issues that examines how physiologic functioning and social environment factors interact to produce health risks (Shin, Fernandes, and Bird 2010). Specific to health disparities by race and ethnicity, incorporating biomarker data provides the opportunity to study how the construct of race and ethnicity "get under skin" through exposure to psychosocial stressors such as racial discrimination and migration-related stress. Using 2003-2008 National Health and Nutrition Examination Survey (NHANES) data, this analysis will add to the literature by looking at the independent role of ethnic heterogeneity, an understudied measure of residential multigroup composition. It also extends previous research by specifically focusing on effect modification in the neighborhood-health association and examines how the independent effects of racial diversity differ by sex, age groups, urban status, and neighborhood poverty.

Chapter 4 takes data from the 2006 and 2008 Southeastern Pennsylvania (SEPA) Household Health Survey and empirically examines racial/ethnic isolation and immigrant

concentration as two distinct domains of residential patterns that can have divergent implications. Given the high fertility trend and projected population growth of Hispanics in the US, research on environmental influences on Hispanic Americans and Hispanic immigrants will provide much-needed empirical evidence that helps design intervention programs and reduces health risks for America's future. More importantly, this analysis directly addresses the problem of sample selection bias by utilizing the Propensity Score Matching method to correct for potential situations of structural confounding. This approach will help achieve better causal inference in the neighborhood and health link.

CHAPTER 2

RACIAL CONCENTRATION AND BLACK-WHITE OBESITY RISKS: IDENTIFYING POTENTIAL PATHWAYS

Background

The alarming obesity prevalence in the US has triggered increasing public health attention to the population distribution and contributory factors of this epidemic in an effort to prevent related health problems such as diabetes and cardiovascular diseases (Mokdad 2003). The prevalence of obesity has been rising among major segments of the US population for a long period of time over the past decades (Mokdad 2003). Although recent evidence has suggested stagnation of this rising trend in recent years (Ogden et al. 2014), overall obesity prevalence in the US remains very high and racial disparities in obesity persist (Wang and Beydoun 2007). National survey data reveal that female obesity prevalence has risen from 21.2% to 30.0% among whites and from 30.2% to 54.0% among blacks from the years 1988-1994 to 2003-2004 (Ogden et al. 2006). Individual risk factors such as socioeconomic status (SES) are shown to affect black and white adults differentially (Griffith et al. 2011), and are limited in explaining black–white disparities in obesity (Bleich et al. 2010). Beyond individual characteristics, a burgeoning line of research is now shifting to an ecological approach looking at community-level

environmental influences on individual obesity risks and disparities by gender and race/ethnicity (Wen and Kowaleski-Jones 2012; Wen and Maloney 2011).

Neighborhood Racial Composition and Obesity

Residential racial composition emerges as a salient neighborhood factor of residents' health and lifestyle given the projected population growth among racial/ethnic minorities and persistent residential segregation in the US. Yet the picture is far from clear in terms of how and why neighborhood-level racial/ethnic composition may play a role in contributing to individual-level obesity risks and disparities. Williams and Collins posit that racial segregation is a fundamental cause of black–white disparities in health, mostly because institutional and personal racism disproportionately places members of minority groups into concentrated disadvantages (Williams and Collins 2001). Most recently, however, another theoretical framework has been established for the protective effects of minority concentration on health, the so-called ethnic density effects (Becares et al. 2012; Pickett and Wilkinson 2008). The underlying assumption within this framework is that residing with co-ethnics may foster better social cohesion, provide more health-promoting resources, and protect minorities from discrimination and related stress. These two competing frameworks have shed light on the dynamic pillars pertaining to US communities and have both received supporting evidence across a range of health outcomes (Becares et al. 2012; Kramer and Hogue 2009).

This complexity is also reflected in the mixed findings in empirical work examining black concentration and weight status. Although researchers have generally agreed on its significant role, black concentration has received less attention compared to other contextual factors. In many cases, it is either considered as a control variable or as

an indicator of socioeconomic disadvantage in the neighborhood environment. In the research that has focused on the independent effect of black concentration, two studies report that living in black neighborhoods is associated with higher obesity risks, even after adjusting for neighborhood socioeconomic characteristics (Boardman et al. 2005; Chang, Hillier, and Mehta 2009), and other research in New York City finds no effect in this association (Black et al. 2010). These studies, unfortunately, ignore the fact that living in black neighborhoods encompasses substantially different meanings and experiences for different racial groups, limiting the insight into independent effects of race. When community racial composition and individual race/ethnicity are jointly considered, three nationwide multilevel studies reported null findings in the relation between percentage black and body mass index (BMI) among blacks (Do et al. 2007; Kirby et al. 2012; Robert and Reither 2004). The effects of neighborhood minority concentration for whites remain largely untested. It is increasingly recognized that segregation patterns in the US not only affect the minority populations, but also influence whites in meaningful and distinct ways.

The Mediating Role of Neighborhood Social and Built Environment

Community-resilient features such as socioeconomic resources and social cohesion benefit individual health regardless of individual race (Kawachi, Subramanian, and Kim 2008). However, area deprivation in association with racial segregation may sort whites with lower socioeconomic status into minority and deprived neighborhoods leading to their increased health risks, including obesity. Among residents of black-concentrated neighborhoods, whites are likely to be poorer than blacks (Alba et al. 2000) and may suffer from heightened levels of relative deprivation, which can exert additional

detrimental effects on health net of absolute deprivation (Wen and Christakis 2005). From the perspectives of the social disorganization theory, minority concentration may strengthen social cohesion for minority residents but may erode social cohesion for white residents. For instance, evidence suggests that living with co-ethnics fosters better social cohesion (Becares et al. 2011) and that residing in black-concentrated neighborhoods protects blacks, buffering against health risks (Hutchinson et al. 2009). Considering these complex circumstances, it is possible that the health impact of living in minority-concentrated communities is ambivalent for minorities but detrimental for whites. Indeed, a recent study shows that the positive association between living in neighborhoods with a high Hispanic concentration (≥25%) and the odds for obesity was slightly greater for whites than for Hispanics (Kirby et al. 2012). More studies are needed to explore this pattern and to examine whether this association differs by gender.

Moreover, neighborhood built environment, defined as human-formed, developed, or structured areas (Centers for Disease Control and Prevention 2005), is well recognized as another important dimension of the community facilitating or obstructing active living. Neighborhood features such as street connectivity and presence of exercise facilities exert influence on residents' health-promoting behaviors as well as weight status (Feng et al. 2010), yet such physical environment resources are not equally distributed in the US and their effects differ across racial groups (Duncan et al. 2013; Lovasi et al. 2009; Wen et al. 2013). Regarding the link between neighborhood racial composition and obesity, it remains an empirical question whether neighborhood built environment serves as mediating or suppressing pathways and whether their effects differ by socio-demographic groups.

The above mediating pathways linking neighborhood racial composition and obesity can be visualized in a conceptual model presented in Figure 2.1.

Study Aims

To address these gaps in the literature, this chapter specifically focuses on neighborhood black concentration and its association with residents' obesity risks and has two aims: (1) to test whether neighborhood black concentration is positively or negatively associated with obesity risks, net of individual characteristics; (2) to explore what roles neighborhood social and built-environmental attributes, particularly social cohesion, SES, street connectivity, and park access, play in the link between black concentrations and obesity risks. Because theoretical and empirical evidence suggests that neighborhood

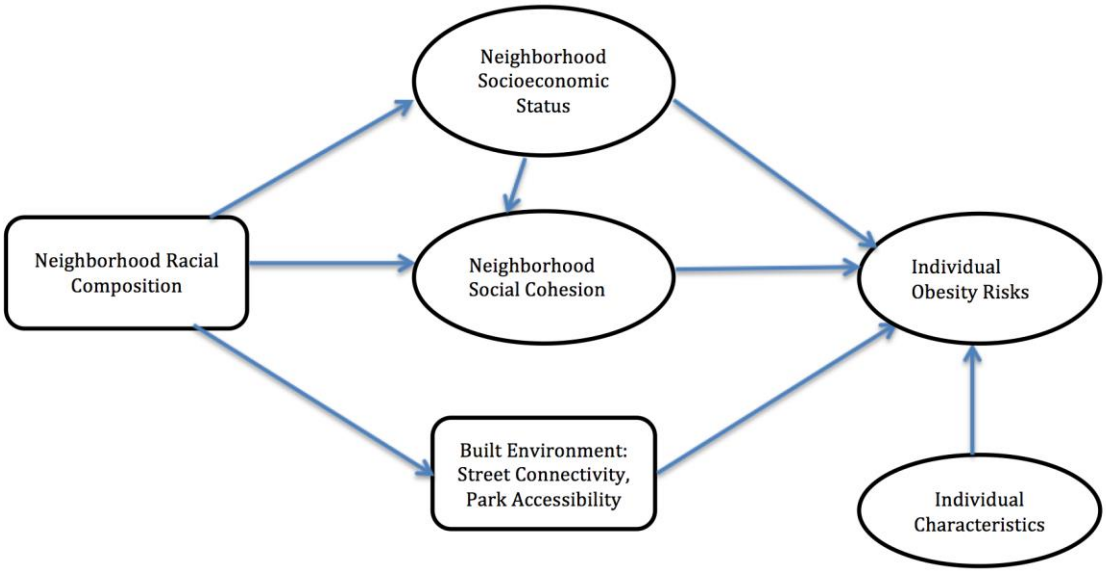

FIGURE 2.1

A conceptual model displaying neighborhood social and built environment as pathways linking neighborhood racial composition and obesity.

effect on individuals depend on gender, with women spending more time at home and more likely to be influenced by community social environment (Kershaw, Albrecht, and Carnethon 2013; Wen and Zhang 2009), I conduct analysis by specific gender and race. Looking at moderating and mediating effects pertinent to multilevel facets of racial composition would presumably advance our understanding of individual–environment interactions and the processes underlying health risks.

Methods

Data

This study is based on pooled data from the 2006 and 2008 Southeastern Pennsylvania (SEPA) Household Health Survey administrated by the Public Health Management Corporation in Bucks, Chester, Delaware, Montgomery, and Philadelphia counties. This biennial cross-sectional survey drew a stratified probability sample from 54 service areas where each had about 30,000 to 75,000 adult residents, and was conducted through telephone interviews with people aged 18 and older. One eligible adult respondent was chosen from each household based on selection criteria. People aged 60 and older were over-sampled for the purpose of asking specific questions to this age group. Self-reported person-level data from the SEPA Household Health Survey were then linked to census-tract profiles obtained from 2005-2009 American Community Survey (ACS), US Census, and Geographic Information System (GIS) built-environment data. After excluding tracts with only 1 residents (N = 13) and respondents who had missing values on weight status or any of the predictors (N = 557), the final analytical sample included 12,730 whites and 4,290 blacks residing in 953 tracts, with an average of 21 residents per tract (SD: 11; range: 2-196). The SEPA Household Health Survey data

are particularly suitable for this study due to the relatively high level of black isolation in the Philadelphia metropolitan area and also because the survey asked respondents a range of questions pertaining to neighborhood social cohesion.

Individual-level Measures

The outcome variable was a dichotomous indicator of being obese if a respondent had a BMI equal to or higher than 30.[1] BMI was calculated based on self-reported height and weight following the formula BMI = weight(kg)/height(meters)^2. Individual covariates included self-reported race (white vs. black), gender (male vs. female), age, marital status (married/living with partner vs. single/separated/divorced/widowed), nativity (US born vs. foreign born), educational attainment (high school or below, some college, and college or above), income (below 100% federal poverty line, 100%-200% federal poverty line, and at or above 200% federal poverty line), smoking status (currently a smoker or not), and survey year (2006 vs. 2008). I also included an age-squared term in the models to account for curvilinear relationship between age and obesity.

Neighborhood-level Measures

Percentage black was obtained from ACS 2005-2009 data and was a measure of black concentration in each census tract (ranging from 0 to 1). The original continuous measure was then dichotomized as whether a census tract had 25% or more black

[1] I recognize that BMI as a measure of obesity has its own limitation. Because the calculation is solely based on a person's height and weight, BMI is not able to distinguish body fat from fat-free mass. Thus individuals who have high BMI may be high in muscle and bone, but not in fat. Evidence has shown that BMI in general is less valid classifying men than women (Burkhauser and Cawley 2008).

residents (Kirby et al. 2012; Wen et al. 2009). Racial/ethnic concentration as a proxy measure of residential isolation has often been used in prior studies (Borrell et al. 2013; Chang et al. 2009; Salinas et al. 2012; Wen and Maloney 2011), and also allows the examination of health risks or benefits of living with co-ethnics or with certain groups (White and Borrell 2011).

Taking into account neighborhood SES is essential in detangling ethnic density effects from area deprivation associated with residential segregation (Becares et al. 2012; Roy, Hughes, and Yoshikawa 2012). I used information on tract-level percentage of college graduates, percentage of unemployed residents, percentage of residents living in poverty, and percentage of households with annual income $75,000 or above from ACS 2005-2009 data, and performed principle-component factor analysis to construct a summary scale based on the above four items (ranging from -3.20 to 1.73; alpha = 0.82).

Neighborhood social cohesion was an aggregated summary measure based on the following three questions in the SEPA Household Health Survey: (1) “Please rate how likely people in your neighborhood are willing to help their neighbors with routine activities such as picking up their trash cans, or helping to shovel snow. Would you say that most people in your neighborhood are always, often, sometimes, rarely, or never willing to help their neighbors?” (2) “Please tell me if you strongly agree, agree, disagree, or strongly disagree with the following statement: I feel that I belong and am a part of my neighborhood.” (3) “Please tell me if you strongly agree, agree, disagree or strongly disagree with the following statement: Most people in my neighborhood can be trusted.” I reversed the item responses and created a summary score of perceived social cohesion for each respondent using principle component factor analysis (alpha = 0.66), with higher

value indicating more social cohesion. I then aggregated this score to each census tract based on mean response (ranging from -1.92 to 1.47).

GIS-based objective measures of neighborhood built environment consisted of street connectivity and park accessibility. Street connectivity was measured by the number of intersections per square mile in each census tract (ranging from 8.79 to 1,071.43) and spatial park accessibility was measured by weighted distance (in miles) from the neighborhood centroid to the nearest seven parks (ranging from 0.22 to 8.92) (Wang, Wen, and Xu 2013; Zhang, Lu, and Holt 2011). Finally, tract-level percentage of residents living in the same house in the year 1995 was obtained from the 2000 US Census and is used as a covariate in the analysis to capture residential stability (ranging from 0.05 to 0.92).

Statistical Analysis

I used multilevel random intercept logistic regression models to account for the clustering nature of the data, where individuals are nested within census tracts. Weighted group-specific analyses were conducted to examine contextual effects of black concentration on obesity risks separately for white women, white men, black women, and black men. In each set of the stratified analysis, I first examined the crude effect of black concentration on obesity risks while adjusting for individual-level covariates (Model 1). In Model 2, I included social cohesion, one indicator of neighborhood social environment, to assess whether it served as a pathway for black concentration. Then in Model 3, neighborhood SES was added to see whether the observed association between black concentration and obesity was attributable to area deprivation and socioeconomic disadvantage. Finally, I added the two built-environment measures of street connectivity

and park accessibility as potential mediators (Model 4). Results were reported in odds ratios with 95% confidence intervals. I incorporated individual sampling weights to account for study design and sampling selection bias and performed our analyses in Stata 11.2 and M*plus* 7.11.

Following multivariate regression analysis, I performed formal multilevel mediation analysis to assess the effect that each hypothesized mediator had in attenuating the association between black concentration and individual obesity. My approach followed a single 2→2→1 mediation model, where a level-2 mediator was examined in the relation between a level-2 predictor and a level-1 outcome (Krull and MacKinnon 2001). First, a single-level model involving only neighborhood variables was specified to obtain the coefficient between black concentration and each potential mediator (a-path). Second, a multilevel random intercept model was specified to examine each mediator and individual obesity risks (b-path), adjusting for black concentration (c'-path). Mediated effects were computed by multiplying coefficients for the a- and b-paths (β_a*β_b); standard errors were calculated using the first-order Taylor series expression; and the Sobel test was used to test for the significance of mediated effects.

Results

Descriptive Statistics

Unweighted descriptive statistics are presented in Table 2.1, stratified by race and gender. Consistent with the national trend, obesity prevalence was much lower among whites than it was among blacks, and this gap was even larger for women. The sample consisted of more women than men, and black respondents on average were younger than whites were. The majority of whites were married or living with a partner, whereas the

TABLE 2.1

Unweighted sample characteristics (SEPA Survey 2006 and 2008)

Individual-level Measures	**White Women**	**White Men**	**Black Women**	**Black Men**
Obese	20.74%	24.06%	40.79%	31.27%
Age	54.57 (0.18)	53.65 (0.24)	49.28 (0.30)	50.52 (0.48)
Married / living with partner	58.74%	66.78%	30.33%	45.27%
US born	96.40%	96.12%	95.07%	92.35%
Educational attainment				
High school or below	37.10%	31.36%	52.50%	54.90%
Some college	19.97%	18.75%	24.73%	22.51%
College or above	42.94%	49.89%	22.78%	22.59%
Income				
< 100% FPL	4.54%	3.04%	20.03%	13.14%
100%-200% FPL	13.33%	9.61%	27.29%	24.91%
≥ 200% FPL	82.14%	87.35%	52.69%	61.94%
Current smoker	18.21%	18.66%	25.56%	28.18%
Survey year 2008	50.78%	49.51%	51.89%	55.24%
Sample Size (N)	**8,224**	**4,506**	**3,126**	**1,164**
Neighborhood-level Measures				
Percent black ≥ 25%	20.75%	19.05%	46.29%	55.28%
Social cohesion	0.06 (0.01)	0.06 (0.01)	-0.15 (0.02)	-0.18 (0.02)
Socioeconomic status	0.15 (0.03)	0.20 (0.03)	-0.37 (0.04)	-0.52 (0.05)
Street connectivity	166.51 (4.96)	163.03 (5.06)	227.00 (6.59)	243.40 (7.63)
Park accessibility	1.59 (0.04)	1.62 (0.04)	1.16 (0.04)	1.10 (0.04)
Residential stability	0.58 (0.00)	0.58 (0.00)	0.57 (0.01)	0.57 (0.01)
Number of Tracts	**853**	**819**	**553**	**407**

Note. Data shown are percentage or mean (standard deviation).

majority of blacks were not married. SES achievement gap by race was apparent in terms of education and income. Nearly half of whites had a college degree, more than twice as many as blacks. In contrast, many more blacks lived below the federal poverty line compared to only a few whites. At the neighborhood level, black concentration was noticeable as about one half of blacks' neighborhoods were composed of 25% or more black residents, but the corresponding number was about one fifth for whites. Neighborhood SES and social cohesion were both higher among whites than blacks. But blacks seemed to live in neighborhoods with better street connectivity and shorter distances to parks. Bivariate associations between neighborhood variables are presented in Table 2.2.

Multivariate Regression Analysis

Table 2.3 presents results from the multilevel random intercept logistic regression models for whites. Among white women, Model 1 indicates that black concentration was associated with higher odds of being obese (OR = 1.43, $p < 0.001$). But this association became weaker (OR = 1.28, $p < 0.05$) when neighborhood social cohesion was added in

TABLE 2.2

Correlation matrix between neighborhood-level variable

	(1)	**(2)**	**(3)**	**(4)**	**(5)**
(1) Percent black ≥ 25%	1.000				
(2) Social cohesion	-0.512	1.000			
(3) Socioeconomic status	-0.635	0.657	1.000		
(4) Street connectivity	0.400	-0.439	-0.565	1.000	
(5) Park accessibility	-0.365	0.376	0.404	-0.495	1.000
(6) Residential stability	-0.045	0.128	0.002	-0.130	0.059

TABLE 2.3
Odds ratio from multilevel logistic regression predicting obesity for white women and men

	White Women (N=8,224)				**White Men** (N=4,506)			
Neighborhood-level Measures	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 1**	**Model 2**	**Model 3**	**Model 4**
Percent black ≥ 25%	1.43*** [1.17-1.75]	1.28* [1.02-1.60]	1.09 [0.86-1.36]	1.09 [0.87-1.38]	0.89 [0.66-1.20]	0.74^{+} [0.53-1.03]	0.64* [0.45-0.92]	0.65* [0.46-0.93]
Social cohesion		0.77* [0.62-0.97]	1.11 [0.86-1.44]	1.08 [0.83-1.41]		0.67** [0.50-0.89]	0.93 [0.66-1.29]	0.89 [0.63-1.24]
Socioeconomic status			0.74*** [0.66-0.83]	0.72*** [0.64-0.81]			0.75*** [0.64- 0.88]	0.69*** [0.58-0.81]
Street connectivity				1.00 [1.00-1.00]				0.99** [0.99-1.00]
Park accessibility				1.02 [0.96-1.09]				0.99 [0.91-1.08]
Residential stability	1.33 [0.64-2.75]	1.48 [0.71-3.1]	1.18 [0.57-2.40]	1.12 [0.53-2.33]	1.90 [0.76-4.77]	2.25^{+} [0.88-5.73]	1.83 [0.72-4.67]	1.41 [0.54-3.66]
Individual-level Measures								
Age	1.11*** [1.08-1.14]	1.11*** [1.08-1.14]	1.11*** [1.08-1.14]	1.11*** [1.08-1.14]	1.14*** [1.10-1.18]	1.14*** [1.10-1.18]	1.14*** [1.10-1.18]	1.14*** [1.10-1.18]
Age squared	1.00*** [1.00-1.00]	1.00*** [1.00- 1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]
Married	0.69*** [0.60-0.79]	0.70*** [0.60-0.80]	0.71*** [0.61-0.81]	0.70*** [0.61-0.81]	1.03 [0.92-1.16]	0.97 [0.80-1.17]	0.99 [0.82-1.20]	0.98 [0.81-1.18]

TABLE 2.3 Continued

	White Women (N=8,224)				**White Men** (N=4,506)			
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 1**	**Model 2**	**Model 3**	**Model 4**
US born	1.72**	1.73**	1.71**	1.71**	1.10	1.11	1.12	1.13
	[1.20-2.48]	[1.20-2.50]	[1.19-2.48]	[1.18-2.47]	[0.72-1.68]	[0.73-1.70]	[0.73-1.72]	[0.74-1.72]
Education[a]	0.73***	0.74***	0.77***	0.77***	0.79***	0.80***	0.83***	0.84***
	[0.67-0.79]	[0.68- 0.80]	[0.70-0.83]	[0.70-0.84]	[0.72-0.87]	[0.73-0.88]	[0.75-0.92]	[0.76-0.92]
Income[b]	0.77***	0.78***	0.80**	0.80**	0.90	0.92	0.95	0.93
	[0.68-0.87]	[0.69-0.89]	[0.70-0.91]	[0.70-0.91]	[0.75-1.07]	[0.77-1.10]	[0.79-1.13]	[0.78-1.12]
Current smoker	0.69***	0.68***	0.68***	0.68***	0.73**	0.72**	0.71**	0.71**
	[0.57-0.83]	[0.57-0.82]	[0.56-0.82]	[0.56-0.82]	[0.58-0.91]	[0.58-0.91]	[0.57-0.89]	[0.57-0.89]
Year 2008	1.00	1.00	0.99	0.99	1.19*	1.18*	1.18*	1.18*
	[0.88-1.13]	[0.88-1.13]	[0.87-1.12]	[0.87-1.12]	[1.01-1.39]	[1.00-1.38]	[1.01-1.39]	[1.00-1.38]
Level 2 variance	0.20***	0.20***	0.17***	0.17***	0.35***	0.33***	0.30***	0.28***
Intraclass correlation	0.06	0.06	0.05	0.05	0.11	0.10	0.09	0.08
AIC	7881.35	7877.96	7855.43	7857.40	4828.89	4822.07	4810.86	4804.41
BIC	7965.53	7969.15	7953.64	7969.64	4905.85	4905.44	4900.65	4907.02

Note. 95% Confidence Intervals are in parentheses.

a. Education is treated as a continuous variable in the models. It has three levels: "high school or below," "some college," "college or above."

b. Income is treated as a continuous variable in the models. It has three levels: "below 100% FPL," "100-200% FPL," "at or above 200% FPL."

***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

Model 2 (OR = 0.77, $p < 0.05$). In Model 3, this association seemed to be explained away by area deprivation as adding neighborhood SES rendered the effect of black concentration insignificant. Neighborhood SES was a significant predictor that was negatively associated with obesity risks (OR = 0.74, $p < 0.001$). In Model 4, none of the neighborhood built-environmental variables were significant for white women.

The second part of Table 2.3 shows model estimates for white men. Model 1 shows no significant relationship of black concentration and odds of obesity for white men, but neighborhood social cohesion was a significant and negative correlate of obesity in Model 2 (OR = 0.67, $p < 0.01$). Black concentration was negatively linked to obesity (OR = 0.64, $p < 0.05$) only when neighborhood SES was controlled for, which was a negative predictor itself (OR = 0.75, $p < 0.001$) (Model 3). The effect of black concentration remained stable when the two built-environmental measures were added in Model 4. Notably, street connectivity was significantly and negatively associated with obesity among white men (OR = 0.99, $p < 0.01$). The analyses did not reveal significant effects of black concentration for blacks, as shown in Table 2.4.

Mediation Analysis

Based on the patterns shown in the multivariate regression, I performed formal multilevel mediation analysis among white women to assess the mediated effect of each hypothesized neighborhood-level mediator. Figure 2.2 and Figure 2.3 present results from single mediation models for neighborhood social cohesion and neighborhood SES, respectively. Figure 2.2 shows that black concentration was significantly associated with neighborhood social cohesion ($\beta a = -0.46$, SE = 0.03, $p < 0.001$), which was significantly associated with obesity itself ($\beta b = -0.26$, SE = 0.11, $p < 0.05$). The association between

TABLE 2.4
Odds ratio from multilevel logistic regression predicting obesity for black women and men.

	Black Women (N=3,126)				**Black Men** (N=1,164)			
Neighborhood-level Measures	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 1**	**Model 2**	**Model 3**	**Model 4**
Percent black ≥ 25%	1.10 [0.89-1.37]	1.03 [0.76-1.29]	0.95 [0.74-1.22]	0.93 [0.72-1.20]	0.90 [0.64-1.27]	0.87 [0.60-1.25]	0.87 [0.59-1.28]	0.95 [0.63-1.43]
Social cohesion		0.77^{+} [0.59-1.00]	0.92 [0.66-1.27]	0.93 [0.67-1.29]		0.92 [0.60-1.41]	0.91 [0.53-1.56]	0.87 [0.51-1.50]
Socioeconomic status			0.88^{+} [0.76-1.01]	0.90 [0.77-1.05]			1.01 [0.79-1.28]	1.01 [0.78-1.29]
Street connectivity				1.00 [1.00-1.00]				1.00 [1.00-1.00]
Park accessibility				0.93 [0.77-1.13]				1.21 [0.95-1.56]
Residential stability	0.47^{+} [0.20-1.10]	0.53 [0.23-1.24]	0.44^{+} [0.18-1.08]	0.45^{+} [0.19-1.16]	1.42 [0.37-5.44]	1.51 [0.40-5.72]	1.53 [0.41-5.77]	1.32 [0.35-5.00]
Individual-level Measures								
Age	1.14*** [1.10-1.17]	1.14*** [1.10-1.17]	1.14*** [1.10-1.17]	1.14*** [1.10-1.17]	1.17*** [1.11-1.24]	1.17*** [1.11-1.24]	1.17*** [1.11-1.24]	1.17*** [1.11-1.24]
Age squared	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]	1.00*** [1.00-1.00]
Married	0.79* [0.66-0.96]	0.79* [0.66-0.96]	0.79* [0.66-0.96]	0.80* [0.66-0.97]	0.90 [0.75-1.08]	1.07 [0.79-1.46]	1.07 [0.78-1.46]	1.08 [0.79-1.47]

TABLE 2.4 Continued

	Black Women (N=3,126)				**Black Men** (N=1,164)			
	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 1**	**Model 2**	**Model 3**	**Model 4**
US born	1.86**	1.84**	1.82**	1.81**	2.52**	2.47**	2.48**	2.48**
	[1.25-2.76]	[1.24-2.74]	[1.22-2.71]	[1.21-2.69]	[1.30-4.89]	[1.28-4.78]	[1.28-4.80]	[1.28-4.78]
Education[a]	0.78***	0.79***	0.80***	0.80***	0.85^{+}	0.86^{+}	0.86^{+}	0.86^{+}
	[0.69-0.87]	[0.71-0.88]	[0.71-0.89]	[0.71-0.90]	[0.71-1.02]	[0.72-1.03]	[0.71-1.03]	[0.72-1.03]
Income[b]	0.80***	0.81***	0.81**	0.81**	1.22^{+}	1.24^{+}	1.24^{+}	1.23^{+}
	[0.71-0.90]	[0.71-0.91]	[0.72-0.91]	[0.72-0.92]	[0.97-1.53]	[0.99-1.55]	[0.99-1.56]	[0.99-1.55]
Current smoker	0.67***	0.67***	0.67***	0.66***	0.55**	0.55***	0.55**	0.55**
	[0.55-0.82]	[0.55-0.82]	[0.55-0.81]	[0.54-0.81]	[0.39-0.77]	[0.39-0.77]	[0.39-0.77]	[0.39-0.78]
Year 2008	0.91	0.91	0.92	0.92	1.02	1.03	1.03	1.01
	[0.77-1.08]	[0.77-1.09]	[0.77-1.09]	[0.77-1.09]	[0.76-1.37]	[0.77-1.38]	[0.77-1.38]	[0.76-1.36]
Level 2 variance	0.17***	0.17**	0.18**	0.17**	0.22	0.22	0.22	0.19
Intraclass correlation	0.05	0.05	0.05	0.05	0.07	0.07	0.07	0.06
AIC	4042.83	4041.40	4039.42	4042.17	1376.37	1378.25	1380.25	1381.66
BIC	4115.40	4120.02	4124.08	4138.93	1437.09	1444.02	1451.08	1462.62

Note. 95% Confidence Intervals are in parentheses.

a. Education is treated as a continuous variable in the models. It has three levels: "high school or below," "some college," "college or above."

b. Income is treated as a continuous variable in the models. It has three levels: "below 100% FPL," "100-200% FPL," "at or above 200% FPL."

***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

black concentration and obesity remained significant even when the mediator of social cohesion was controlled for (βc' = 0.25, SE = 0.11, $p < 0.05$). The mediated effect of social cohesion was also statistically significant (βa*βb = 0.12, SE = 0.05, $p < 0.05$).

In Figure 2.3, black concentration was significantly associated with neighborhood SES (βa = -1.26, SE = 0.06, $p < 0.001$), and neighborhood SES was also significantly associated with obesity (βb = -0.27, SE = 0.05, $p < 0.001$). However, the association between black concentration and obesity was no longer significant when neighborhood

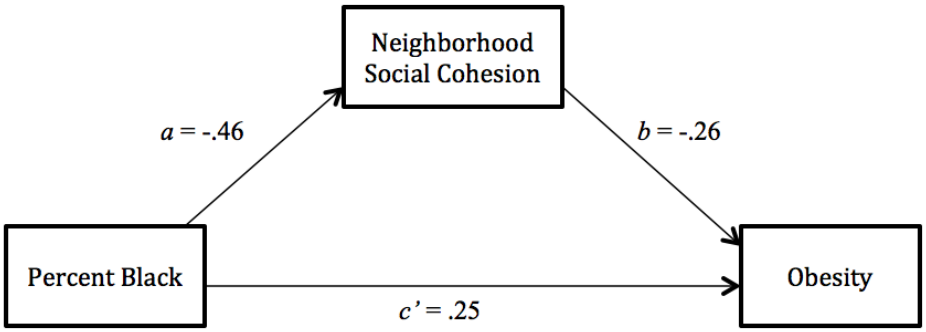

FIGURE 2.2

Path diagram depicting neighborhood social cohesion as the mediator between black concentration and obesity among white women

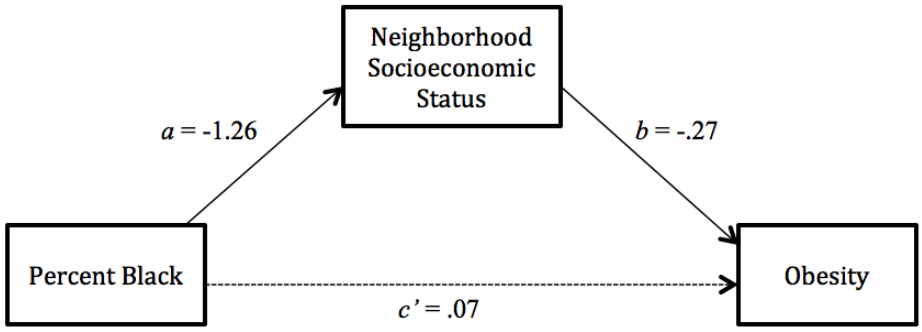

FIGURE 2.3

Path diagram depicting neighborhood SES as the mediator between black concentration and obesity among white women

SES was added as a mediator ($\beta c' = 0.07$, SE = 0.11, $p = 0.57$). The mediated effect of neighborhood SES was statistically significant ($\beta a * \beta b = 0.34$, SE = 0.07, $p < 0.001$).

Discussion

The main purposes of this chapter were to examine whether neighborhood black concentration and residents' obesity risk were linked for four demographic groups defined by gender and black/white race and to explore whether these associations were attributable to neighborhood social and built-environmental features. The results revealed complex patterns at the intersections of race and gender in obesity risks. As expected, black concentration was associated with higher odds of obesity for white women, and this association was mediated by lower level of social cohesion and socioeconomic status in black-concentrated neighborhoods. Among white men and blacks, there was no significant association between black concentration and obesity risks. But white men in black-concentrated neighborhoods were shown to have lower odds of obesity after adjusting for neighborhood SES.

To date, some research focusing on black residential concentration and weight status has found that increasing proportion of black residents in a neighborhood was associated with higher obesity risks without differentiating individual race and gender (Boardman et al. 2005; Chang et al. 2009). This study used recent data and extended previous studies by looking at race and gender groups separately. Results presented here suggest that the observed detrimental effects of black concentration largely apply to white women, and community-level SES profiles and social cohesion play mediating roles between black concentration and obesity risks. These patterns are consistent with social

disorganization theory in that area deprivation and the breakdown of public order as a result of ethnic heterogeneity may increase obesity risks, but only among white women.

After controlling for neighborhood SES, the negative association of black concentration and obesity among white men is unexpected. It is possible that white men living in high density black neighborhoods are more socioeconomically disadvantaged than blacks; therefore, they may be more likely to have manual labor jobs that entail heavy work-related physical activity, or to take public transportation because they do not own a car. Both forces could lead to greater energy consumption among white men compared to their black neighbors.

For blacks, the findings do not support either ethnic density effects hypothesis or the argument that segregation is a fundamental cause of black–white health disparities, and echoes previous studies that reported null findings in the association between black concentration and obesity risks (Do et al. 2007; Kirby et al. 2012; Robert and Reither 2004). It is possible that both protective and detrimental influences on obesity risks operate together in black neighborhoods and lead to this null association, pointing to an arena of research to further explore the dynamic mechanisms underlying this association in order to reduce high obesity prevalence among blacks more efficiently.

It is important to note that the detrimental effects of black concentration for white women should not be interpreted as an argument for the status quo of residential segregation across US communities. Although minority concentration, or proportion of residents belonging to a racial/ethnic group in a neighborhood, has long been used as a proxy measure of racial/ethnic segregation, it entails considerably different meanings distinct from formal segregation measures such as dissimilarity and isolation indices. While segregation indices reflect processes and dynamics of racial inequality and

potential interaction at the societal level (White and Borrell 2011), measures of racial/ethnic concentration may better capture the essence of ethnic density effects. Such distinctions are also reflected in empirical work examining mortality risks, as recent syntheses suggest that using these two types of measures of residential settlement patterns by race/ethnicity could have contradictory findings (White and Borrell 2011). There is emerging scholarly interest to jointly consider these two measures (Warner and Gomez 2010), which could be an important new direction for future research. It is possible that the underpinnings behind residential segregation and racial composition operate together to shape their health implications in the US.

Another unique contribution of this study is testing several hypothesized pathways linking residential racial composition and obesity risks, which has been scarce in existing literature. Neighborhood socioeconomic environment is consistently shown to influence individual health including obesity, above and beyond individual risk factors (Prince et al. 2011; Wen and Kowaleski-Jones 2012). One of my findings that neighborhood SES was negatively associated with obesity risks is consistent with this literature with the exception of blacks, for whom no neighborhood effect was observed. Whether neighborhood SES plays a mediating role for minority concentration has not been well examined, particularly with different social groups. One study in Utah reports that neighborhood SES partially mediated the effect of Latino concentration on obesity (Wen and Maloney 2011), while another national study found little mediating effect for black and Mexican Americans (Kershaw et al. 2013). In this study, while neighborhood SES played a mediating role for white women, it amplified the protective effects of black concentration for white men. Adequately adjusting for area deprivation and concentrated disadvantage related to minority concentration remains a key consideration in researching

segregation and health.

I have also considered perceived neighborhood social cohesion in our analyses. Community-level social cohesion protects residents from a range of physical and psychological health risks (Hutchinson et al. 2009; Rios, Aiken, and Zautra 2012), and my finding that higher social cohesion score is associated with lower odds of obesity among whites is in line with these studies. The results also indicate that neighborhood social cohesion mediates the effects of black concentration among white women, and this mediated effect is further attributable to neighborhood SES. For blacks, the current study did not find any main effect of social cohesion on obesity risks. There are many other aspects of community social environment that we were not able to include in this study, such as racial discrimination and neighborhood safety. Given that neighborhood social environment affects obesity-related risks (Singh et al. 2008), more work on the potential roles of different aspects of social environment is warranted.

With regard to the built environment, this study shows that neighborhood street connectivity had a small but significant protective effect against obesity among white men. Such findings underscore the salience of community physical environment that is favorable to obesity-preventing behaviors such as physical activity (Li and Wen 2013; Wen and Zhang 2009). Interestingly, it was street connectivity rather than park accessibility that had effects for white men. Because street connectivity is a proxy indicator of neighborhood walkability, it is likely that white men living in black-concentrated neighborhoods, compared to their black counterparts, are more engaged in total physical activity for transportation and occupational purposes because they tend to be more socioeconomically disadvantaged. For example, evidence has shown that residents in disadvantaged neighborhoods were more likely to walk than those in

advantageous neighborhoods, despite their concern of neighborhood safety (Ross 2000). This finding suggests that the salutary built-environmental features available in one's neighborhoods may have not been taken advantage of among other groups. Such social disparities in use of neighborhood built-environmental resources have drawn recent attention despite the continuing endeavor to call for health-promoting amenities in community design (Weiss et al. 2011; Wen et al. 2013).

This study is not without limitations. First, the cross-sectional design is limited in handling estimation bias as a result of the nonrandom nature of individuals' neighborhood choice, thus disallowing any causal inference of contextual influences on obesity risks. The selection bias may vary systematically across racial groups, leading to differential effects of residential segregation for different groups. Second, our individual-level measures are based on self-reported responses. It is likely that group-specific bias and recall bias on key measures such as BMI and social cohesion would over- or underestimate group differences. In addition, because this study is based on a sample of whites and blacks collected in the Southeastern Pennsylvania area, generalization of the findings should always be done with caution.

Despite these limitations, this study provides new evidence on the associations between neighborhood social (i.e., black concentration, social cohesion, and neighborhood SES) and built (i.e., street connectivity and park spatial accessibility) environmental features and individual obesity risks, highlighting the importance of considering the intersection of race and gender in neighborhood effects on obesity research. While I find that living in high density black neighborhoods can be a significant correlate of obesity risks, conducting stratified analyses by race and gender is helpful for revealing more nuanced and complex patterns. To my knowledge, this study is among the

first to investigate differential pathways underlying the link between black concentration and obesity risks, particularly for whites. They also point to the need to further examine the roles of residential segregation and minority concentration in contributing to individuals' obesity risk net of individual background. A fruitful elaboration of the current study could be to explore whether residential patterns by race/ethnicity matter to lifestyles factors that are immediately relevant for energy balance, namely physical activity and diet. More evidence is warranted on the mechanisms underlying these observed associations to make policy recommendations tailored for group-specific needs to thus be more effective than a general approach.

CHAPTER 3

RACIAL DIVERSITY AND METABOLIC SYNDROME: EXPLORING EFFECT MODIFICATION ACROSS SOCIAL GROUPS

Background

Biological risk profiles, also called biomarkers, refer to measurable and quantifiable physiological parameters (e.g., blood pressure, cholesterol level, blood sugar) that serve as indices for morbidity and mortality. Incorporating biomarker data into the study of social determinants of health has at least the following three advantages (Crimmins, Kim, and Vasunilashorn 2010). First, biomarkers serve as early indicators of physiological change and the morbidity process (Ridker 2008; Zethelius et al. 2008); therefore, they are considered as useful predictors of disease, disability, and death at a later stage. Second, social scientists can use biomarkers to investigate how distal social, economic, and demographic factors "get under the skin" to produce health risks. Last but not least, biomarker data have the advantage of providing objective and quantified measures; thus they can be a reliable alternative outcome to existing subjective measures such as self-rated health and chronic conditions, which oftentimes suffer from group-specific bias and information (recall or diagnostic) bias.

Although there are numerous biomarkers related to morbidity and mortality risks from the biomedical perspective, only a few of them are routinely examined in large-

scale population studies and are categorized into three groups: cardiovascular risk factors, metabolic risk factors, and inflammation risk factors (Crimmins et al. 2010). Like for many other health indicators, there are unequal distributions of biomarkers in the US population. Analyses of various data sources, such as the National Health and Nutrition Examination Survey (NHANES), the Jackson Heart Study, and the National Social Life, Health, and Aging Project, have all documented that individuals with lower socioeconomic status have higher biological risks in the US, and this inverse association is persistent across age groups from children to the elderly (Crimmins et al. 2010; Herd, Karraker, and Friedman 2012; Hickson et al. 2012). In regard to race and ethnicity, black adults witness higher risks than Latinos and whites while whites have the lowest risks among these three groups (Crimmins et al. 2007). This pattern of racial/ethnic disparities is also observed among adolescents (Rainisch and Upchurch 2013).

Metabolic Syndrome as a Risk Factor

Metabolic syndrome includes a specific cluster of biomarkers that can directly prompt individuals' risk for developing health problems like cardiovascular disease and diabetes. Compared to their counterparts, people who have metabolic syndrome are twice as likely to have heart disease and five times as likely to have type 2 diabetes (National Heart, Lung, and Blood Institute). Particular indicators for metabolic syndrome include a large waistline, a high tryglyceride level, a low HDL cholesterol level, high blood pressure, and high fasting blood sugar. Common factors for developing metabolic syndrome are abdominal obesity, physical inactivity, atherogenic diet, and insulin resistance (Grundy et al. 2005). Many of these underlying risks for metabolic syndrome

are behavioral and lifestyle factors, suggesting that public health efforts can be done to prevent individuals from developing this syndrome.

Prevalence of metabolic syndrome is high in the US adult population and has been consistently increasing in the past decades. An analysis of the NHANES data showed that age-adjusted prevalence was 29.2% in the years 1988-1994, but rose to 34.2% in 1999-2006 (Mozumdar and Liguori 2011). Further examination by racial/ethnic groups revealed that Mexican Americans had the highest prevalence of metabolic syndrome, while non-Hispanic whites and non-Hispanic blacks had also seen significant increase in their prevalence (Mozumdar and Liguori 2011). Using 2001-2006 NHANES data, another study found the observed racial/ethnic differences in metabolic syndrome prevalence were not substantially attenuated by individual lifestyle factors (Karlamangla et al. 2010). There is much need to look beyond individual predictors and explore whether other mechanisms may possibly drive these disparities at the population level.

Neighborhood Effects on Health Risks

Recent scholarship has started to attend to characteristics within residential neighborhood to examine whether they assert contextual influences on individuals' biological risk profiles. Existing evidence has mostly focused on neighborhood socioeconomic status as the contextual predictor. One study analyzing 1988-1994 NHANES III data looked at nine biomarkers representing allostatic load and found that lower neighborhood SES, measured by an index of six census-tract level variables, was associated with worse allostatic load among whites, blacks and Mexican-Americans (Bird et al. 2010). The same study also inquired into the potential moderating roles of gender and race/ethnicity in the association between neighborhood SES and allostatic load, but

did not find significant effect modification by these two demographic variables. Another study using 2002 Chicago Community Adult Health Study data reported that neighborhood affluence was better in predicting cumulative biological risks than neighborhood disadvantage (King, Morenoff, and House 2011).

Several explanations have been proposed to explain the link between neighborhood adversity and individual biological risks. One strand concerns with neighborhood physical characteristics including food environment and recreational facilities. Residents living in low SES neighborhood are less likely to have safe and attractive open space where they can engage in recreational activities (Franzini et al. 2010; Giles-Corti 2002); they are also more exposed to unhealthy food environment including the domination of fast food chains and longer distance from grocery stores (Larson, Story, and Nelson 2009). Lack of physical activity and consumption of atherogenic diet are both lifestyle-based key risk factors elevating cardiovascular disease risks and metabolic syndrome. The second strand of explanations centers on psychosocial pathways through which adverse neighborhood environment affects multiple regulatory physiological systems (Theall, Drury, and Shirtcliff 2012). Neighborhood disorganization and repeated exposure to violence, for instance, have long been proposed as environmental stressors that can negatively influence residents in disadvantaged neighborhoods.

Empirical studies on neighborhood racial/ethnic composition and biomarkers are very limited. One study using 1988-1994 NHANES III data examined racial segregation measured by two formal segregation indices of evenness and exposure (Bellatorre et al. 2011) and found that segregation was positively associated with high risks of allostatic load. They concluded that both whites and blacks paid a health penalty for metropolitan

area segregation, whereas Mexican Americans may be immune to the detrimental effects of segregation. Another analysis on hypertension using 2005 Behavior Risk Factor Surveillance Survey data also confirmed the deleterious effects of metropolitan-level segregation on hypertension, net of individual and spatial SES (Jones 2013). More research is needed to examine the contextual effect of residential segregation or racial/ethnic composition and to explore how this contextual effect is modified by socioeconomic and demographic characteristics.

The Role of Neighborhood Racial/ethnic Diversity

Racial/ethnic diversity is on the rise in both urban and rural America. Logan and Zhang (2010) analyzed US census data from 1980 to 2000 and showed that increasing populations of Hispanics and Asians were blurring the traditional black-white color line in US metropolis. "From the perspective of intergroup exposure, the good news is a powerful trend toward representation of all four main racial/ethnic groups in highly diverse neighborhoods" (Logan and Zhang 2010:1102). In rural America, accelerating racial/ethnic diversity was particularly witnessed during the post-2000 period. Lichter (2012) has offered two explanations driving this pattern. One is white out-migration that has reduced the absolute numbers of white women of reproductive age. Second is the contribution of Hispanic growth, which accounted for over half of all nonmetropolitan population growth between 2000 and 2010.

Increasing racial/ethnic diversity in the US population calls for a newer angle to look at neighborhood racial/ethnic context beyond the traditional black-white segregation. This shift is emphasized by social demographers in their recent scholarship on theorizing and operationalizing additional constructs of multigroup racial/ethnic

composition or segregation (Hao and Fong 2011; Reardon and Firebaugh 2002). Racial/ethnic heterogeneity, one measure that takes into account the relative size and number of multiple groups to reflect diversity in racial/ethnic composition in the populations, has received much less scholarly attention in the neighborhood and health literature compared to other measures such as formal segregation indices and racial/ethnic density.

In other research arenas concerning racial and ethnic relations at the societal level, the Chicago School has proposed long ago that racial diversity or ethnic heterogeneity reflected social disorganization (Sampson and Groves 1989) and some evidence suggested that it deteriorated social cohesion in community life (Putnam 2007). Not surprisingly, social differentiation based on physical attributes or cultural preference can easily lead to this conclusion as people are supposed to have more trust on or more willing to befriend with others who look more like themselves. However, re-examination of the social disorganization theory provides much evidence against the assertion that racial/ethnic diversity and immigration have endangered US communities. In fact, recent evidence on homicide and violence rates (Graif and Sampson 2009; Sampson 2009) and social cohesion (Letki 2008; Portes and Vickstrom 2011) suggests that US communities has benefited from increasing racial/ethnic diversity.

With regard to health, there are a number of reasons why neighborhood racial/ethnic diversity may protect individuals from developing metabolic syndrome. In the US, percentages of black and Hispanics were positively associated with accessibility to health-promoting built environment in terms of distance to parks and green space coverage (Wen et al. 2013). Net of area deprivation or neighborhood socioeconomic adversity, racially diverse areas in urban cities may provide more diverse housing types

and mixed land use, both being favorable factors inviting physical activity (Durand et al. 2011). Local food environment is another important contextual factor of residents' energy balance, and neighborhoods with higher proportion of immigrants tend to have healthier food environment (Dubowitz et al. 2008; Osypuk et al. 2009; Park et al. 2011). This may be attributable to immigrants' low energy-dense diet in their original cultures (Tseng, Wright, and Fang 2014), which results in the availability of healthier ethnic food surrounding ethnic neighborhoods.

In addition to physical presence of health-promoting resources, neighborhood subcultural orientation may also influence residents' health behavior decisions. For example, studies have shown that percentage of residents walking to work in a neighborhood was negatively associated with residents' likelihood of being obese, whereas percentage being obese was positively associated with obesity risks (Wen and Kowaleski-Jones 2012; Wen and Maloney 2011). If racially diverse neighborhoods have more residents engaged in various types of physical activity and/or with normal weight, then an activity-prone and obesity-averse subcultural orientation may be nurtured, which presumably would further influence residents' behaviors and lifestyle choices.

Taken together, these hypothesized mechanisms suggest that neighborhood racial diversity or ethnic heterogeneity should be associated with lower metabolic syndrome-related risks such as physical inactivity, atherogenic diet intake, and stress. In fact, one study using 2003-2008 NHANES data found that ethnic heterogeneity was indeed associated with lower obesity risks, net of a range of neighborhood controls (Wen and Kowaleski-Jones 2012). More empirical research is warranted to explore the role of neighborhood racial diversity on health as compared to other contextual predictors such as racial segregation or co-ethnic concentration.

Effect Modification by Social Groups

Current literature has provided some evidence for individual variations in the relationship between neighborhood and health (Diez Roux and Mair 2010). The most studied effect modification regards sex differences. In general, previous studies showed that neighborhood effects were stronger and more robust for women across a wide range of health outcomes. One common explanation is that women spend more time at home than men, so they are more exposed to various aspects of neighborhood environment. However, some scholars also suggest that how neighborhood effects differ by sex is dependent on the specific contextual predictor one would look into. One study examining neighborhood and obesity among US adults aged 55 years and older suggested that built environment was more salient for women while economic and social environment mattered more for men (Grafova et al. 2008). Specific to neighborhood racial diversity and biomarkers, it is not clear whether this association differs by sex. But research examining racial segregation (Kershaw et al. 2013) and ethnic concentration (Wen and Maloney 2011) and obesity has provided evidence in support of this interaction, with women being more strongly influenced by the neighborhood.

Age is another crucial individual characteristic worth exploring because identifying the environment by age interaction could help understand how residential neighborhood impacts individuals at different life stages. One prior study analyzing two national surveys of US adults showed that the relationship between neighborhood and physical health, measured by chronic conditions and self-rated health, was stronger among older age groups, whereas this association was nonexistent or very weak among respondents of younger adulthood (Robert and Li 2001). Different from physical health outcomes, biomarkers usually reach to a risky level well ahead of actual physiological

change; thus biological risks can be prevailing among younger adults when the actual morbidity process has not yet occurred. Therefore, in the association between neighborhood racial diversity and metabolic syndrome, it is possible that the contextual predictor would have stronger influences among younger adults than among middle-aged or older adults.

Aside from the variations by individual characteristics, different types of neighborhood may also witness differential associations between racial diversity and metabolic syndrome. Past research suggests that neighborhood SES is not only directly associated with health itself, it can also condition the effects of other contextual predictors on health. For example, one study of blacks living in New York City found that black concentration was detrimental for physical health and life satisfaction when neighborhood income was low, but this association was reversed in high income black neighborhoods (Roy et al. 2012). Another study in Texas also showed that the association between Hispanic concentration and obesity prevalence varied by county-level educational attainment (Salinas et al. 2012). Although there has not been enough evidence to aid in hypothesizing how racial diversity and metabolic syndrome will differ by neighborhood SES, Sampson (2009) has offered some justification in his study of linguistic diversity and rates of neighborhood violence. He found that the protective effects of linguistic diversity was stronger in high disorder and high poverty neighborhoods, and implied that diversity and immigration might have re-energized historically disadvantaged neighborhoods with their contextual impacts particularly manifested on residents of these neighborhoods.

Finally, given the different patterns of racial diversity in urban and rural America, one could expect that the association between racial diversity and metabolic syndrome is

likely to differ by levels of urbanization. Because the hypothesized mechanisms linking racial diversity and metabolic syndrome are largely concerned with health-promoting resources such as built and food environment, with greater variations in urban or suburban areas, we may observe stronger contextual effects of racial diversity in urban neighborhoods as compared to rural neighborhoods.

Research Questions

This chapter specifically asks the following two questions:

(1) Is neighborhood racial/ethnic diversity associated with metabolic syndrome in the US?

(2) Does the association between neighborhood racial/ethnic diversity and metabolic syndrome differ by sex, age, neighborhood poverty, and urban status?

Methods

Data

The primary individual-level data source for the current study is the 2003-2008 National Health and Nutrition Examination Survey (NHANES), a series of pooled cross-sectional surveys of about 5,000 US children and adults conducted each year by the National Center for Health Statistics of the Centers for Disease Control and Prevention. The NHANES survey combines both interviews and physical examinations; thus it not only provides self-reported demographic, socioeconomic, and health-related information, but also contains unique and much-needed objective data on medical and physiological measurements. For example, objectively measured biomarker data for metabolic

syndrome, which are rarely found in other nationwide surveys, are available in the NHANES data.

The NHANES survey design is based on stratified, multistage probability sampling of the civilian noninstitutionalized US residents. The four sampling stages include: (1) selection of Primary Sampling Units (i.e., counties or small groups of contiguous counties); (2) segments within PSUs (i.e., blocks or group of blocks); (3) households within segments; and (4) one or more participants within households. More detailed sampling and data collection procedures are provided on the NHANES website: http://www.cdc.gov/nchs/data/series/sr_02/sr02_161.pdf Because NHANES is a nationwide survey, results will have more generalizability in the whole US population. In the analytical sample, I excluded pregnant women and only included respondents aged 20 to 64 years due to the complex relationship between body weight and health among older adults.

The individual data are then linked to the 2000 Decennial US Census, where census-tract socioeconomic and demographic information was obtained. The size and definition of residential boundaries has always been a challenging issue in studying the relationship between neighborhood and health, because variations in spatial scale may encompass different underlying contextual processes. While contextual features of small aggregation such as census tract may be more salient on individual behaviors as they represent more immediate and relevant social and built environment, larger geographic units like county and Metropolitan Statistical Area (MSA) can better capture structural forces resulting from policy influences and levels of social hierarchy. In empirical studies, contextual influences of both income inequality and residential segregation are indeed more robust in larger contexts (Bellatorre et al. 2011; Kershaw et al. 2011; Walton

2009; Wilkinson and Pickett 2006), and using different geographic units of aggregation may lead to different effect estimation in terms of magnitude or even direction. However, because I speculate that local built and food environment are the primary pathways influencing individuals' risks for metabolic syndrome, I choose to use census tract as the unit of analysis at the neighborhood level.

Outcome Variable

Metabolic syndrome. Following the criteria proposed by the American Heart Association and National Heart, Lung, and Blood Institute (Grundy et al. 2005), clinical diagnosis of metabolic syndrome is determined if a person has at least three of the following five biological risks:

(1) Elevated blood pressure (systolic blood pressure≥130mm/Hg or diastolic blood pressure≥85mm/Hg);

(2) Central adiposity (waist circumference ≥102cm for men; ≥88cm for women);

(3) Low serum HDL (<40mg/dL for men; <50mg/dL for women);

(4) Elevated triglycerides (≥150mg/dL);

(5) Elevated fasting glucose (≥100mg/dL).

Thus the outcome variable used in this study is a binary measure indicating whether the respondent had metabolic syndrome (coded as 1 for Yes and 0 for No).

Key Neighborhood-level Variables

Racial diversity. Racial diversity is represented by the index of *racial/ethnic heterogeneity* that takes into account both the relative size and number of groups in the

populations. It is mathematically defined as $1-\sum_i p_i^2$, where p_i is the fraction of the population in a given group. A heterogeneity index approaching one reflects maximum racial/ethnic diversity in a census tract, while a score of zero reflects the presence of only one racial/ethnic group. The calculation of this index was based on proportions of six groups including non-Hispanic whites, non-Hispanic blacks, Asians/Pacific Islander, Hispanics, American Indians/Alaska Natives, and Others in a census tract.

Poverty concentration. Neighborhood poverty concentration serves as both a control variable and a moderating factor in the current study. Here I focus on neighborhood poverty concentration rather than indicators such as neighborhood affluence or neighborhood educational level, because poverty concentration can better disentangle area deprivation or adversity associated with minority-concentrated neighborhoods. Following the categorization of US Census Bureau, poor neighborhood is defined by having at least 20% of residents living below the poverty level (US Census Bureau).

Urban neighborhood. The 2000 rural-urban commuting area (RUCA) codes from the US Department of Agriculture are adopted to capture the urban-rural status of each census tract (US Department of Agriculture 2013). The RUCA codes classify census tracts into 10 primary categories based on measures of population density, urbanization and daily commuting, with 1-3 being metropolitan tracts, 4-6 being micropolitan tracts, 7-9 being small towns, and 10 being rural areas. For this study, urban status is a dichotomous variable indicating whether a census tract is urban (RUCA codes of 1-3) or nonurban (RUCA codes of 4-10).

Individual-level Control Variables

Socio-demographic characteristics. They include age, sex, race/ethnicity, marital status, nativity status, educational attainment, and household income. In addition to the continuous measure of *age*, I include an *age-squared* term in the models to account for possible curvilinear relationship between age and biomarkers. The binary variable for *age group* used in age-stratified analyses distinguished younger adults (aged ≥20 but <45) from middle-aged adults (aged ≥45 but ≤64). *Sex* (male vs. female), *marital status* (married/living with partner vs. single/separated/divorced/widowed), *nativity* (US born vs. foreign born), and *educational attainment* (college graduate or higher vs. less than college) were all coded as binary variables. *Race and ethnicity* was limited to four categories: non-Hispanic whites, non-Hispanic blacks, US-born Hispanics, and foreign-born Hispanics. Other respondents identified themselves as other racial/ethnic categories were excluded in the analysis. I specifically distinguish nativity status among Hispanics because prior studies suggested that US-born Hispanics had higher biological risks than foreign-born Hispanics (Crimmins et al. 2007). Besides educational attainment, another individual socioeconomic status indicator is included in the analysis and is measured by a continuous variable of *household poverty income ratio*, which is calculated by dividing annual household income by the federal poverty line.

Prescribed medication use. Because patients whose biomarkers diagnosed at risky levels are likely to use drug treatment to control their elevated risks, all models adjust for medication use available in NHANES interview data. This includes self-reported response to the survey questions asking whether the respondent was taking prescribed medicine to control for high blood pressure or to control for high cholesterol level (both coded 1 for Yes and 0 for No).

Statistical Analysis

I used multilevel random intercept logistic regression models to examine the contextual effects of neighborhood racial/ethnic diversity on individual risk of metabolic syndrome, with individual predictors at Level 1 and tract-level predictors at Level 2. Because the main purpose was to test whether the racial diversity-metabolic syndrome association differed by sex, age, neighborhood poverty, and urban status, stratified analyses were performed separately for these four hypothesized moderators. In each set of the stratified analysis, Model 1 tested the crude effect of neighborhood ethnic heterogeneity on metabolic syndrome, while adjusting for individual-level controls. Then in Model 2, neighborhood poverty concentration was included to see if the effect of ethnic heterogeneity remained statistically significant net of neighborhood poverty. All analyses were performed in SAS software and were remotely accessed through the National Center for Health Statistics Research Data Center.

Results

Descriptive Statistics

Table 3.1 presented sample characteristics for metabolic syndrome and individual and neighborhood predictors. About 20.5% of respondents had metabolic syndrome. Among the five metabolic syndrome biomarkers, low serum HDL (56.1%) and waist obesity (49.0%) were more prevalent, and elevated fasting glucose (17.9%) and elevated triglycerides (13.4%) were less prevalent. Prevalence for elevated blood pressure was about 24.5%. As the NHANES survey was designed to be nationally representative, socio-demographic characteristics were largely similar to the US population. Average respondent age was 41 years old and the sample comprised slightly more male (50.4%)

TABLE 3.1

Descriptive Statistics of Individual and Neighborhood Variables
(NHANES 2003-2008)

	Percentage or Mean
Individual-level Variables	
Metabolic syndrome	20.48%
Elevated blood pressure	24.47%
Waist obese	49.02%
Low serum HDL	56.10%
Elevated triglycerides	13.43%
Elevated fasting glucose	17.92%
Age	41.17 (0.25)
Male	50.41%
Race/ethnicity	
White	73.37%
Black	12.65%
US-born Hispanic	4.87%
Foreign-born Hispanic	9.11%
Married	65.41%
US born	86.10%
College or higher	25.04%
Poverty Income Ratio	3.10 (0.05)
Urban	75.00%
Medication for blood pressure	15.03%
Medication for cholesterol	9.71%
Neighborhood-level Variables	
Ethnic heterogeneity	0.29 (0.02)
Poverty concentration	15.37%
Sample Size (N)	**10,122**

Note. Standard errors are in parentheses.

than female (49.6%). The majority were whites (73.4%) and blacks accounted for 12.7%. About 14% were Hispanics, with more foreign-born (9.1%) than native-born (4.9%). The majority of respondents lived in urban areas (75%). At the neighborhood level, the diversity measure of ethnic heterogeneity stood at an average of 0.29. About 15.4% of respondents lived in census tracts where at least 20% of their residents were in poverty.

Stratified Analyses

Table 3.2 presented coefficients from multilevel random effects logistic regression models. Among women, Model 1 showed that increasing ethnic heterogeneity was significantly associated with lower risks of having metabolic syndrome ($\beta = -0.44$, $p < 0.05$). In Model 2, living in poor neighborhoods was significantly associated with higher risks of metabolic syndrome ($\beta = 0.25$, $p < 0.01$), while the effect of ethnic heterogeneity became marginally significant ($\beta = -0.38$, $p < 0.10$). Results did not show any significant effects of either ethnic heterogeneity or neighborhood poverty among men.

Table 3.3 presented coefficients from multilevel regression analyses stratified by age groups. It showed that increasing ethnic heterogeneity in a neighborhood was consistently and significantly associated with lower risks of having metabolic syndrome among younger adults aged between 20 and 44 years, both before ($\beta = -0.51$, $p < 0.05$) and after ($\beta = -0.50$, $p < 0.05$) adjusting for neighborhood poverty, but not among middle-aged adults between 45 to 64 years. Living in poor neighborhoods was marginally and positively associated with metabolic syndrome risks among the middle-aged adults ($\beta = 0.17$, $p < 0.10$). A side finding here was the sex differences in metabolic syndrome risks between the two age groups. Men were at higher risks for metabolic syndrome among the younger adults, but this sex difference was reversed among the middle-aged group where women observed greater likelihood of having metabolic syndrome.

Results for urban-rural stratified analyses were presented in Table 3.4. Neighborhood contextual predictors seemed to matter only in the urban stratum. Both ethnic heterogeneity and poverty concentration were significantly associated with metabolic syndrome, but directions of their impact were on the opposite. Similar to the

TABLE 3.2

Multilevel Random Effects Logistic Regression Models
Predicting Metabolic Syndrome, by Sex

	Women		**Men**	
	Model 1	Model 2	Model 1	Model 2
Neighborhood-level Variables				
Ethnic heterogeneity	**-0.441***	-0.382+	-0.229	-0.222
	(0.520)	(0.205)	(0.209)	(0.209)
Poverty concentration		**0.246****		0.089
		(0.094)		(0.097)
Individual-level Variables				
Age	0.110***	0.111***	0.135***	0.136
	(0.023)	(0.023)	(0.022)	(0.022)
Age^2	-0.001**	-0.001**	-0.001***	-0.001
	(0.000)	(0.000)	(0.000)	(0.000)
Race/ethnicity				
White	-0.341	-0.284	-0.186	-0.167
	(0.221)	(0.221)	(0.214)	(0.215)
Black	-0.128	-0.136	-0.663**	-0.663**
	(0.220)	(0.220)	(0.214)	(0.214)
US-born Hispanic	-0.008	0.004	-0.218	-0.209
	(0.244)	(0.244)	(0.242)	(0.243)
Foreign-Born Hispanic	--	--	--	--
Married	0.197*	0.203*	0.120	0.124
	(0.080)	(0.080)	(0.081)	(0.081)
US born	0.112	0.114	0.413*	0.412*
	(0.201)	(0.201)	(1.195)	(0.195)
Education (College)	-0.483***	-0.470***	-0.346**	-0.343**
	(0.110)	(0.110)	(0.104)	(0.104)
Poverty Income Ratio	-0.098**	-0.083**	-0.033	-0.028
	(0.271)	(0.028)	(0.026)	(0.026)
Urban	0.006	-0.006	0.128	0.127
	(0.106)	(0.105)	(0.104)	(0.104)
Medication for BP	0.722***	0.714***	0.790***	0.790***
	(0.094)	(0.094)	(0.097)	(0.097)
Medication for cholesterol	0.175	0.178	0.373**	0.374**
	(0.113)	(0.113)	(0.111)	(0.111)
Intercept	-4.405***	-4.568***	-4.798***	-4.854***
	(0.520)	(0.525)	(0.477)	(0.481)

Note. Standard errors are in parentheses.
***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

TABLE 3.3

Multilevel Random Effects Logistic Regression Models
Predicting Metabolic Syndrome, by Age Group

	Aged 20-44 Years		**Aged 45-64 Years**	
	Model 1	Model 2	Model 1	Model 2
Neighborhood-level Variables				
Ethnic heterogeneity	**-0.510***	**-0.495***	-0.225	-0.185
	(0.222)	(0.221)	(0.199)	(0.200)
Poverty concentration		0.158		0.174+
		(0.100)		(0.094)
Individual-level Variables				
Male	0.246**	0.245**	-0.199**	-0.199**
	(0.078)	(0.078)	(0.068)	(0.068)
Race/ethnicity				
White	-0.155	-0.120	-0.424	-0.384+
	(0.230)	(0.231)	(0.207)	(0.209)
Black	-0.238	-0.238	-0.587	-0.589**
	(0.230)	(0.230)	(0.207)	(0.207)
US-born Hispanic	-0.166	-0.150	-0.221	-0.212
	(0.257)	(0.257)	(0.232)	(0.232)
Foreign-Born Hispanic	--	--	--	--
Married	0.309**	0.314**	0.122	0.129+
	(0.083)	(0.083)	(0.077)	(0.077)
US born	0.272	0.269	0.262	0.262
	(0.211)	(0.211)	(0.187)	(0.188)
Education (College)	-0.195+	-0.195+	-0.499***	-0.489***
	(0.116)	(0.116)	(0.099)	(0.099)
Poverty Income Ratio	-0.061*	-0.052+	-0.049*	-0.040
	(0.029)	(0.029)	(0.025)	(0.025)
Urban	0.169	0.167	0.002	-0.004
	(0.116)	(0.116)	(0.100)	(0.100)
Medication for BP	1.343***	1.346***	0.722***	0.716***
	(0.134)	(0.134)	(0.076)	(0.076)
Medication for cholesterol	0.821***	0.824***	0.254**	0.256**
	(0.187)	(0.187)	(0.086)	(0.086)
Intercept	-2.124***	-2.208***	-0.691***	-0.791***
	(0.183)	(0.190)	(0.159)	(0.168)

Note. Standard errors are in parentheses.
***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

TABLE 3.4

Multilevel Random Effects Logistic Regression Models
Predicting Metabolic Syndrome, by Urbanity

	Urban		**Rural**	
	Model 1	Model 2	Model 1	Model 2
Neighborhood-level Variables				
Ethnic heterogeneity	**-0.398***	**-0.367***	-0.315	-0.338
	(0.167)	(0.167)	(0.454)	(0.448)
Poverty concentration		**0.161***		0.247
		(0.079)		(0.177)
Individual-level Variables				
Age	0.139***	0.140***	0.056	0.056
	(0.018)	(0.018)	(0.034)	(0.034)
Age^2	-0.001***	-0.001***	-0.000	-0.000
	(0.000)	(0.000)	(0.000)	(0.000)
Male	0.010	0.010	-0.013	-0.017
	(0.058)	(0.064)	(0.108)	(0.108)
Race/ethnicity				
White	-0.333*	-0.295	0.071	0.069
	(0.165)	(0.166)	(0.495)	(0.494)
Black	-0.468**	-0.469	-0.104	-0.139
	(0.164)	(0.164)	(0.512)	(0.511)
US-born Hispanic	-0.212	-0.202	0.152	0.168
	(0.185)	(0.185)	(0.526)	(0.525)
Foreign-Born Hispanic	--	--	--	--
Married	0.090	0.096	0.166	0.172
	(0.064)	(0.064)	(0.123)	(0.123)
US born	0.319*	0.320*	0.064	0.055
	(0.150)	(0.150)	(0.427)	(0.426)
Education (College)	-0.381***	-0.375***	-0.449*	-0.446*
	(0.083)	(0.083)	(0.187)	(0.187)
Poverty Income Ratio	-0.064**	-0.054*	-0.051	-0.045
	(0.022)	(0.022)	(0.040)	(0.040)
Medication for BP	0.787***	0.784***	0.770***	0.766***
	(0.078)	(0.078)	(0.133)	(0.133)
Medication for cholesterol	0.281**	0.284**	0.258	0.254
	(0.091)	(0.091)	(0.163)	(0.163)
Intercept	-4.767***	-4.882***	-3.418***	-3.468
	(0.393)	(0.397)	(0.765)	(0.767)

Note. Standard errors are in parentheses.
***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

patterns shown above, increasing ethnic heterogeneity was associated with lower risks for metabolic syndrome among urban residents ($\beta = -0.37$, $p < 0.05$), but neighborhood poverty was associated with higher risks ($\beta = -0.16$, $p < 0.05$)(Model 2).

Table 3.5 presented coefficients by neighborhood poverty status. In neighborhoods where 20% or more residents were living below the poverty line, ethnic heterogeneity was significantly associated with lower likelihood of having metabolic syndrome ($\beta = -0.79$, $p < 0.01$). This association was not statistically significant among residents living in nonpoverty neighborhoods.

Discussion

Using nationally representative data from the 2003-2008 NHANES survey, this chapter examined the contextual effects of neighborhood racial/ethnic diversity on metabolic syndrome among US adults. The analysis particularly focused on differential associations by sex, age, urban status, and poverty concentration. Results indicated that neighborhood racial/ethnic diversity indeed seemed to exert contextual influences on individual biological risks among US adults, net of individual socio-demographic characteristics and neighborhood SES. In particular, increasing racial/ethnic diversity was associated with lower risks for having metabolic syndrome among women, younger adults, and residents living in urban and poor neighborhood. Meanwhile, this association did not seem to exist among men, older adults, or residents living in rural or nonpoor neighborhood.

This study extends past literature in several important ways. First, it is among the first to examine the independent effects of racial/ethnic diversity in the neighborhood and health literature. Past research has paid unanimous attention to the effects of residential

TABLE 3.5

Multilevel Random Effects Logistic Regression Models Predicting Metabolic Syndrome, by Neighborhood Poverty

	Poverty ≥ 20%	**Poverty<20%**
	Model 1	Model 1
Neighborhood-level Variables		
Ethnic heterogeneity	**-0.786****	-0.306
	(0.285)	(0.188)
Individual-level Variables		
Male	-0.318**	0.056
	(0.103)	(0.058)
Race/ethnicity		
White	-0.025	-0.302+
	(0.330)	(0.178)
Black	-0.467	-0.441*
	(0.323)	(0.178)
US-born Hispanic	-0.250	-0.247
	(0.359)	(0.198)
Foreign-Born Hispanic	--	--
Married	0.173	0.237**
	(0.109)	(0.065)
US born	0.282	0.241
	(0.310)	(0.158)
Education (College)	-0.239	-0.392***
	(0.209)	(0.081)
Poverty Income Ratio	0.007	-0.018
	(0.042)	(0.021)
Urban	0.178	0.057
	(0.199)	(0.091)
Medication for BP	1.140***	1.068***
	(0.131)	(0.074)
Medication for cholesterol	0.245	0.535***
	(0.167)	(0.089)
Intercept	-1.299***	-1.601
	(0.261)	(0.146)

Note. Standard errors are in parentheses.
***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

segregation or ethnic concentration on individual health and largely ignored the multi-group context in racial/ethnic composition. Using the measure of racial/ethnic heterogeneity at census tract level to operationalize multigroup composition provides a different angle to look at the health impact of neighborhood racial/ethnic composition. It allows the opportunity to assess the influence of immediate residential environment that is largely distinct from larger structural forces measured by metropolitan-level segregation indices. In this sense, findings from this study are a unique supplement to the past literature largely focusing on black and white segregation and highlighting the detrimental effects of racial segregation on health outcomes in the US. Findings from this study provide compelling evidence against the anxiety towards increasing racial diversity or immigration in the US (Brader, Valentino, and Suhay 2008).

Second, this study utilized objectively measured biomarker data available in a nationally representative survey. The surging scholarly interests in biomarker data to study social determinants of health is, to a large extent, due to the fact that biomarkers are free of response bias and can effectively capture the biological "wear and tear" processes underlying social constructs of individual characteristics (Das 2012) or social environment (Bird et al. 2010). Findings from this study show that rising diversity within residential neighborhood plays a role in the individual biological "wear and tear" process, and this role seems independent of neighborhood adversity and other individual risk factors. Assessing neighborhood racial/ethnic diversity and metabolic syndrome, in particular, provides a more comprehensive view to understand neighborhood effects on physiological dysregulation.

Third, I systematically examined effect modification in the relationship between racial diversity and metabolic syndrome across several hypothesized modifiers. This was

motivated by the lack in previous literature in exploring interactions effects, especially a few understudied modifiers such as age group and urban status. Results were consistent with the hypotheses in that significant associations between racial diversity and metabolic syndrome were only observed among women, younger adults, and residents living in urban and poor neighborhood. The relationship between neighborhood environment and health is complex, and it is crucial to test whether the effects of one contextual predictor would change according to another independent variables. A unique strength of the current study is to test such interaction effects by both individual-level and neighborhood-level characteristics. Results shown here provide a fuller and more nuanced picture of the intricate relationship between neighborhood racial/ethnic composition and health.

The current racial discourse and ongoing debate on immigration in the US have stimulated a soaring scholarship to examine the influences of neighborhood racial/ethnic composition on various aspects of social life and population wellbeing in general. Much discrepancy within this topic centers on the fundamental question about whether increasing minority populations bring in positive or negative consequences in residential communities. In their examination of spatial heterogeneity and neighborhood homicide rates, Graif and Sampson (2009) called for a need for reformulation of the traditional negative connotation of racial/ethnic heterogeneity from the social disorganization tradition. Their interpretation of the beneficial effects of immigrant concentration specifically distinguished two equally important aspects associated with the immigrant effects, the component of *segregation* and the component of *diversity*. The component of diversity, "brought about by the influx of new cultures, skills, and worldviews into urban

neighborhoods," as they suggested (Graif and Sampson 2009:258), is a primary driving force benefiting and energizing urban neighborhoods.

"Cities are back." In his further discussion on the diversity effects, Sampson (2009) has offered insights into the new urbanism where diversity is valued and appreciated rather than being disparaged. Within the new generation of urban neighborhoods, artistic tastes in favor of "neo-bohemia" and "grit as the new glamour" have replaced the traditional impression of decaying and disordered inner cities or ethnic enclaves. More and more people, mostly the young and creative, are drawn into these neighborhoods seeking diversity and social differences. Moreover, Sampson posited that diversity in urban neighborhoods is attracting those "against race" and against the homogenization dominant in suburban sprawl. In sum, Sampson believed that increasing diversity, driven largely by immigration, has revitalized many inner-city neighborhoods, both economically and socially (Sampson 2008). His theoretical arguments received empirical support from this study in that the protective effects of racial diversity were only observed in urban and poor neighborhoods.

Another unique finding of the current study regards the interaction by age groups. Few studies in the past literature have explored how neighborhood effects on health vary by age, which makes it difficult to systematically compare the current study to other empirical evidence. Contrary to an early study of neighborhood SES and physical health that showed stronger contextual effects among older adults, this study found the protective effects of racial diversity were only significant among younger adults aged between 20 and 45 years old. It is plausible to attribute the age variations to different health indicators used in these two studies. As speculated, because the biological "wear and tear" process starts early in the life course, perhaps even traced back to the childhood

and adolescence (Dowd, Zajacova, and Aiello 2010; Rainisch and Upchurch 2013; Theall et al. 2012), neighborhood environment can exert influences on individual physiological dysregulation during early life stages, as compared to later life stages when the morbidity process actually occurs. Another possibility for this age variation could be related to the specific contextual predictor examined in the analysis. As the "new urbanism" has been an explanation for the protective effects of neighborhood diversity (Sampson 2009), and the younger generation is perhaps the major group particularly attracted to its glamour, it is not surprising to find the positive influences of diversity only among this age group.

Limitations

Study limitations should be mentioned. First, the cross-sectional design of this analysis has limited the possibility in handling estimation bias as a result of the nonrandom nature of individuals' neighborhood choice, thus disallowing any causal inference of contextual influences on obesity risks. The selection bias may vary systematically across groups, leading to differential effects of residential racial diversity for different groups. Second, although local built environment and healthy food accessibility were speculated as the underlying mechanisms linking neighborhood racial diversity and metabolic syndrome, this study only focused on effect modification and did not directly test potential mediators.

CHAPTER 4

A PROPENSITY SCORE APPROACH TO EXAMINING ETHNIC DENSITY, IMMIGRANT CONCENTRATION, AND HISPANIC HEALTH RISK FACTORS

Background

Recent decades have seen rapidly accelerating immigrants to the US, particularly Hispanic immigrants from Latin America. Along with this immigration trend is increasing segregation and isolation among Hispanics (Charles 2003). Compared to the black-white segregation, Hispanic-white segregation is moderate. Charles (2003) studied black, Hispanic, and Asian segregation in the 50 largest metropolitan regions in 2000, and reported that only five of them have a Hispanic-white dissimilarity that exceeds 60, compared to 28 areas for blacks. However, Hispanics are still more isolated from whites compared to Asians, and recent studies mostly confirmed that their isolation level is on the rise (Charles 2003; Frey and Farley 1996; Iceland 2004).

Immigrant concentration is another distinctive residential pattern in contemporary America. Upon arrival, many of the new immigrants are constrained by social, cultural, and financial barriers, and tend to cluster in co-ethnic or mixed minority ghettos in order to seek affordable housing, familiar culture, and social networks (Wen, Lauderdale, and Kandula 2009). And this phenomenon is also manifested among the Hispanic population

(Iceland and Scopilliti 2008). Although one might suspect that Hispanic immigrant concentration can be highly correlated with Hispanic co-ethnic density, historically, immigrant enclaves tended to be inclusive of people of various origins and were never homogeneous to one specific group (Williams and Collins 2001). Because Hispanic co-ethnic density is more of a reflection of residential segregation driven by structural forces whereas immigrant concentration can be a result of self-preference during an immigrant's assimilation process, it is thus important to examine the effect of *ethnic density* and the effect of *immigrant concentration* separately.

Neighborhood Racial/ethnic Context and Hispanic Health Risks

Health risk factors such as high blood pressure and high cholesterol level serve as early biological indicators for cardiovascular diseases and other health problems later in life. The Hispanic population has witnessed higher risks of these factors than whites in the US (Crimmins et al. 2007). However, research that has focused on multilevel determinants of Hispanic biological risks, including the roles of Hispanic co-ethnic density and immigrant concentration, is very limited.

Whether minority concentration is good or bad for minority health has generated much debate in recent years (Becares et al. 2012; Kramer and Hogue 2009; Pickett and Wilkinson 2008). One strand of argument centers on the deleterious effects of residential segregation that persistently produce health risks as a result of area deprivation and socio-political isolation. The other side points to the potential benefits of residing with co-ethnics or other minorities, which some believe may foster better social cohesion, provide more health-promoting cultural and economic resources, and protect minorities from discrimination and related stress. Although both sides of arguments have received some

empirical support, it is increasingly recognized that results may depend on the specific contextual predictor one would look at and whether it is an index of racial/ethnic segregation, co-ethnic density, or immigrant concentration that is used in the analysis. In their interpretation of the beneficial effects of immigration concentration on neighborhood violence, Graif and Sampson (2009) specifically distinguished two equally important aspects associated with the immigration effects, the component of *segregation* and the component of *diversity*. Their efforts also provide insight into the health literature on the debate on minority concentration and health outcomes.

Studying the Hispanic population offers a great opportunity to address this debate and to distinguish the effect of residential isolation from the effect of immigrant concentration. On the one hand, many individuals of Hispanic ethnicity are socioeconomically disadvantaged compared to whites and Asians, and Hispanic co-ethnic concentration can be associated with neighborhood disadvantage with the patterns (but to a lesser extent) comparable to black-white segregation. On the other hand, Hispanics account for the largest share of all foreign-born in the US; thus the protective effects, if there are any, are supposed to be manifested among them.

With regard to ethnic density effects, one study of Hispanic adults in Chicago using cross-sectional data in the years 2001-2003 found that an increasing proportion of Hispanics and foreign-born individuals (combined together) was associated with lower hypertension risks among Hispanics (Viruell-Fuentes, Ponce, and Alegría 2012). At the same time, the same study also showed that, among those living with hypertension, Hispanic and immigrant concentration was associated with having worse hypertension care and treatment. Yet this study did not investigate into Hispanic ethnic density and immigrant concentration separately, so it was unclear whether their effects were in the

same direction or the opposite. Another study focused on the sociocultural features of Mexican American neighborhoods in Brownsville, Texas, and used items such as Mexican nameplates and placards in Spanish to measure the Mexican cultural environment within residential neighborhoods (Salinas et al. 2012). It showed that persons living in neighborhoods with greater Mexican cultural environment had higher risk of having diabetes and unstable blood glucoses.

Review of past literature did not show much empirical evidence on immigrant concentration and biological risks. However, research on other health outcomes largely confirmed that immigrant concentration was indeed beneficial for Hispanics in many aspects. In a study examining the prevalence of asthma and other breathing problems, neighborhoods with higher proportions of immigrants witnessed significantly lower health risks among foreign-born Hispanics (Cagney, Browning, and Wallace 2007). This pattern was also observed in outcomes related to cardiovascular diseases. For example, a study of Hispanics aged 45 to 84 found that immigrant concentration in a census tract was associated with lower likelihood of consuming high-fat foods and better availability of healthy food (Osypuk et al. 2009). Other studies also showed that immigrant enclaves may protect against obesity risks among Hispanic children and adults (Kimbro and Denney 2013; Nobari et al. 2013; Wen and Maloney 2011).

Achieving Causal Inference in Neighborhood Effects

Statistical advancement in multilevel modeling has spurred academic research in neighborhood effects on health in the past decade. Still, sample selection bias remains a fundamental challenge in addressing contextual influences on individual health outcomes (Diez Roux and Mair 2010). That is, residents with certain health risks are not randomly

assigned to different neighborhoods. On the opposite, residents of similar individual characteristics may be more likely to choose to live in neighborhoods of similar physical or social environment. For instance, cross-sectional studies may suggest that neighborhoods with more favorable recreational resources, such as better accessibility to open space, would have positive influences on their residents' engagement in physical activity. However, it is also widely believed that persons with healthier lifestyles would be more likely to move into these types of neighborhood so that it is more convenient for them to fulfill their active lifestyle demand. This kind of situation thus violates the very basic assumption of random sampling in observational studies and incurs the endogeneity or reverse causation problem.

Addressing the problem of selection bias is not new to social science research. Decades ago, Heckman inquired into the problem of marriage effects on women's wage in the marketplace, which motivated his groundbreaking work on sample selection models (Heckman 1974, 1979). Recent scholarship has started to utilize various approaches to address sample selection in the neighborhood-health link. Chaix and colleagues (2011) used cross-sectional data in the Paris metropolitan area of France to study the neighborhood education-diabetes association, and their analytical approach was in line with the traditional Heckman selection models by estimating separate and joint models for neighborhood predictors of the likelihood of study participation and of diabetes. The authors found the residual geographic variations in participation weakly biased this association. More specifically, after taking into account the selection bias, their results showed an 18% decrease in the log prevalence odds ratio for low versus high neighborhood education in terms of odds of diabetes.

Another group of researchers used cross-sectional comparisons by age group as a tool to address sample selection bias (Smith et al. 2011). Their strategy was based on the assumption that youth had less residential choices compared to adults. In particular, the authors studied neighborhood walkability and weight status by comparing two groups of Utah residents aged 17-20 and 27-30. They found that neighborhood effects on overweight or obesity were less prone to selection effects compared to BMI as the outcome. They also found that women exhibited greater selection effects than men in the neighborhood-obesity association.

The Propensity Score Matching Approach

First introduced by Rosenbaum and Rubin (1983), the Propensity Score Matching method aimed to use variables available in the observational data to estimate a propensity score for each individual subject, defined as the probability of receiving treatment (or being in the exposed group) versus being in the control group given this vector of observed variables. Then by comparing the overlap in propensity scores between the two groups (treatment and control) and by matching propensity scores of each respondent to other respondents in the same sample, one can create a matched sample where the two groups will share similar characteristics and mimic experimental designs that are often believed as the golden standard of assessing causal inference.

Compared to other approaches addressing sample selection bias, Propensity Score Matching method has the following advantages in researching neighborhood effects on health. First, it can be used as a diagnostic tool to initially look at the sample distribution. For example, one study of cumulative neighborhood risk and allostatic load among US adolescents showed that adolescents living in high-risk neighborhoods had a greater

propensity for living in such areas (Theall et al. 2012). The authors thus adjusted for such differences in their multilevel regression analysis based on this diagnosis. More importantly, Propensity Score Matching can directly aid in addressing selection bias. One study of neighborhood built environment and walking in the US used propensity score for population weights to estimate propensity-score adjusted effects of built-environment measures on walking (Boer et al. 2007). Another study of neighborhood SES and obesity in France compared multilevel regression results from both propensity-score matched sample and unmatched sample, and confirmed the inverse relationship between neighborhood SES and obesity risks (Leal et al. 2011). Overall, these studies suggested that neighborhood did exert contextual influences on individual health after considering the sample selection effects. However, to my knowledge, few studies have applied Propensity Score Matching method to address selection bias in the neighborhood racial/ethnic context.

Study Aims

This chapter aims to distinguish the effect of Hispanic co-ethnic density and the effect of immigrant concentration among Hispanic adults while utilizing Propensity Score Matching approach to address sample selection bias. I specifically ask the following two research questions: (1) Are Hispanic ethnic density and immigrant concentration positively or negatively associated with them having high blood pressure and high cholesterol level? (2) Are these associations robust after taking into account sample selection bias?

Methods

Data

This study is based on pooled data from the 2006 and 2008 Southeastern Pennsylvania (SEPA) Household Health Survey administrated by the Public Health Management Corporation in Bucks, Chester, Delaware, Montgomery, and Philadelphia counties. This biennial cross-sectional survey drew a stratified probability sample from 54 service areas where each had about 30,000 to 75,000 adult residents, and was conducted through telephone interviews with people aged 18 and older. One eligible adult respondent was chosen from each household based on selection criteria. People aged 60 and older were oversampled for the purpose of asking specific questions to this age group. Self-reported person-level data from the SEPA Household Health Survey were then linked to census-tract profiles obtained from the 2005-2009 American Community Survey (ACS). The final analytical sample included 1,563 Hispanic adults aged between 18 and 91 years.

Individual-level Measures

The outcome measures are two indicators of health risk factors. *High blood pressure* was determined if a respondent in the survey answered "Yes" to the question "Have you ever been told by a doctor or other health professional that you have high blood pressure or hypertension?" Respondents who answered "No" or "Only during pregnancy" were considered not having high blood pressure. Similarly, *high cholesterol level* was determined if a respondent in the survey answered "Yes" to the question "Have you ever been told by a doctor or other health professional that you have high

cholesterol?" Respondents who answered "No" were considered not having high cholesterol level.

There were two sets of individual-level covariates, one for the multilevel models and the other for the predictor model of being in the exposed group in the propensity score analysis. In the multilevel regression analysis, individual socio-demographic controls included self-reported age (ranging from 18-91), sex (male vs. female), marital status (married/living with partner vs. other), nativity (US born vs. foreign born), educational attainment (high school or below, some college, and college or above), income (below 100% federal poverty line, 100%-200% federal poverty line, and at or above 200% federal poverty line), and survey year (2006 vs. 2008).

In the predictor model of the propensity score analysis, covariates were considered predictors of being in the exposed group. In the current study, they were predictors of residents living in Hispanic- and immigrant-concentrated neighborhoods. They included most individual controls in the multilevel regression models predicting the two health risk factors: age, sex, nativity, educational attainment, and poverty income ratio.[2] Additional variables were also included to predict the likelihood of living in these neighborhoods. *Family size* summarized the total number of related adults and children in one household. It was then dichotomized by indicating whether a household had less than five people (coded as 0) or had five or more people (coded as 1). Finally, I included an ordinal measure for housing discrimination (never, once, two to three times, four or more times). This was based on the survey question "Have you ever experienced discrimination, been prevented from doing something, or been hassled or made to feel

[2] To achieve balancing property in the matching process, marital status was dropped from the predictor model and age-squared was added to the predictor model.

inferior in getting housing because of your race, ethnicity, or color? Yes or No." and the follow-up question "How many times?" if respondents answered "Yes" in the previous question.

Neighborhood-Level Measures

All neighborhood measures were drawn from census-tract information in the 2005-2009 American Community Survey (ACS) estimates. *Hispanic density* was based on the raw ACS measure of percentage of Hispanic residents in each census tract (ranging from 0 to 0.86). The original continuous measure was then dichotomized as whether a census tract had 25% or more Hispanic residents. Tracts with 25% or more Hispanic residents were coded 1 to indicate high Hispanic density neighborhoods, while those having less than 25% Hispanic residents were coded 0 and were considered low Hispanic density neighborhoods (Kirby et al. 2012; Wen et al. 2009).

Immigrant concentration was based on the raw ACS measure of percentage of foreign-born residents in each census tract (ranging from 0 to 0.572). The original continuous measure was categorized based on tertiles in the analytical sample to indicate low, medium, or high immigrant concentration in a neighborhood.

Taking into account neighborhood SES is essential in detangling ethnic density effects from area deprivation associated with residential segregation (Becares et al. 2012; Roy et al. 2012). Tract-level information on percent residents living in poverty was first obtained from ACS data. Following the categorization of the Census Bureau (US Census Bureau), I then created a dichotomous measure of *neighborhood poverty* indicating whether a neighborhood had at least 20% of residents living below the poverty level.

Statistical Analysis

The analysis proceeded in two steps. In the first step, I estimated traditional two-level random effects logistic regression models predicting high blood pressure and high cholesterol level, respectively. For each biomarker, I first examined the crude effect of Hispanic ethnic density while adjusting for individual-level covariates (Model 1). In Model 2, I examined the crude effect of immigrant concentration, also adjusting for individual-level covariates. Then in Model 3, I included both Hispanic density and immigrant concentration, while adjusting for neighborhood poverty and individual socio-demographic controls. Results were reported in odds ratios with standard errors.

In the second step, I utilized Propensity Score Matching method to assess whether the results obtained from multilevel regression analysis were susceptible to sample selection bias. My analysis followed the procedures recommended by Oakes and Johnson (2006). First, I estimated propensity scores for each respondent from a logistic regression model predicting living in a high Hispanic- or high immigrant-concentrated neighborhood on a set of individual predictors. Specifically, for Hispanic ethnic density, living in tracts with 25% or more Hispanics was the exposed group (also referred to as the treatment group in the analysis) and living in tracts with less than 25% Hispanics was the control group. For immigrant concentration, living in the high tertile was the exposed group and living in the low tertile was the control group.[3] As mentioned earlier, individual predictors were hypothesized variables that could potentially influence respondents' residential choices: age, sex, nativity, educational attainment, income, family size, and housing discrimination.

[3] For the ease of comparison, the medium tertile category of immigrant concentration was excluded in the propensity score matching analysis.

After a propensity score was estimated for each respondent, I then examined the overlap in propensity scores between the two comparison groups (exposed and control). If the overlap was sufficient and satisfactory for the matching, I then proceeded with "nearest neighbor" matching within a caliper of 0.01. The next step was to check covariate balance by looking at the standardized difference (%bias) between the exposed group and the control group. I used 10% as the cutoff point to determine whether covariate balance was achieved and matched pairs were appropriate; that is, the matching was acceptable only when the standardized difference (%bias) was less than 10%. Once satisfied, the final step was to estimate a causal effect of the exposure, shown as the average effect of the treatment on the treated (ATT). Bootstrapping standard errors with 100 replications were used to calculate t-statistics and to determine if the ATT was significant at the 0.05 level. Both regression analysis and propensity score matching were conducted in Stata 11.2. List-wise deletion was used in dealing with missing data, with percentage missing less than 3% for any variable.

Results

Descriptive Statistics

Descriptive information on study participants was presented in Table 4.1. Prevalence for both health risk factors was relatively high among Hispanic respondents. About 27% respondents were told they had high blood pressure, and about 24% respondents were told they had high cholesterol level. The majority (69%) of respondents were female and over half (57%) of them were foreign born. Socioeconomic indicators showed that only 38% respondents had some college education or above, and about 27% living below the poverty line. Among health behaviors and health outcomes, current

TABLE 4.1

Descriptive Statistics (SEPA Survey 2006 and 2008)

Individual-level Measures	**Percentage or Mean**	**Observations**
High Blood Pressure	26.73%	1,560
High Cholesterol	23.88%	1,554
Age	41.83 (15.01)	1,563
Male	30.90%	1,563
Marital Status		1,555
Married / living with partner	49.45%	
Separated/divorced/widowed	31.19%	
Single	19.36%	
US born	42.77%	1,555
Educational attainment		1,554
High school or below	61.78%	
Some college	18.73%	
College or above	19.50%	
Income		1,563
< 100% FPL	26.68%	
100%-200% FPL	31.99%	
≥ 200% FPL	41.33%	
Survey year 2008	32.35%	1,563
Family size (5 or more)	15.04%	1,563
Housing discrimination		1,530
Never	91.05%	
Once	2.22%	
Two to three times	4.71%	
Four or more times	2.03%	
Neighborhood-level Measures		
Percent Hispanic ≥ 25%	38.86%	1,562
Immigrant concentration		1,562
Low	21.45%	
Medium	36.88%	
High	41.68%	
Neighborhood poverty ≥ 20%	56.57%	1,561
Sample Size (N)		**1,563**

Note. Unweighted sample included both US-born and foreign-born Hispanics. Standard errors are in parentheses.

smoking was least prevalent (21%), while physical inactivity and self-rated health as fair or poor were most prevalent (about 32%). Chronic conditions (26%) and obesity prevalence (28%) ranged in between. Experience of housing discrimination was also visible. About 9% of respondents reported that they had ever experienced discrimination in getting housing because of their race/ethnicity or color. At the neighborhood level, both Hispanic density and immigrant concentration were prevalent. About 39% respondents lived in neighborhoods that had 25% or more of their co-ethnics, and 42% lived in the high tertile in terms of percentage of foreign-born residents. Not surprisingly, 57% respondents lived in neighborhoods where 20% or more residents were in poverty, indicating area deprivation among Hispanics' neighborhoods.

Multilevel Regression Analysis

As the first step of my analysis, I regressed the likelihood of having high blood pressure and high cholesterol level, respectively, on a set of neighborhood- and individual-level covariates using two-level random effects logistic models. Results were presented in Table 4.2. The key variables of interest here were Hispanic density and immigrant concentration. Patterns were largely consistent across the two outcome measures. That is, among Hispanic, living in neighborhoods with 25% or more Hispanic residents were significantly associated with higher likelihood of having high blood pressure and high cholesterol level. At the same time, compared to Hispanics living in low immigrant-concentrated neighborhoods, those living in high immigrant-concentrated neighborhoods were significantly associated with lower likelihood of having these two biological risks. Neighborhood poverty was not significantly associated with having either blood pressure or high cholesterol level.

TABLE 4.2
Odds Ratio from Multilevel Logistic Regression Models Predicting High Blood Pressure and High Cholesterol Level

	High Blood Pressure			**High Cholesterol Level**		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Neighborhood-level Variables						
Percent Hispanic ≥ 25%	**1.429***		1.367	**1.446****		**1.507***
	(0.232)		(0.265)	**(0.207)**		**(0.273)**
Immigrant concentration (medium)		0.732	0.692+		0.935	0.842
		(0.148)	(0.140)		(0.174)	(0.151)
Immigrant concentration (high)		**0.590****	**0.569****		0.691+	**0.671***
		(0.114)	**(0.108)**		(0.132)	**(0.118)**
Neighborhood poverty ≥ 20%			1.249			0.953
			(0.229)			(0.170)
Individual-level Variables						
Age	1.061**	1.060**	1.061**	1.055**	1.054**	1.054**
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)
Male	1.108	1.114	1.135	1.124	1.121	1.137
	(0.161)	(0.163)	(0.165)	(0.162)	(0.163)	(0.165)
Separated/divorced/widowed	1.308+	1.320+	1.250	1.584**	1.626**	1.564**
	(0.204)	(0.208)	(0.197)	(0.248)	(0.256)	(0.247)
Single	0.972	0.959	0.921	1.618*	1.609*	1.581*
	(0.193)	(0.192)	(0.184)	(0.314)	(0.316)	(0.310)
US born	1.283+	1.199	1.249	0.732*	0.678**	0.719*
	(0.181)	(0.171)	(0.177)	(0.104)	(0.0973)	(0.103)
Some college	0.849	0.829	0.844	0.789	0.774	0.785
	(0.152)	(0.151)	(0.152)	(0.144)	(0.144)	(0.144)
College or above	0.599*	0.581**	0.628*	0.706+	0.672*	0.717+
	(0.123)	(0.120)	(0.129)	(0.141)	(0.134)	(0.144)
100%-200% FPL	0.633**	0.618**	0.632**	0.853	0.842	0.850
	(0.105)	(0.104)	(0.105)	(0.144)	(0.145)	(0.144)
200% FPL or above	0.482**	0.442**	0.488**	0.931	0.867	0.923

TABLE 4.2 Continued

	High Blood Pressure			High Cholesterol Level		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
	(0.088)	(0.081)	(0.090)	(0.169)	(0.158)	(0.169)
Survey year 2008	1.055	1.003	1.041	1.045	1.000	1.025
	(0.161)	(0.155)	(0.160)	(0.160)	(0.156)	(0.157)
Constant	0.029**	0.051**	0.039**	0.025**	0.038**	0.033**
	(0.008)	(0.016)	(0.013)	(0.007)	(0.013)	(0.011)
Observations	**1,532**	**1,532**	**1,532**	**1,532**	**1,532**	**1,532**
Number of tracts	**525**	**525**	**525**	**525**	**525**	**525**

***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

Propensity Score Matching Analysis for Hispanic Density

Table 4.3 presented results from logistic regression model predicting the propensity of participants living in a neighborhood with 25% or more Hispanics. It showed that being male sex, born in the US, higher educational attainment, and higher income were all negatively associated with the likelihood of living in Hispanic-concentrated neighborhoods. However, Oakes and Johnson (2006) suggested that for such prediction models, we should not pay too much attention to assess statistical significance of predictors. Therefore, all the original variables were used to estimate the propensity score for each respondent.

Figure 4.1 illustrated the propensity score overlap between the two exposure groups of neighborhood Hispanic density. As an indication of selection bias, the overlap showed that respondents living a neighborhood with 25% or more Hispanics had a greater propensity for living in such areas. Yet Stata's test of balancing property of the

TABLE 4.3

Logistic Regression Model Predicting the Propensity of Living in Hispanic-concentrated Neighborhood

	Coefficient (Standard Error)
Age	0.027 (0.020)
Age^2	-0.000 (0.000)$^{+}$
Male	-0.352 (0.126)**
US born	-0.526 (0.120)***
Educational attainment	-0.597 (0.085)***
Income FPL	-0.487 (0.077)***
Family size (5 or more)	-0.032 (0.163)
Housing discrimination	0.047 (0.094)

Note. N=1,519. Educational attainment, income FPL, and Housing discrimination were all treated as continuous variables.
***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

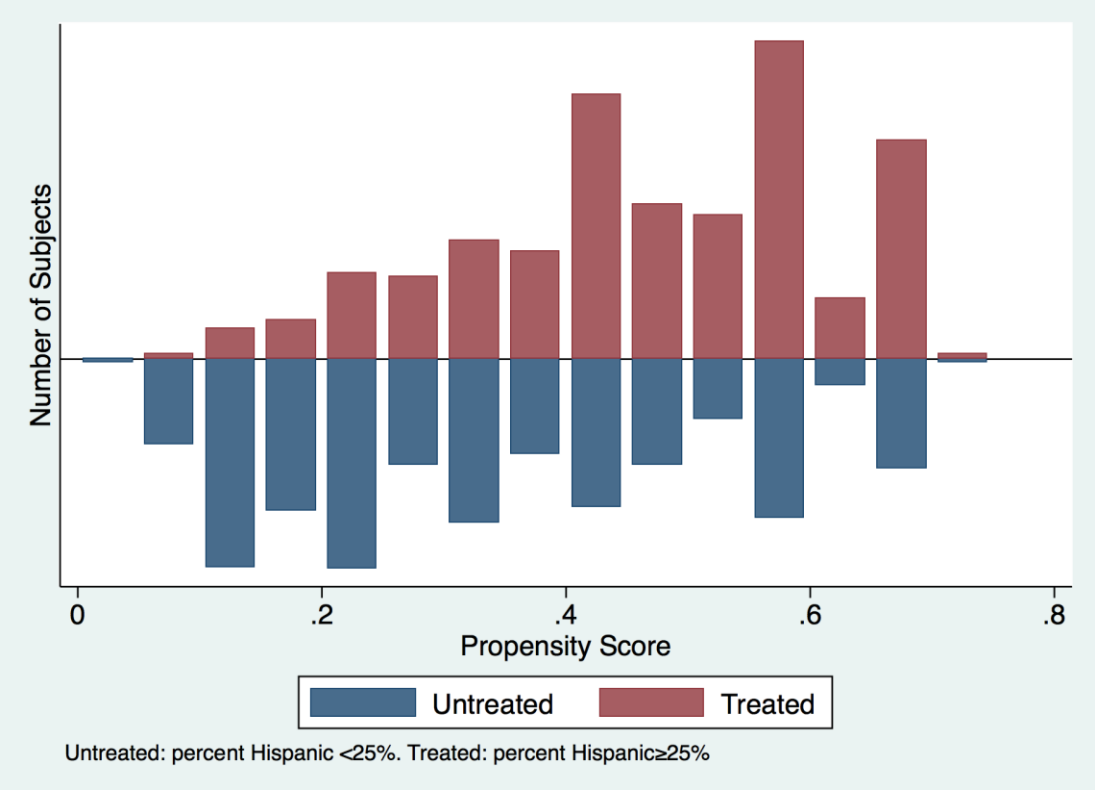

FIGURE 4.1

Overlap in Propensity Score by Neighborhood Hispanic Density

propensity score suggested there was sufficient overlap in the distribution of propensity scores. So I went ahead with the patching process using “nearest neighbor” matching.

After the matching, assessment of covariate balance showed significant reduction in standardized difference (%bias) between the exposed group and the control group. All covariates became more balanced and %bias were all within the 10% threshold. Estimated of the average effect of the treatment on the treated (ATT) were then conducted. Tables 4.4 and 4.5 presented ATT estimates for Hispanic density when high blood pressure and high cholesterol level were the outcomes, respectively. After taking

TABLE 4.4

ATT for Hispanic Ethnic Density when High Blood Pressure is the Outcome

Sample	Treated	Controls	Difference	Standard Error	t-Statistic
Unmatched	0.325	0.229	0.096	0.024	4.13
ATT	0.315	0.244	0.070	0.028	2.52

Note. Bootstrapping standard error is 0.027 (with 100 replications).

TABLE 4.5

ATT for Hispanic Ethnic Density when High Cholesterol Level is the Outcome

Sample	Treated	Controls	Difference	Standard Error	t-Statistic
Unmatched	0.284	0.207	0.078	0.022	3.47
ATT	0.289	0.217	0.071	0.027	2.61

Note. Bootstrapping standard error is 0.028 (with 100 replications).

into account sample selection bias, the estimated difference for having high blood pressure between respondents living in neighborhoods where Hispanics≥25% and those living in neighborhoods where Hispanics<25% remained at about 0.07. Bootstrapping standard error with 100 replications was 0.027. Thus the ATT was significant at the 0.05 level (t-statistic was 2.59). Similarly, the estimated difference for having high cholesterol level remained at about 0.071 and the ATT was also significant at the 0.05 level (t-statistic was 2.54). They suggested that residents living in Hispanic-concentrated neighborhoods were, indeed, more likely to have both biological risks.

Propensity Score Matching Analysis for Immigrant Concentration

Table 4.6 presented results from a logistic regression model predicting the propensity of participants living in a neighborhood with percentage of immigrants in the high tertile versus the low tertile. Both increasing age and being obese were negatively associated with living in an immigrant-concentrated neighborhood. Born in the US and being a current smoker were marginally associated with living in an immigrant-concentrated neighborhood. Again, as assessment of statistical significance of these repressors did not matter in calculating propensity scores, all the original variables were used to estimate the propensity score for each respondent.

Figure 4.2 illustrated the propensity score overlap between the two comparison groups of neighborhood immigrant concentration. It showed sufficient overlap in the distribution of propensity scores, indicating it was very likely to find appropriate matches for most residents living in immigrant-concentrated neighborhoods.

TABLE 4.6

Logistic Regression Model Predicting the Propensity of Living in Immigrant-concentrated Neighborhood

	Coefficient (Standard Error)
Age	-0.021 (0.024)
Age^2	0.000 (0.000)
Male	0.213 (0.164)
US born	-0.378 (0.145)**
Educational attainment	0.102 (0.095)
Income FPL	-0.089 (0.368)
Family size (5 or more)	0.241 (0.213)
Housing discrimination	-0.163 (0.109)

Note. N=955. Educational attainment, income FPL, and Housing discrimination were all treated as continuous variables.
***p<0.001, **p<0.01, *p<0.05, $^{+}$p<0.10 (two-tailed test)

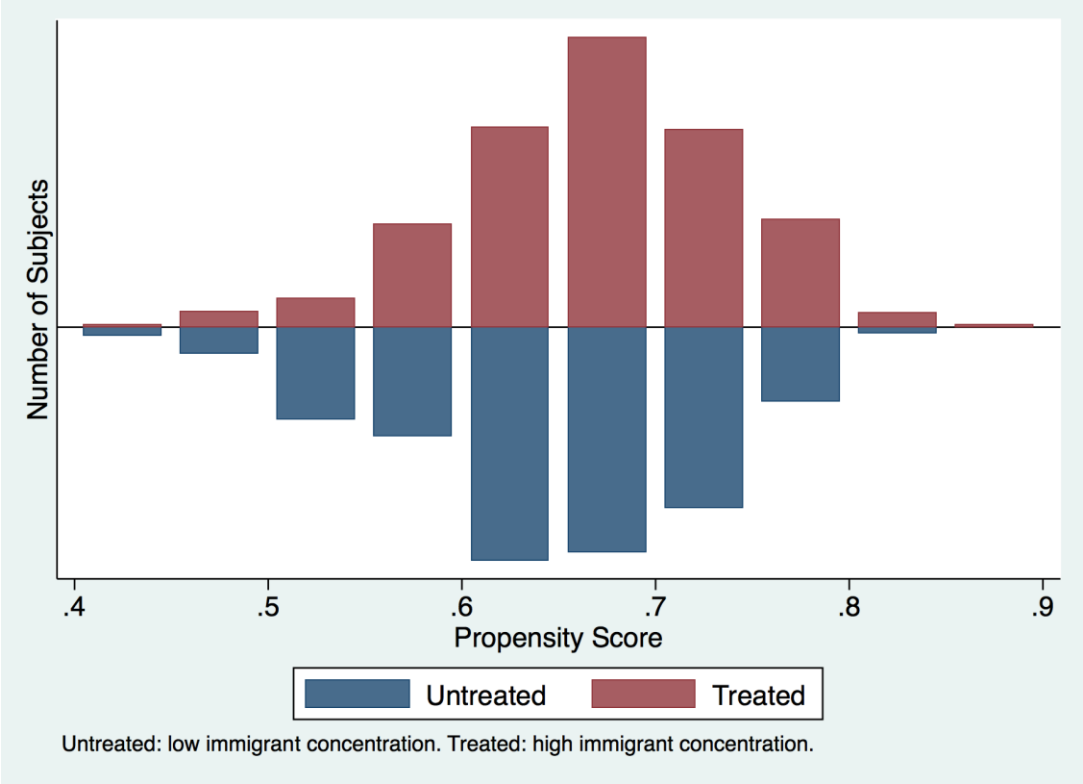

FIGURE 4.2

Overlap in Propensity Score by Neighborhood Immigrant Concentration

Assessment of covariate balance after matching showed significant reduction in standardized difference (% bias) between the exposed group and the control group. All covariates became more balanced and standardized differences were all within the 10% threshold. Estimated effects of the average effect of the treatment on the treated (ATT) were then conducted. Table 4.7 and 4.8 presented ATT estimates for immigrant concentration for the two health risk factors, respectively. After taking into account sample selection bias, the estimated difference for having high blood pressure between respondents living in neighborhoods in the high tertile of immigrant concentration and those living in neighborhoods in the low tertile was about 0.039. The estimated difference

TABLE 4.7

ATT for Immigrant Concentration when High Blood Pressure is the Outcome

Sample	Treated	Controls	Difference	Standard Error	t-Statistic
Unmatched	0.219	0.316	-0.096	0.030	-3.26
ATT	0.265	0.304	-0.039	0.036	-1.07

Note. Bootstrapping standard error is 0.031 (with 100 replications).

TABLE 4.8

ATT for Immigrant Concentration when High Cholesterol Level is the Outcome

Sample	Treated	Controls	Difference	Standard Error	t-Statistic
Unmatched	0.193	0.276	-0.083	0.028	-2.94
ATT	0.218	0.273	-0.055	0.035	-1.59

Note. Bootstrapping standard error is 0.037 (with 100 replications).

for having high cholesterol level was about 0.055. Neither of these two ATT estimates was statistically significant (t-statistic was -1.26 for blood pressure and -1.49 for cholesterol level using bootstrapping standard error). This result suggested that Hispanics living in high immigrant-concentrated neighborhoods did not significantly differ from those living in low immigrant-concentrated neighborhoods, and selection bias may have played a major role in the observed negative association between immigrant concentration and the two health risk factors shown in traditional multilevel models.

Discussion

This chapter focused on neighborhood racial/ethnic contexts in predicting biological risks among Hispanic adults. I applied Propensity Score Matching approach in the analysis to correct for sample selection bias to achieve better causal inference. Results

from both multilevel models and propensity score matching analyses confirmed that neighborhood Hispanic ethnic density indeed exerted contextual influences on individual biological risks. For both blood pressure and cholesterol level, Hispanic density had detrimental effects on Hispanic adults. However, the negative association between immigrant concentration and Hispanic health risks observed in multilevel models was not confirmed in Propensity Score Matching analysis.

This study is unique in that it seeks to single out the effect of racial/ethnic isolation from the effect of immigrant concentration, both of which can be observed among the Hispanic population in the US. Although recent scholarship has started to attend to the health consequences of residential racial/ethnic composition, studies that specifically compared the influence of Hispanic density and the influence of immigrant concentration were very limited. One nationwide study of birthweight using the 2000 detail natality data found that exposure to ethnic enclaves was negatively associated with birthweight among infants of US-born mothers of Mexican origin; in contrast, greater exposure to immigrant enclaves was associated with higher birthweight among US-born mothers (Osypuk, Bates, and Acevedo-Garcia 2010). Similar patterns were also witnessed in another study of obesity in Utah. The authors found Hispanic residential isolation was positively linked to the risk of obesity, but immigrant concentration was negatively associated with the risk of obesity (Wen and Maloney 2011). Results from multilevel models shown in the current study were consistent with these two studies in that the effects of Hispanic density and immigrant concentration on Hispanic biological risks were on opposite directions.

More importantly, the current study has made one step further in making causal inference in the neighborhood and health literature. Past research has largely relied on

observational data with cross-sectional design, and has been criticized for its ineffectiveness in addressing sample selection bias in that residents were not randomly assigned across different neighborhoods. This problem has limited the possibility of making causal influences of the neighborhood context. One major contribution of the current study was to utilize the Propensity Score Matching method to correct for potential situations of structural confounding. This was achieved by performing analyses among participants who were exchangeable between neighborhood exposure groups (i.e., neighborhood Hispanic density, neighborhood immigrant concentration) on the basis of a set of individual predictors that could possibly influence their residential choices. In doing this, observed differences in the outcomes (i.e., high blood pressure, high cholesterol level) between the exposed group and the unexposed group was inferred to be the result of the exposure alone (Oakes and Johnson 2006). Results from Propensity Score Analysis showed that, whereas the positive association between Hispanic density and health risks was more robust, the negative association between immigrant concentration and health risks was likely a result of selection bias.

I specifically focused on the two outcomes of high blood pressure and high cholesterol level because health risk factors like them can well capture the biological "wear and tear" processes underlying social constructs of individual characteristics (Das 2012) or social environment (Bird et al. 2010). Findings from this study showed that racial/ethnic context within residential neighborhood indeed played a role in the individual biological "wear and tear" process, net of neighborhood poverty and other individual risk factors. Yet the influence of Hispanic density and the influence of immigrant concentration were on the opposite directions. While Hispanic density had detrimental effects on Hispanics' biological risks, the effects of immigrant concentration

seemed to be beneficial. Comparing these two contextual predictors provided a more comprehensive view to understand neighborhood influences on physiological dysregulation among the Hispanic population.

Drawing on theoretical and empirical work on residential segregation can help explain the negative effects of Hispanic density on their biological risks. Many of the Hispanic population in the US are socioeconomically disadvantaged and they also tend to be living in more deprived residential neighborhoods. Cumulative disadvantages can heighten the level of both individual- and neighborhood-level stressors that are shown to be playing crucial roles in the "wear and tear" process and physiological dysregulation (McEwen 2001). Although I used Hispanic density instead of formal segregation measure in this study, this finding is in line with Williams and Collins' position that residential segregation is a fundamental cause of racial disparities in health (Williams and Collins 2001). It also echoes empirical evidence that showed negative consequences of co-ethnic concentration on Hispanics' health behaviors and risk factors such as substance use and delinquency (Frank, Cerda, and Rendon 2007), weight status (Do et al. 2007; Wen et al. 2013), birthweight (Osypuk et al. 2010), and self-reported health (Roy, Hughes, and Yoshikawa 2013).

Whether immigrant concentration exerts contextual influences on individual health risks warrants further examination. Recent evidence from multilevel analysis based on cross-sectional designs has shown that immigrant concentration can protect against neighborhood violence (Graif and Sampson 2009) and health risks such as low birthweight (Osypuk et al. 2010) and obesity (Kimbro and Denney 2013; Nobari et al. 2013; Wen and Maloney 2011). Immigrant-concentrated neighborhoods also had better food environment in terms of availability of healthy food and consumption of high-fat

foods (Osypuk et al. 2009), which can be linked to both blood pressure and cholesterol level. In addition, immigrant enclaves have been suggested to help newly arrived immigrants buffer against migration-related stress and thus may lower their biological risks. Taken together, these perspectives provided some support for the beneficial effects of living in immigrant-concentrated neighborhoods for Hispanics, but the current study found that the observed effects of immigrant concentration could be attributable to neighborhood selection, echoing a recent analysis showing that selection bias played a major role in neighborhood effects on obesity (Smith et al. 2011). Indeed, some scholars suggested that immigrant concentration was more likely a result of "compositional effects" as compared to residential segregation and racial/ethnic density, because recent immigrants were more likely than US natives to choose their residential neighborhoods as part of their assimilation process (Mair et al. 2010). Propensity Score Analysis based on observational studies is still not a final solution to draw causal inference (Oakes 2004). Future research may utilize more rigorous study designs, and apply additional operationalization such as language use or cultural preferences to better measure the effect of immigrant concentration.

Limitations

A few limitations are noteworthy. First of all, the key individual-level variables were based on self-reported measures, including the biomarker outcomes. It is likely that some respondents were living with high blood pressure and/or high cholesterol level at the time of interview without knowing it. And this could be more possible in the survey subsample of Hispanics as many of them were socioeconomically disadvantaged or foreign-born, which could be structural or cultural barriers for them in seeking medical

care or health information. Therefore, the prevalence of both biological risk outcomes could be underestimated. In addition, as the issue with missing data was not directly addressed in the whole process of propensity score matching analysis, the propensity score for each respondent would be missing if it had missing values on any propensity-score predictors. This would have further reduced the sample size. Moreover, because this study was based on a sample of Hispanics collected in the Southeastern Pennsylvania area, findings from this study should not be automatically generalized elsewhere.

CHAPTER 5

CONCLUSIONS

In this dissertation, I investigated the association between neighborhood racial/ethnic contexts and health-related risk factors among US adults. Chapter 2 empirically examined the association between black concentration and obesity among blacks and whites in the metropolitan areas surrounding Philadelphia, and focused on mechanisms underlying this association. Results showed a positive association between black concentration and obesity risks among white women and a negative association in this link among white men. Among blacks, the analyses did not reveal significant association between black concentration and obesity risks. Mediation analyses further suggested the positive association observed among white women be mediated by lower level of social cohesion and socioeconomic status in black-concentrated neighborhoods. This chapter presents one of the first empirical analyses to test for mediating effect in the racial isolation/segregation and obesity link.

Chapter 3 turned to neighborhood multigroup racial/ethnic diversity as the contextual predictor and biomarkers as health risks. It focused more explicitly on effect modification and examined how individual- and neighborhood-level characteristics (i.e., sex, age groups, urban status, and neighborhood poverty) moderated the association between racial/ethnic diversity and metabolic syndrome. I found that increasing

racial/ethnic diversity within residential neighborhood was associated with lower risks of diagnosing metabolic syndrome among women, younger adults, and residents living in urban and poor neighborhood. At the same time, racial/ethnic diversity was not significantly associated with metabolic syndrome among men, older adults, or residents living in rural or nonpoor neighborhood.

Chapter 4 applied Propensity Score Matching method as a way to correct for sample selection bias in order to achieve better causal inference. This study also aimed to single out the segregation/isolation effect from immigration effect by simultaneously looking at Hispanic ethnic density and immigrant concentration. Results from both multilevel models and propensity score matching analyses suggested that neighborhood Hispanic density did have impact on Hispanic biological risks over and above individual risk factors, while the observed effects of immigrant concentration were likely due to neighborhood selection bias.

Several themes emerge from the findings in this dissertation research. The first is the complex influence of neighborhood racial/ethnic context. For all health risk factors examined in this dissertation, neighborhood racial diversity and ethnic density did seem to exert influences on individual residents. Although these contextual predictors may not hold significant impacts for every segment of social groups, their importance cannot be ignored. In addition, by applying statistical methods such as Propensity Score Matching analysis, the often-criticized selection bias was specifically addressed. Although this approach is still not able to fully address the counterfactual nature in observational studies (Oakes 2004), it does have advantages over the conventional multilevel modeling approach in achieving causal inference. The apparent discrepancy between the multilevel modeling approach and Propensity Score Matching on the effect of immigrant

concentration shown in Chapter 4 may suggest the strong but often-ignored presence of selection bias in neighborhood effects on health research.

The second theme that can be drawn is that influence of neighborhood racial/ethnic context differs by individual and neighborhood characteristics. For obesity, black concentration mattered for whites but did not show significant effects for blacks. For metabolic syndrome, racial diversity mattered by sex, age, urban status, and neighborhood SES. These findings suggest that any uniform or dichotomous perspective to investigate neighborhood effects will undoubtedly hinder the complexity (Sharkey and Faber 2013). They also suggest that individual-environment interactions can be existent at multiple levels and to fully understand neighborhood effects, we should not hold back at any single level.

Social processes underlying contextual environment such as urban neighborhoods are believed to be complex. This study opens up promising venues for future research to explore underlying mechanisms, especially psychosocial pathways, in the link between neighborhood racial/ethnic context and biological risks. For example, Das (2012) studied black-white disparities in metabolic outcomes and suggested that, other than the regularly claimed pathways of social isolation or poor health behavior, these disparities could derive more consistently from a biological "weathering" mechanism induced by cumulative and multidimensional stress among elderly black men. Bird (2010) also placed emphasis on the role of stress in dysregulation in multiple biological systems. It would be worth examining whether the stress paradigm or the behavior/lifestyle pathways serve as a more salient mechanism linking neighborhood racial diversity and biological risk factors.

To achieve better causal inference, future research may consider more superior study designs such as utilizing longitudinal data and randomized community trials (Oakes 2004). This will require collaborative efforts from almost every segment of scientific research, from data collection to performing analyses. Epidemiological and social science research on neighborhood effects has thrived in the past decade and, to a large extent, data availability has limited the possibility to make major advancement. If more longitudinal data and randomized community trials are available, current research should be able to delve more into the "compositional versus contextual" debate and the "selection versus causation" debate. At that time, scientific research will have more confidence in making policy recommendations and making translational community-based contributions.

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