LIFE COURSE NEIGHBORHOOD POVERTY EXPERIENCES AND THE RISK OF OBESITY IN ADULTHOOD

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ABSTRACT

Gandarvaka Loka Miles: Life Course Neighborhood Poverty Experiences and the Risk of Obesity in Adulthood
(Under the direction of Anna Maria Siega-Riz)

Obesity has been a major public health problem in the US for more than two decades: it is a risk factor for each of the top ten leading causes of death and over one third of American adults are obese. Living in a socioeconomically deprived neighborhood has been implicated as playing an important role in the development of obesity. However, findings in the literature are mixed, and positive findings have been relatively modest after accounting for individual-level characteristics associated with neighborhood selection. Much of this literature, however, is comprised of cross-sectional studies that do not account for variability in the neighborhood environment over the life course or investigate potential latent obesogenic effects of living in a deprived neighborhood in early life. Integration of the life course perspective, which is rooted in sociological theories of the relationship between the social environment and human development, may yield important insights about the influence of neighborhood disadvantage on the risk of obesity in adulthood. Capitalizing on the rich data available from the National Longitudinal Study of Adolescent to Adult Health, this study investigates three life course models of the relationship between the social environment and health: the social trajectories model, the accumulation model, and the critical periods model. The central hypothesis of this research is that the timing, sequence, and accumulation of exposure to neighborhood poverty over the life course influences the

risk of obesity in adulthood. We present empirical evidence of a direct effect of adolescent neighborhood poverty on the risk of obesity in adulthood through pathways not mediated by adult neighborhood poverty among males, females, and non-Hispanic whites. Considering the estimated effects of both adolescent and adult neighborhood poverty on adult obesity, findings for males and non-Hispanic whites are consistent with a critical periods model whereby adolescence neighborhood poverty has a direct effect on adult obesity status and adult neighborhood poverty has no effect. findings for females are consistent with an accumulation model of the life course. These findings advance our understanding of the role of neighborhood disadvantage in shaping adult health providing information that can help guide program and policy interventions aimed at curbing the American obesity epidemic.

I dedicate this work, and all that I do, to my Lord and Savior. Thank you to the many relatives, friends, teachers, and mentors who helped guide and encourage me along this journey. I especially want to thank my husband (RayQuan Miles), mother (Sarah Davis-White), sister (Carmen Gray), and grandmother (Cassie Mosley) for the profound impact they have had on my life. I am eternally grateful for their enduring love, inspiration, and affirmation. I also thank my dear friends from the Department of Epidemiology: Aderonke Akinkugbe, Eboneé Butler, Terra Fatukasi, and Shelly-Ann Love. I will always treasure the bond we developed as we traveled this unique journey together—may that bond continue to grow and deepen in the years to come. Lastly, I dedicate this work to the many, many people from so-called "disadvantaged backgrounds" who will come after me and, too, aspire to accomplish the seemingly impossible.

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LIST OF ABBREVIATIONS

Add Health	National Longitudinal Association of Adolescent to Adult Health
BMI	Body mass index
CARDIA	Coronary Artery Risk Development in Young Adults
CDC	Centers for Disease Control and Prevention
CI	Confidence interval
DAG	Directed acyclic graph
ECLS-K	Early Childhood Longitudinal Study Kindergarten Cohort
IPW	Inverse probability weights
MESA	Multi-Ethnic Study of Atherosclerosis
MTO	Moving to Opportunity for Fair Housing Demonstration Program
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHES	National Health Examination Survey
NH	Non-Hispanic
RD	Risk difference
SES	Socioeconomic status
US	United States
WHO	World Health Organization

CHAPTER 1: RESEARCH AIMS AND HYPOTHESES

Obesity has been linked to each of the top ten leading causes of death in the United States (US), and over 33% of adults and 17% of children are obese [Hoyert & Xu, 2012; Ogden, Carroll et al., 2014]. Although the mechanisms have yet to be fully explained, the neighborhood environment has been implicated as playing an important role in the development of obesity [McNeill et al., 2006; Lee et al., 2009; Carter & Dubois, 2010; Wells et al., 2010; Ding et al., 2011; Kimbro et al., 2011; Ludwig et al., 2011; De Vet et al., 2012; Ding & Gebel, 2012]. It is clear that residential environments, to some degree, regulate access to material and social resources that can, in turn, influence health outcomes [Sharkey & Faber, 2014].

Relative to more affluent neighborhoods, poor neighborhoods tend to lack access to physical activity resources and healthful foods and have a greater density of fast food outlets [Boone-Heinonen et al., 2013]. Additionally, socioeconomically disadvantaged neighborhoods often experience higher crime rates and, thus, their residents tend to feel less safe and socially connected to neighbors [Quillian, 2003]. Numerous studies have reported positive associations between neighborhood socioeconomic status and obesity pointing to these characteristics as the link between the environment and weight-related behaviors [Black & Macinko, 2008]. However, null or weak estimates of these associations have also been reported. This inconsistency in the literature may reflect the variability in design and

analytic methods seen across studies in this field, or nuances in the relationship between neighborhood socioeconomic disadvantage and obesity [Gordon-Larsen, 2014].

This study used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) to test the central hypothesis that exposure to neighborhood poverty over the life course increases the risk of obesity in adulthood. The aims of this research were three-fold:

- Aim 1: Investigate the association between cumulative level of exposure to neighborhood poverty on incident obesity in adulthood.

  Hypothesis #1: Increasing levels of lifelong exposure to neighborhood poverty is associated with a greater risk of adult obesity in a dose-response manner.

- Aim 2: Estimate the association between patterns of exposure to neighborhood poverty and incident obesity in adulthood.

  Hypothesis #2: Distinct patterns of exposure to neighborhood poverty from adolescence to adulthood will be associated with varying levels of adult obesity risk.

  Hypothesis #3: Persistent exposure to high poverty neighborhoods will be associated with the greatest risk of adult obesity.

- Aim 3: Estimate the direct effect of adolescent neighborhood poverty on the risk of obesity in adulthood in order to evaluate adolescence as a "critical" period.

  Hypothesis #4: Exposure to high neighborhood poverty during adolescence (ages 12-18 years) will be associated with a greater risk of obesity in adulthood (≥25 years) independent of adult neighborhood poverty status.

Additionally, an overarching aim of this research was to investigate whether certain population subgroups are more vulnerable to the adverse health effects of poor

neighborhoods. This was accomplished by testing for effect modification by race/ethnicity and sex in each research aim.

This is the first study to explicitly test these theories of the life course with respect to the relationship between neighborhood poverty and adult obesity risk in an adolescent cohort. Therefore, completion of these research aims advances our understanding of the ways in which neighborhood contexts function in the development of obesity, and provides some useful insights that may explain the diversity of findings across studies on this topic.

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CHAPTER 2: BACKGROUND AND SIGNIFICANCE

2.1 Introduction

Obesity is a chronic disease characterized by the storage of excess energy in fat cells resulting in their enlargement or proliferation [Bray, 2004]. This excess fat accumulation, especially accumulation of visceral fat, has been associated with an increased risk of morbidity and mortality in humans [Bray, 2003]. Body mass index (BMI), which is calculated by dividing weight (in kilograms) by height (in meters^2), is the most widely used method for assessing adiposity in obesity research and clinical practice. BMI is both easy to measure and performs fairly well as a proxy for total body fat [Bray, 2004]. Although, it performs less well at distinguishing between individuals with a greater accumulation of visceral versus subcutaneous fat and obscures sex differences in fat accumulation.

BMI classification schemes, developed with the intent of identifying individuals at greatest risk for morbidity and mortality, transform the continuous measure into weight status groups [World Health Organization Expert Committee on Physical Status, 1995]. The World Health Organization (WHO) defines obesity in adults as a BMI $\geq$ 30kg/m^2 with the following sub-categories: Class I, 30 kg/m^2 $\geq$ BMI < 35 kg/m^2; Class II, 35 kg/m^2 $\geq$ BMI <40 kg/m^2; and Class III, BMI $\geq$ 40 kg/m^2. Since children and adolescents experience substantial physiological changes and growth patterns vary greatly by age and sex, childhood obesity is defined differently for 2-18 year olds. In the US, childhood obesity is defined as an age- and sex-specific BMI at or above the 95th percentile relative to a population standard [Krebs et

al., 2007].

The causes of obesity are believed to be both complex and multifactorial, but remain poorly understood. Empirical evidence suggests the underlying mechanisms operate in varying ways throughout the lifespan beginning as early as the gestational period [Caballero, 2007]. This chapter summarizes the epidemiology of obesity in the US; the individual, community, and structural determinants of obesity; the state of the literature on the role of neighborhood poverty in the development of obesity; and the unique contributions of the present study to the obesity epidemiology literature.

2.2 Obesity is a major public health problem in the US

Obesity has reached epidemic proportions in the US (Figure 1). Compared to the early 1960s, the average American adult is nearly 3 times as likely to be obese today [Fryar, Carroll, et al., 2014; Ogden, Carroll, et al., 2014]. Estimates from the National Health Examination Survey (NHES) and the National Health and Nutrition Examination Survey (NHANES) indicate the prevalence of obesity among Americans aged 20 years and older was 13.4% in 1960-1962 and 34.9% in 2011-2012. Furthermore, trends among children and adolescents mirror those seen in adults: from 1980-2012, the prevalence of obesity among 6-11 years olds increased from 7% to 18% and increased from 5% to 21% among 12-19 year olds [National Center for Health Statistics (NCHS), 2012; Ogden, Carroll, et al., 2014].

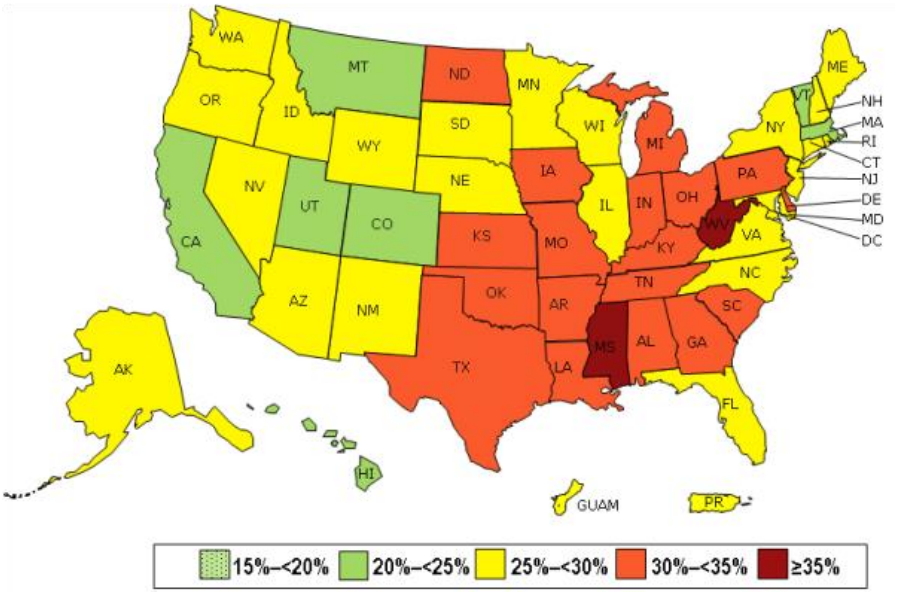

Figure 1**.** Prevalence of self-reported obesity among adults ≥ 18 years old—US, 2013. Source: Behavioral Risk Factor Surveillance Systems, Centers for Disease Control and Prevention.

The rise in obesity prevalence has occurred disproportionately across socioeconomic and racial/ethnic subgroups. Among children ages 2-19 years, obesity prevalence increased across all levels of income to poverty ratio from 1988 to 2008: +8.6 percentage points among boys and +6.8 percentage points among girls living at <130% of poverty, +7.3 percentage points among boys and +5.5 percentage points among girls living at between 130% and 350% of poverty, +5.4 percentage points among boys and +7.4 percentage points among girls living at ≥ 350% of poverty [Ogden, Lamb, et al., 2010]. Furthermore, among non-Hispanic whites in this age group, there was a significant inverse trend between income to poverty ratio and obesity prevalence in 2005-2008 and a similar pattern was observed across levels of parental education. The racial/ethnic disparities in obesity prevalence evident among children are even more pronounced in adults [Ogden, Carroll, et al., 2014]. In addition to its high prevalence and unequal burden in the population, obesity warrants attention from public health practitioners and policymakers alike because of its profound health and social consequences.

The health consequences of obesity are numerous and diverse and, although it has been associated with increased survival in certain populations [Banack & Kaufman, 2014; Carnethon et al., 2012; Oreopoulos et al., 2008; Romero-Corral et al., 2014; Zhao et al., 2014], carrying excess weight has mostly been associated with reduced quality of life and decreased longevity [Dixon et al., 2014]. A thorough discussion of the medical consequences of obesity has been provided elsewhere [Bray, 2003]. In brief, obesity poses risks to almost every major body system:

- Cardiovascular System: increased risk of dyslipidemia and heart disease.
- Digestive System: increased risk of non-alcoholic fatty liver disease, gallbladder

disease, colon and rectal cancer in men, and gallbladder cancer in women.

- Endocrine System: increased risk of Type 2 Diabetes Mellitus, insulin resistance, and the metabolic syndrome.
- Integumentary system: increased risk of striae (or "stretch marks"), acanthosis nigricans, and hirsutism in women.
- Muscular and Skeletal Systems: increased risk of osteoarthritis.
- Neurological System: increased risk of sleep apnea.
- Reproductive System: increased risk of polycystic ovarian syndrome, infertility, and breast cancer in women, and prostate cancer in men.
- Respiratory System: decrease in residual lung volume.

In the pregnant woman, the consequences of carrying excess body fat are compounded, affecting not only the woman but the developing fetus as well [Siega-Riz & Gray, 2014]. Infants born to women who were obese prior to pregnancy are at an increased risk of fetal macrosomia, late fetal death, birth defects, and early neonatal death. Gaining too much weight during pregnancy poses similar risks [Viswanathan M et al., 2008].

Individuals carrying excess weight can also suffer psychosocially due to the stigmatization of obese body types or as a result of the physical comorbidities of obesity [Okifuji & Hare, 2015]. High body mass is associated depression [Preiss, Brennan, et al., 2013] and lower levels of self-esteem [Sikorski, Luppa, et al., 2015]. Among patients with schizophrenia and bipolar disorder, weight gain and obesity is associated with greater severity of symptoms and reduced functioning [Cerimele & Katon, 2013].

Largely a byproduct of its health and social consequences, obesity places a huge economic burden on individuals and society. Obesity was responsible for an estimated $147

billion in medical spending in the US during 2008; this is likely a conservative estimate given that it was based on BMI assessed via self-reported height and weight, which tends to underestimate the prevalence of obesity [Finkelstein et al., 2009]. Furthermore, this estimate does not include costs that were not medically-related such as those resulting from loss of work-related productivity [Hammond & Levine, 2010]. Therefore, the true economic impact of obesity is likely to be even greater than the estimate reported by Finkelstein et al. (2009), and may have increased over time with the rising prevalence of obesity in the population.

Alarmingly, some researchers predict US trends in obesity will continue through the next several decades. A study by Clarke and colleagues (2009) using data from the Monitoring the Future Study revealed that the prevalence of health-promoting weight behaviors such as adequate sleep, exercise, and fruit and vegetable consumption have declined in some young adult populations compared to young adults in the mid-1980s. Results from a study of cohort effects in obesity by Robinson and colleagues (2013) were consistent with these findings: younger cohorts (born after 1980) appear to be more susceptible to the obesogenic (i.e. obesity-promoting) environment than older cohorts (born prior to 1980). Given the profound health and social consequences associated with obesity, understanding and intervening upon the underlying mechanisms driving this epidemic is critical for public health.

2.3 Trends in weight-related behaviors do not fully explain obesity epidemic

Much of the early research on obesity focused on understanding its pathology in humans and the behavioral mechanisms that influence energy balance, especially diet and exercise [Bray, 1990; Caballero, 2007]. It is only within the last 20 years that obesity prevention efforts (and chronic disease prevention efforts, in general) have broadened to include the more distal determinants of obesity [Diez-Roux, 2007]. This shift was largely

motivated by a growing recognition that trends in individual behaviors, alone, could not fully explain temporal trends in obesity incidence and prevalence because:

a) The rate of change in obesity occurred more rapidly than would be expected based on evolution.

   While molecular research over the past three decades has yielded interesting discoveries on the biological determinants of obesity, no findings to date come close to identifying a strong genetic basis for the worldwide obesity epidemic. Single gene mutations associated with obesity are extremely rare and polygenetic mutations, although slightly more common, are not deterministic [Qi & Cho, 2008; Walley et al., 2009]. Furthermore, the prevalence of obesity has increased dramatically since the mid-20th century—far more rapidly than would be expected based solely on changes to the gene pool [Hu, 2008]. This points to changes in the environment as playing a key role in the rapid increase in the prevalence of obesity in the US and abroad.

b) The rise in obesity coincides with changes in the nutrition and physical activity environments of Americans.

   Americans are exposed to more calories—especially calories from energy-dense foods and beverages—than we were in the early and mid-1900s. A trend toward larger portion sizes for prepared food items was nearly universal in the marketplace during the latter half of the 20th century [Dietz & Gortmaker, 2001]. For example, the largest size fountain soda available at three popular US food retailers was 2.5-6 times larger in 2002 than the largest size available when the items were first introduced into the market several decades earlier [Young & Nestle, 2003]. It is not surprising, then, that Americans'

average daily energy intake has increased substantially. Using data from three nationally representative surveys conducted in the US from 1977-2010, Duffey and Popkin (2013) found that the average daily energy intake of American children and adolescents had increased by 108 kcal. However, the researchers concluded that the number of daily eating and drinking occasions was the biggest contributor to this trend. In addition to increasing in size, the composition of Americans' diets has changed. From 1971 to 2006, the proportion of total energy intake from carbohydrates increased among American adults while intake from protein and dietary fat decreased; this trend was observed across all weight status groups [Austin, Ogden et al., 2011]. Additionally, a study of cohort trends in weight-related behaviors among young adults in the US found that the frequency of fruit and vegetable consumption declined among males and fruit consumption declined among Hispanic females from 1984 to 2006 [Clarke et al., 2009]. In addition to these dietary trends, the way Americans work and socialize has become increasingly sedentary, resulting in less daily physical activity. A trend toward more sedentary lifestyles in the US during the latter part of the 20th century has been previously reported [Owen et al., 2010]. Additionally, Clarke and colleagues (2009) reported steady declines in the frequency of exercise among non-Hispanic black women only.

c) The burden of obesity in the US (and other developed country settings) mirrors the relative distribution of social inequalities.

The prevalence of obesity in the US has risen among all population subgroups since the latter half of the 20th century [Fryar et al., 2014]. However, there are clear socioeconomic and racial/ethnic differences in its burden within the population such that disadvantaged

subgroups including women and some racial/ethnic minority groups are at a greater risk of becoming obese. Similar patterns have been observed across most high and, more recently, middle income countries [Popkin & Gordon-Larsen, 2004; Pampel et al., 2012; Devaux & Sassi, 2013].

These observations point to factors in the environment, especially factors more common among or that render disadvantaged groups increasingly vulnerable to developing obesity. These environmental factors have, likely, influenced changes in individual diet and physical activity habits and thus serve as potential drivers of the obesity epidemic. Despite extensive public health efforts over the past 30 years aimed at promoting improved physical activity and dietary habits, little progress has been made in lowering the prevalence of obesity in the US [Institute of Medicine, 2012]. As a result, the 21st century has seen a shift among obesity research and policy stakeholders alike away from the focus on individual determinants of obesity toward a broader systems approach to tackling the problem. Implementing this new approach, however, requires a clearer understanding of the role social contexts play in the development of obesity.

2.4 The determinants of obesity are complex and multi-factorial

The development of obesity is believed to result from multiple layers of influence ranging from proximal causes at the individual level to distal causes that include structures, policies, and systems. In order to conceptualize the determinants of obesity and obesity disparities, the Centers for Disease Control and Prevention (CDC) developed the Socioecological Model shown in Figure 2. This framework, which is based on an adaptation

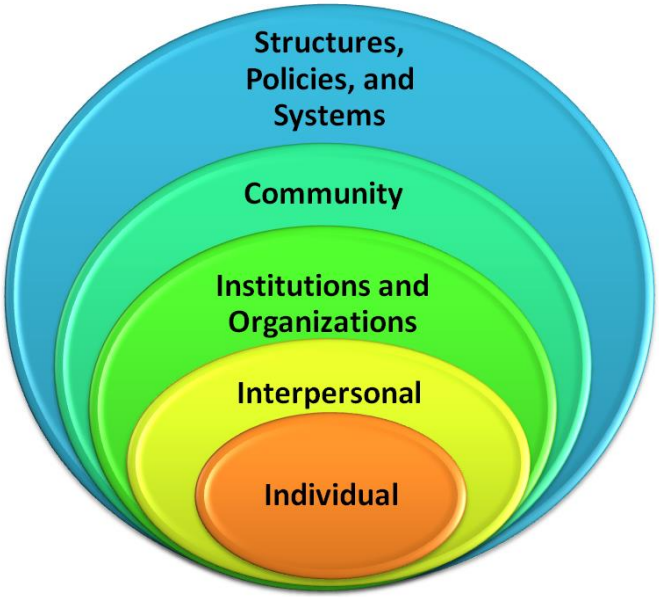

Figure 2. An ecological model of the determinants of obesity. Source: Centers for Disease Control and Prevention. Addressing Obesity Disparities: Social Ecological Model. Available at: https://www.cdc.gov/nccdphp/dnpao/state-local-programs/health-equity/framing-the-issue.html.

of Urie Brofenbrenner's Ecological Systems Theory of Human Development [Brofenbrenner, 1979], emphasizes how one's embeddedness within multiple social and physical contexts shapes his or her weight-related behaviors and characteristics. In the series of concentric circles, each successive layer consists of risk and protective factors hypothesized to be increasingly proximal to the disease.

2.4.1 Individual Determinants

The individual determinants of obesity include the biological (e.g. genes, sex), behavioral (e.g. physical activity, diet), and psychosocial (e.g. health knowledge and beliefs) factors, as well as the knowledge and skills, that influence energy balance.

Obesity in humans is believed to result, at the most fundamental level, from a genetic predisposition to fat accumulation [Speakman, 2013]. However, the genetic origins of obesity are not well-understood. More clear is that age and sex mediate the genetic determinants of fat accumulation via hormones. The sex differences in obesity, whereby adult females are more likely to be obese than adult males, are largely attributed to the distinct functions of estrogen (the primary sex hormone in females) and testosterone (the primary sex hormone in males). Age-graded trends in the prevalence of obesity that mirror fluctuations in estrogen and testosterone levels across the life span provide additional evidence of the critical roles of these hormones in energy metabolism. Other hormones such as cortisol, which is modified by stress and physical activity, also aid in the breakdown and accumulation of fat.

A review of the early life determinants of overweight and obesity found that behaviors in early development—including maternal behaviors during pregnancy—influence weight status later in life [Monasta et al., 2010]. Individuals born to mothers with gestational or chronic diabetes, or mothers who smoked during pregnancy are more likely to become

overweight or obese. Additionally, early feeding (e.g. no breastfeeding or short breastfeeding duration, consumption of sugar sweetened beverages) and physical activity (e.g. <30 minutes daily physical activity, television viewing) habits, and short sleep duration during infancy and early childhood have been associated with high body mass in adulthood.

Diet and physical activity habits continue to influence weight status and the risk of developing obesity throughout the life course. As previously mentioned, there is a well-established literature documenting the relationships among these so-called "weight-related behaviors" and adiposity [Bray, 1990; Caballero, 2007]. However, a broad range of personal factors influence diet and physical activity behaviors including health knowledge, beliefs, and skills [Wilcox et al., 2002]; psychosocial outcomes including depression [Puder & Munsch, 2010; Polanka et al., 2017] and anxiety [Puder & Munsch, 2010]; personality traits such as self-control and impulsivity [Gerlach et al., 2015]; and sleep adequacy [Patel & Hu, 2008; Chaput, 2014]. Beyond these individual-level factors, there are also more distal determinants of obesity.

2.4.2 Interpersonal Determinants

The interpersonal determinants of obesity include characteristics of family, peers, and social networks that influence the individual determinants of obesity. Parents are the first role models of healthy eating and tend to regulate the food and physical activity throughout childhood [Dietz, 1994; Harris & Bargh, 2009; Birch & Doub, 2014]. During late childhood and adolescence, people tend to take on greater responsibility for these behaviors and, thus, their peers [Dietz, 1994; Powell et al., 2015] and the environment [Alvarado, 2016] become increasingly influential.

2.4.3 Institutions and Organizations

The institutions and organizations that we interact with in our daily lives help shape the interpersonal and individual determinants of obesity. Employment in certain industries (e.g. social services) and occupations (e.g. administrative support) [Luckhaupt et al., 2014], and working long hours has been associated with a greater risk of obesity [Solovieva et al., 2011; Luckhaupt et al., 2014]. School plays an important role in shaping the food [Welker et al., 2016] and physical activity [Harrison & Jones, 2012; Morton et al., 2016] environments to which children and adolescents are exposed. School-based interventions aimed at improving nutrition and physical activity knowledge have been shown to lower obesity risk [Sobol-Goldberg et al., 2013]. Religious institutions [Anshel & Smith, 2014] can also influence obesity status through their role in shaping behaviors and lifestyle choices.

2.4.4 Communities

The community-level determinants of obesity are those aspects of the social environment that influence the more proximal determinants. Although the evidence is limited, positive social interactions among community members (i.e. collective efficacy, social cohesion) and between community members and institutions/organizations (i.e. social capital) have been hypothesized to decrease obesity risk by encouraging outdoor recreation, active transport, and a healthy diet [Glonti et al., 2016].

2.4.5 Structures, Policies, and Systems

The most distal level of influence in the etiology of obesity includes factors such as laws, regulations, guidelines, customs, societal norms, and community infrastructure that can influence the determinants of obesity at all other levels [Wilson et al., 1994; Leahey et al., 2011; Kanter & Caballero, 2012]. Research has shown that gender norms, culture, and the

media can influence perceptions of acceptable body types [Wilson et al., 1994; Kanter & Caballero, 2012]. Laws and regulations influencing food cost and availability are among the most fundamental determinants of diet. A recent study concluded that the US food supply is not consistent with dietary guidance [Miller et al., 2015]. An analysis of data from the 2007–2010 NHANES indicated that there is a strong positive association between energy-adjusted diet costs and HEI-2010 scores, especially among women [Rehm et al., 2015]. On a smaller scale, the community built environment has been associated with diet [Cobb et al., 2015], physical activity [Boone-Heinonen et al., 2010; Ding & Gebel, 2012], and obesity [Lovasi et al., 2009; Schwartz et al., 2011].

2.5 Neighborhood poverty: a fundamental cause of obesity

Residential neighborhoods are believed to play a role in the development of obesity through their impact on the determinants of obesity at multiple layers of influence (Figure 1). Neighborhoods help determine the laws, built and social environments, social networks, and norms to which people are exposed [Sharkey & Faber, 2014]. Poor neighborhoods (i.e. neighborhoods marked by a high concentration of low income residents) often possess a greater number of obesogenic environmental characteristics and their residents tend to be at greater risk for obesity.

Numerous studies have documented positive associations among neighborhood poverty, obesity, and obesity-related behaviors in both child and adult populations. Prospective studies tend to report modest, but statistically significant associations between increasing neighborhood disadvantage and obesity risk after accounting for individual characteristics in both youth and adult populations [e.g. Burdette & Needham, 2012; Carter et al., 2012; McTigue et al., 2015; Alvarado, 2016]. Living in a materially deprived neighborhood during infancy was associated with a faster rate of change in BMI z-score in

early childhood (age 4 to 10 years) [Carter et al., 2012]. High compared to low census tract poverty was associated with an additional 0.08 kg/m^2 increase in BMI per year among white and black girls in the Pittsburgh Girls Study. In the only experimental study of neighborhood poverty and health, a modest difference in the 5-year risk of obesity was observed between mothers randomly assigned a voucher for subsidized housing in middle-income neighborhoods (experimental group) and control women (-4.9 and -10.3 percentage points in intention to treat analysis and as-treated analyses, respectively) [Kling et al., 2007]. At 10-15 years post-randomization, only a modest reduction in the risk of extreme obesity was seen among women in the experimental group compared to controls [Ludwig et al., 2011]. However, these differences were not observed the children involved in the study.

Neighborhood poverty has been called a "fundamental cause" of obesity because of its influence on several "downstream" neighborhood mechanisms that are believed to influence weight status more directly (Figure 3).

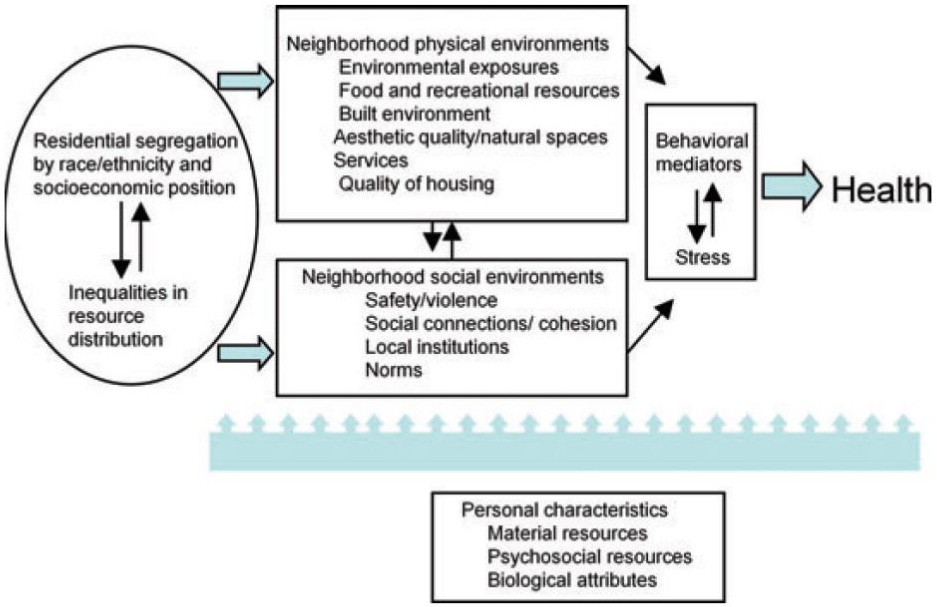

Figure 1. Schematic representation of the contributions of neighborhood environments to health inequalities.

Figure 3. Conceptual diagram of the proposed mechanisms linking neighborhood poverty to obesity. Source: Diez Roux AV, Mair C. Neighborhoods and health. Ann. N.Y. Acad. Sci. 1186 (2010) 125–145.

2.5.1 Hypothesized causal mechanisms linking neighborhood poverty and obesity

Food environment

The neighborhood food environment is often characterized in terms of access—as defined by absolute count, density, or distance—to certain types of foods (e.g. fruits, vegetables, fast foods) or food retail outlets (e.g. convenience stores, fast food or non-fast food restaurants, supermarkets, large- or small-size grocery stores) [Lovasi et al., 2009]. Poor or otherwise socioeconomically disadvantaged neighborhoods tend to have lower access to healthful food such as those available in supermarkets, although the reverse has also been reported [Lamichhane et al., 2015], and/or greater access to energy-dense foods of lower nutritional value such as those frequently offered at convenience stores and fast food restaurants. It has been hypothesized that an obesogenic food environment leads to an unhealthy diet among residents in poor neighborhoods and, in turn, puts them at greater risk of becoming obese.

Findings from review studies have found fairly consistent associations between neighborhood poverty, the food environment, and body mass index; however, the associations between the food environment and diet have been less consistent. One systematic review concluded that residents of low-income neighborhoods have reduced access to supermarkets and healthful food, yet greater availability of energy-dense foods such as those sold in fast food restaurants and convenience stores [Larson et al., 2009]. In this same review, most studies investigating the associations between the food retail environment and diet found that people living in neighborhoods with better access to stores selling healthful foods tend to have healthier food group consumption. In a review of 45 mostly cross-sectional US studies conducted between 1995 and early 2009, Lovasi et al. (2009) concluded that studies investigating the association between neighborhood food

environments and obesity risk in disadvantaged populations (e.g. low income, blacks, Hispanics) yielded inconsistent findings. Access to restaurants and fast food outlets was consistently found to not be associated with obesity risk whereas findings for supermarkets/stores with lost-cost produce and small-size grocery stores/convenience stores were mixed [Lovasi et al., 2009]. A more recent systematic review of 28 mostly cross-sectional and US-based studies, Giskes et al. (2011) concluded that the neighborhood food environment was consistently associated with BMI/weight status. Low area-level socioeconomic status was consistently associated with worse dietary habits whereas other environmental characteristics (e.g. community participation, food environment) did not demonstrate consistent associations with these habits. Most recently, a review conducted by Cobb et al. (2015) also concluded that the association between neighborhood food environment and obesity was inconclusive, and deemed most studies to be of low quality.

Few longitudinal studies have examined associations among neighborhood poverty, the neighborhood food environment, and weight- and/or weight-related behaviors in children. One study found a positive association between availability of convenience stores (store count per 1000 population) within a 0.25-mile buffer and 3-year risk of overweight/obesity and BMI z-score, and a negative association between availability of produce vendors and farmer's markets within a 1.0-mile buffer and 3-year overweight/obesity risk among 6 and 7-year-old girls in a San Francisco-based cohort [Leung et al., 2011]. A Los Angeles County-based study of 2- to 5-year old children enrolled in a federal nutrition program (Special Supplemental Nutrition Program for Women, Infants and Children or WIC) found a non-linear trend between healthy food outlets and 3-year change in weight-for-height: having

either a low or high density of supermarkets and produce markets in the census tract was associated with a more rapid growth rate [Chaparro et al., 2014].

In the only national, population-based longitudinal study of the link between the neighborhood food environment and weight status in children, Chen and Wang (2015) found positive associations among girls. The researchers examined data from the Early Childhood Longitudinal Study Kindergarten Cohort (ECLS-K), which follows a nationally representative cohort of US children from kindergarten in 1998-1999, to estimate the association between changes in food environment and BMI in the transition from late childhood to adolescence. The study by Chen and Wang began in 2004, when respondents were in the 5^{th} grade, and followed them until 3 years later. Girls living in neighborhoods with ≥2 supermarkets at baseline had a lower BMI after 3 years than girls who lived in neighborhoods without any supermarkets (e.g. girls living in neighborhoods with ≥3 had a BMI that was 0.62 kg/m^2 [95% CI: -1.05, -0.18] lower). However, an increasing number of small grocery stores or limited-service restaurants at baseline was associated with a higher BMI 3 years later among girls (e.g. girls who lived in neighborhoods with ≥26 limited-service restaurants had a BMI that was 1.02 kg/m^2 [95% CI: 0.36, 1.68] greater than girls living in neighborhoods with no more than 1 limited-service restaurant). A decrease in the number of small grocery stores was associated with lower BMI by eighth grade among girls. The neighborhood food retail environment was not associated with change in BMI among boys.

Two widely-used studies for longitudinal research on the links between the neighborhood environment and BMI are the Multi-Ethnic Study of Atherosclerosis (MESA) and the Coronary Artery Risk Development in Young Adults (CARDIA). MESA follows a

cohort of black and white Americans, aged 45-84 years at baseline in 2000-2002. One study found that diet quality was inversely associated with supermarket density and positively associated with self- and interviewer-rated quality of the neighborhood food environment in the MESA cohort [Moore et al., 2008]. CARDIA began following a young adult cohort (18-30 years at baseline) in 1985-1986. In a 2013 study by Boone-Heinonen et al., the impact on BMI changes over an 18-year period of simulated changes in the neighborhood environment were investigated using CARDIA data. An increase in supermarket density was associated with intra-individual, between-exam decreases in BMI of 0.09 kg/m^2 (95% CI: -0.16, -0.02). Declines in fast food restaurant and convenience store density were not associated with changes in BMI. Richardson and colleagues (2014) found that individual-level trajectories of neighborhood socioeconomic status (including measures of area-level income and poverty) were associated with longitudinal exposure to convenience stores and restaurants. Individuals experiencing a stable low or downwardly mobile (i.e. high to low) neighborhood SES trajectory over a 20-year period were exposed to fewer (fast food and sit down) restaurants and a greater number of convenience stores over time than individuals experiencing stable high or upwardly mobile trajectories. In another study, these researchers also found that greater access to fast food restaurants was positively associated with consumption of fast foods, while living near sit down restaurants was negatively associated with fast food consumption (Richardson et al., 2015). Both fast food restaurant and sit-down restaurant exposure were indirectly associated with BMI through fast food consumption. However, they found no direct or indirect pathways from neighborhood supermarkets and convenience stores to BMI through food group consumption.

In addition to the actual foods available in obesogenic environments, the increased presence of food cues has been theorized to contribute to the American obesity epidemic [Martin & Davidson, 2014]. Isgor et al. (2016) found that stores in low income neighborhood were more likely to post exterior advertisements than those in higher income neighborhoods. Limited service stores, in particular, were more likely (35% greater odds in adjusted analysis) to post soda advertisements in low income compared to middle- and high-income neighborhoods.

Physical activity environment

The neighborhood physical activity environment encompasses natural and built features that support physical activity. Studies of the physical activity environment use measures such as walkability scores (which assess the extent of pedestrian supports such as street connectivity, presence/quality of sidewalks, etc.), and presence of supports for active (bike lanes) or leisure (e.g. social destinations) transport or recreation (e.g. green space, playgrounds, gyms). In their study of all US census tracts (N=64,885) in 2001-2002, King and Clarke (2015) found that tract-level poverty was positively associated with greater walkability: tracts with more residents at or below poverty had shorter block length, greater street node density, and higher density of street segments. However, higher poverty was negatively associated with open space (i.e. tracts with more poor residents tended to be more developed than tracts with less poverty/higher income residents). In contrast, the association between the physical activity environment, physical activity, and BMI is less conclusive.

Reviews of mostly cross-sectional studies conducted in the US suggest some neighborhood characteristics may be associated with certain types of physical activity and BMI. A review of 50 studies published between 1998 and 2005 found that 80% reported a

positive association between the presence of physical activity resources and physical activity levels among residents [Kaczynski & Henderson, 2008]. A systematic review of studies published between 2002 and 2006 concluded that population density, land use mix, and proximity to non-residential locations were consistently positively associated with walking for transport [Salens & Handy, 2008]. In that same study, the researchers concluded that there is some evidence to suggest walking for recreation is associated with a greater number of pedestrian supports, better neighborhood aesthetic quality, and land use mix. In a review by Lovasi et al. (2009), only access to physical activity facilities and safety (as indicated by crime and traffic) were consistently associated with obesity risk across studies. Studies investigating other aspects of the physical activity environment (e.g. walkability, sprawl, proximity to parks or trails) produced mixed findings.

A CARDIA study found that neighborhood deprivation (an index including measures of census tract poverty and income) was negatively associated with lower physical activity in blacks, but not whites over a 15-year period [Boone-Heinonen et al., 2011]. In a 2013 CARDIA study investigating the impact on BMI changes over an 18-year period of simulated changes in the neighborhood environment, increasing the density of commercial physical activity facilities predicted BMI reductions in men of up to 0.22 kg/m^2 (-0.37, -0.08) [Boone-Heinonen et al., 2013]. Increasing both the density of supermarkets and physical activity facilities produced the largest reductions in BMI among men.

In MESA, Hirsch et al. (2014a) found that moving to a neighborhood with a higher walkability score was associated with longer average weekly transport walking durations (16.04; 95% CI = 5.13, 29.96) and a lower BMI (0.06 kg/m2; (95% CI = –0.12, –0.01). Similarly, Hirsch et al. (2014b) found that certain types of changes to the neighborhood built

environment (i.e. increases in the number of social destinations, the number of walking destinations, and street connectivity) were associated with positive change in walking for transportation.

Social environment

Neighborhood safety has been hypothesized to influence physical activity [Foster & Giles-Corti, 2008; Lovasi et al., 2009; Diez Roux and Mair, 2010] and BMI [Lovasi et al., 2009; Diez Roux and Mair, 2010], but review studies have found these associations to be relatively inconsistent. Discrepancies, however, may be attributable to variation in the health effects of safety across populations or limitations in study design. Studies of other aspects of the neighborhood social environment including social capital, social cohesion, and collective efficacy have similarly produced mixed findings with respect to their associations with obesity risk [Diez Roux and Mair, 2010]. In fact, neighborhood safety and social cohesion were inversely associated with BMI among men in the MESA [Mujahid et al., 2008].

Neighborhood poverty has also been hypothesized to influence BMI through physiological responses to unsafe or disordered social environments, however, the evidence is limited. Neighborhood socioeconomic disadvantage has been associated with higher average resting cortisol levels [Chen & Paterson, 2006; Brenner et al., 2012] and greater cortisol reactivity [Hackman et al., 2012]. Using data from the National Comorbidity Survey Replication Adolescent Supplement, a nationally representative sample of adolescents in the US, Rudolph et al. (2014) demonstrated that high neighborhood disadvantage (an index that included measures of neighborhood-level income) was associated with higher pre-interview cortisol levels and steeper rates of decline. Burdette and Hill (2008) found in their study of

Texan adults that the positive association between neighborhood disorder and BMI was fully mediated by psychological distress.

Chemical exposures

Obesogens are a class of endocrine-disruptive chemicals that promote adiposity by "altering programming of fat cell development, increasing energy storage in fat tissue, and interfering with neuroendocrine control of appetite and satiety" [Janesick & Blumberg, 2016]. A growing body of literature is uncovering how obesogens may function in the development of obesity across the life course. For example, the risk of childhood obesity has been positively associated with maternal exposure to ambient air polycyclic aromatic hydrocarbons [Rundle et al., 2012] and phthalates [Buckley et al., 2016] during pregnancy. Residents in poor neighborhoods are hypothesized to have greater exposure to environmental toxicants [Rauh et al., 2008]. However, the relationships among neighborhood poverty, chemical "obesogens," and obesity have not been investigated; this is an area ripe for future research.

2.5.2 Trends in neighborhood poverty: implications for future trends in obesity

After a period of decline from 1990-2000, the number of Americans residing in neighborhoods with greater than 40% of households with annual incomes at or below the federal poverty level has increased dramatically [Jargowsky, 2013]. By 2011, more than 11 million Americans lived in these so-called "severely distressed" neighborhoods. As shown in Figure 4, this trend toward increasing neighborhood poverty was observed across the US, but was more pronounced in the East and Southeast regions of the country. Furthermore, the racial/ethnic composition of high-poverty neighborhoods became increasingly diverse: whereas non-Hispanic blacks were the dominant racial group in nearly 40% of severely distressed census tracts in 1990, they predominated in less than 30% of these neighborhoods

in 2007-2011 (Figure 5). The Great Recession and subsequent housing market crash in the first decade of the 21st century has been implicated as the driving force behind the rise in extreme neighborhood poverty [Owens & Sampson, 2012]. As Owens and Sampson (2012) note, given the "large body of social science research on the importance of neighborhoods as a social context," the consequences of the recession may not be fully revealed for several decades.

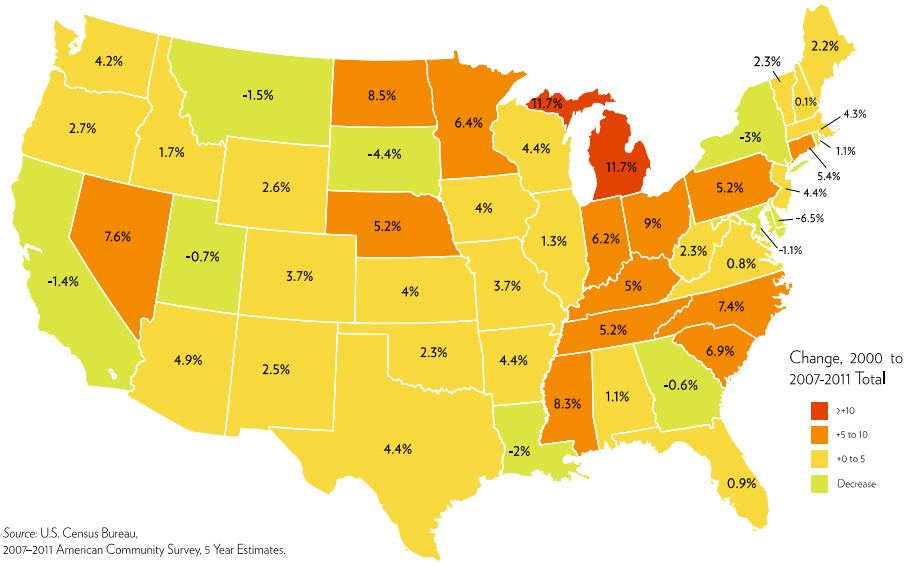

Figure 4. Change in concentrated poverty (census tracts with >40% of households below the federal poverty level) in the US from 2000 to 2007-11. (Sources: 2000 US Census and 2007-2011 American Community Survey).

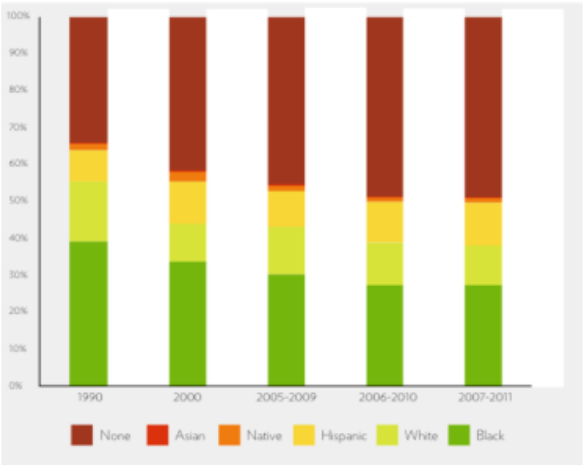

Figure 5. Dominant racial/ethnic group in high poverty census tracts (census tracts with >40% of households below the federal poverty level) in the US from 1990 to 2007-11. (Sources: 1990 US Census, 2000 US Census, and 2007-2011 American Community Survey).

2.6 Critiques of neighborhood effects research

Despite plausible causal mechanisms linking the neighborhood environment to health, mixed findings across the literature as well as conceptual and methodological limitations in study design present a challenge to drawing causal conclusions about positive associations between neighborhood poverty and health outcomes including obesity. Even the definition of "neighborhood" remains the subject of controversy [Diez Roux & Mair, 2010]. Commonly used administrative boundaries may not always capture the activity space relevant to the exposure and mechanisms being studies. In one study, increasing time spent in more socioeconomically advantaged environments for routine activities (e.g. work, shopping, medical care, worship) diminished the association between residential neighborhoods and health [Inagmi et al., 2007].

Second, much of the literature on neighborhood health effects is based on cross-sectional studies with only a single measurement of neighborhood environment [Black & Macinko, 2008; Feng, Glass et al., 2010]. Common to studies employing cross-sectional designs, it is difficult if not impossible to establish temporality in these studies. Furthermore, the implicit assumption of using this approach to exposure assessment is that neighborhood environments are unchanging, or that one's exposure history is unimportant with respect to health. In reality, people change neighborhoods: Americans are highly mobile, especially at younger ages and in lower socioeconomic groups that are more likely to rent versus own their homes [US Census Bureau]. Additionally, neighborhoods themselves evolve over time. In general, a single measure of dynamic exposures can be misleading and even multiple point exposure estimates may not capture experiences during important developmental periods. Both cases can lead to substantial exposure misclassification and biasing of effect estimates [Do et al., 2012].

Third, many studies of the neighborhood SES-obesity relationship have reported null or weak associations when accounting for individual characteristics that may influence neighborhood selection [Oakes et al., 2015]. For example, some studies have reported null or weak associations between neighborhood disadvantage and weight status [Jokela, 2014] or weight-related behaviors [Boone-Heinonen et al., 2011] when using fixed effects regression methods to control for unmeasured confounding by time-invariant characteristics. This has led some researchers to conclude that neighborhood effects on health result from neighborhood composition effects (i.e. the effects of individual-level characteristics and behaviors that influence neighborhood selection) instead of context (i.e. the effects of the neighborhood environment itself) [Jokela, 2014].

Undergirding arguments emphasizing the preeminence of composition over context in neighborhood health effects is the notion that: 1) people freely choose their residential environments, 2) they tend to move into neighborhoods that reflect their preferences, and 3) these preferences predict their current or future health status [Clark, 2005; Bruch and Mare, 2006; Krysan et al, 2009]. There is some empirical data to suggest that health status is associated with later neighborhood socioeconomic status, but the relationship is not clear. Data from the Nurses' Health Study demonstrated that individuals with higher BMI tended to move to neighborhoods with lower median incomes and home values and higher levels of poverty [James et al., 2015]. However, people scoring higher on a walking and physical activity scale were also more likely to move to more socioeconomically disadvantaged neighborhoods. Disadvantaged neighborhoods have been shown to exhibit greater walkability than more advantaged neighborhoods [King & Clarke, 2015]. On the other hand, empirical data also demonstrate that for poor families and racial/ethnic minorities,

neighborhood selection is more of a constrained choice and often a reaction to an exogenous shock such as loss of employment or income [Sampson, 2008; Sampson & Sharkey, 2008; DeLuca et al., unpublished]. Therefore, the link between health preferences and neighborhood selection may only exist for socioeconomically advantaged individuals and families [Lovasi & Goldsmith, 2014].

Randomized community trials have been touted as a solution to disentangling the compositional and contextual effects and temporality issues permeating the epidemiologic literature on neighborhood effects [Oakes, 2014; Oakes et al., 2015]. However, given their costliness and difficulty to implement, few randomized studies have been completed to date. The Moving to Opportunity for Fair Housing Demonstration Program (MTO), a randomized housing mobility study funded by the US Department of Housing and Urban Development, shed some light on the potential causal effects of changes in neighborhood socioeconomic position on health [Ludwig et al., 2011]. The impact of moving to a higher SES neighborhood on obesity was minimal. However, researchers reported low adherence to treatment assignment in this study—evidence of the challenges in attempting to randomize neighborhood environments. Therefore, it is possible that the true effect of decreasing exposure to neighborhood poverty on obesity risk was underestimated in this study.

Fourth, research on the health effects of neighborhood poverty has been conceptually limited [Oakes et al., 2015]. Although researchers have long recognized the need to understand the influence of adverse neighborhood environments over the life course on adult health [Diez-Roux, 2000; O'Campo, 2003], relatively few studies have sought to explicitly operationalize and test life course theories of neighborhood health effects. A common methodological limitation in the extant literature extends from a disregard for potential life

course mechanisms linking the neighborhood environment to adult health: overadjustment. Socioeconomic characteristics such as educational attainment and income may be shaped by one's neighborhood environment in early life [Diez Roux & Mair, 2010]. Although these variables are most often treated as confounders of neighborhood-health associations, they may simultaneously operate as confounders and mediators [Kawachi & Berkman, 2003; Morenoff & Lynch, 2004; Diez Roux & Mair, 2010]. Integrating life course models of the social environment into research on neighborhood health effects can help guide analytic decisions and, thus, provide a more accurate accounting of the obesogenic effects of neighborhood poverty. Extending well-established theories of individual socioeconomic position on adult health to the study of neighborhoods can also provide stronger evidence for causal inference than less theoretically-grounded studies. Given the low feasibility of experimental research on the health effects of neighborhood poverty, the field would benefit greatly from well-designed longitudinal studies developed with these goals. The present study was motivated to fill this gap in the literature.

2.7 The roles of time and timing in neighborhood effects: a critical research gap

The importance of duration and timing of adverse exposures on human development has long been recognized in the social sciences [Elder, 1998]. In his pioneering research in Great Depression-era birth cohorts, Glen Elder and colleagues investigated how drastic social change and economic hardship affected people who experienced it at different stages of the life course. Distinct patterns in long-term health and life chances emerged across generations and population subgroups. For example, the Depression affected young boys and girls differently [Elder & Caspi, 1988]. Joining the military minimized the long-term impact of growing up during the Depression on the lives of some men, and placed them on a different socioeconomic trajectory than their peers [Elder, 1986]. Life course concepts have since been

adapted across a range of disciplines.

There is an established epidemiologic literature documenting the links between childhood socioeconomic environment and adult health. Three potential mechanisms linking childhood socioeconomic conditions to adult health have been proposed (Figure 6) [Berkman, 2009]. First, the childhood socioeconomic environment can shape the development of enduring health behaviors that have long-term effects on disease risk independent of later socioeconomic conditions (Figure 6A). Second, because childhood socioeconomic position constrains adult socioeconomic position by influencing access to resources such as quality education, adult socioeconomic circumstances may add to the cumulative breakdown of biological systems compounding damage done by poor childhood conditions (Figure 6B). Third, childhood socioeconomic environment and adult socioeconomic may influence adult health through a sequence of linked adverse exposures that raise disease risk (Figure 6C). Consistent with an accumulation/cumulative exposure model, studies investigating the effects of timing of exposure to low SES have generally found that both childhood and adult socioeconomic circumstances are associated with disease and longevity [e.g. Elo & Preston, 1992; Frankel et al., 1999; Kuh et al, 2002].

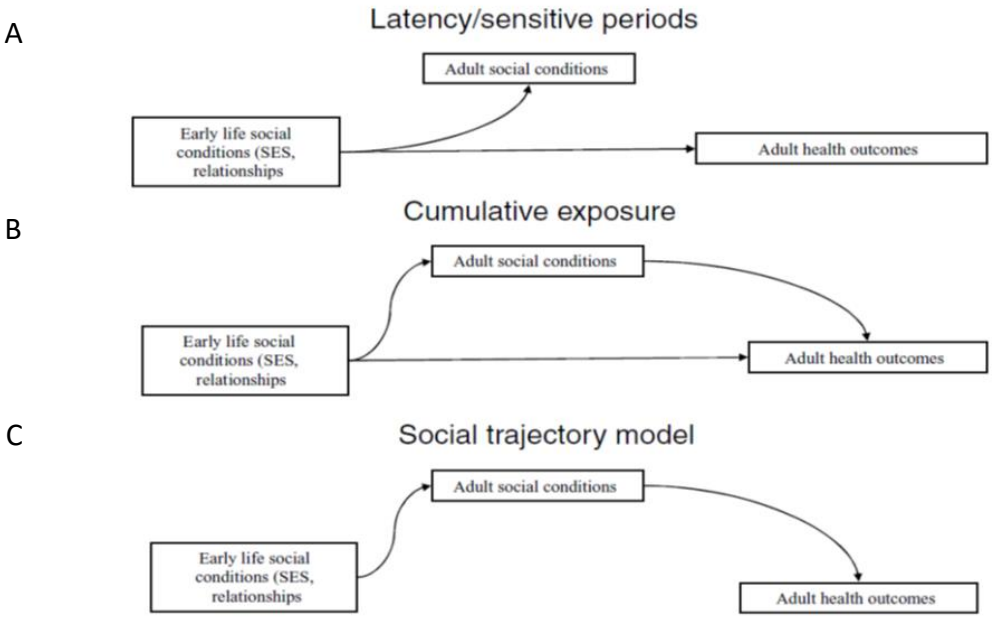

Figure 6. Three life course models of disease. Source: Berkman LF. Social Epidemiology: Social Determinants of Health in the US: Are We Losing Ground? Annual Review of Public Health. 2009; 30:2.

The potential roles of timing, accumulation (or cumulative dose), and patterns of exposure to neighborhood socioeconomic disadvantage on health outcomes including obesity have historically been understudied [Cummins et al., 2007; Merlo, 2011]. As previously discussed (see Section 2.5), the majority of studies on the obesogenic effects of neighborhood poverty have been limited to adult populations or studies focused on a single life stage. However, the development of dietary and physical activity preferences begins in early life [Dietz, 1994; Birch & Doub, 2014] and the relative influence of neighborhood contexts on these behaviors may vary across the life span [Sharkey & Faber, 2014]. Furthermore, inter- and intra-generational obesity trends suggest potential life course effects of the social environment: 1) there are persistent (i.e. intergenerational) age-graded patterns in weight development across the life span; 2) group differences in obesity reflect the social patterning of socioeconomic disadvantage in most developed countries; and 3) these group differences become more pronounced with age. Differential effects of timing, duration, and sequence of exposure to poor neighborhoods can provide clues about obesity etiology [Diez-Roux, 2000; O'Campo, 2003; Kuh & Ben-Shlomo, 2004]. These insights would serve as an important precursor to studies that aim to identify specific biological, behavioral, and social mechanisms by 1) providing a deeper understanding of the ways in which people tend to be exposed to neighborhood poverty, and 2) determining how the manner of exposure is associated with obesity risk. These explorations could unmask underlying variability in the effect of exposure to neighborhood poverty on obesity and, thus, help explain some of the inconsistencies found in this literature. Elucidating life course mechanisms linking early life neighborhood poverty to adult obesity can also help identify resilience/protective factors that can break the chain of risk [Schoon &Bynner, 2003].

2.8 Integrating life course and neighborhood effects

There is an established literature on the influence of individual-level socioeconomic position [Giskes et al., 2008; Lee et al., 2009; Smith et al., 2016] and a growing body of literature on neighborhood SES [Lee et al., 2009; Murray et al., 2010; Burdette & Needham, 2012; Chen & Wang, 2015] across the life course on BMI. This study extends this research by investigating the life course effects of neighborhood socioeconomic disadvantage on weight status in the National Longitudinal Study of Adolescent to Adult Health (Add Health) cohort, a nationally-representative sample of school-attending youth followed into adulthood. Specifically, investigate the effects of timing, accumulation, and patterns of exposure to neighborhood poverty on adult obesity risk based on the three models of life course show in Figure 6.

Regardless of the timing of adverse exposures, persistent exposure to disadvantaged neighborhoods resulting in the accumulation of neighborhood poverty experiences may increase the risk of becoming obese as adult in a dose-response manner (Hypothesis #1). Evidence at the individual level suggests that the accumulation of poverty from birth to age 18 years is associated with the risk of overweight/obesity [Hernandez & Pressler, 2014]. The accumulation of neighborhood disadvantage has been associated with worse health outcomes including preterm low birthweight [Kramer et al., 2014] and BMI [Murray et al., 2010; Chen & Wang, 2015].

Glen Elder and colleagues (2003) defined trajectories as simply a "sequence of [individual] behaviors or experiences." Consistent with a social trajectories model of the life course, the effects of adolescent neighborhood poverty on the risk of obesity in adulthood may operate through a series of adverse exposures associated with distinct patterns of neighborhood poverty experiences over the life course. For example, findings from the MTO

study indicated that the "window of plasticity" for upward neighborhood social mobility is short: poor children living in impoverished neighborhoods after age 13 years tend to remain poor, and live in high poverty neighborhoods in adulthood [Chetty et al., 2015]. Therefore, the adolescent neighborhood environment may be indirectly linked to adult health due to the relative lack of social mobility from adolescence to adulthood in the US. Prior research in the MESA cohort demonstrated that neighborhood trajectories characterized by persistent or increasing poverty over the life course are associated with incident obesity in older females [Murray et al., 2010]. Based on these observations, we hypothesized that: distinct patterns of exposure to neighborhood poverty from adolescence to adulthood will be associated with varying levels of adult obesity risk (Hypothesis #2) and persistent exposure to high poverty neighborhoods will be associated with the greatest risk of adult obesity (Hypothesis #3).

The third aim of this research was to investigate adolescence as a "critical period" in the development of adult obesity for exposure to neighborhood poverty. There are several reasons why adolescence may be an especially vulnerable period for the development of obesity. First, adolescence has been recognized as an important life stage for health-related behavior development [Kuh & Ben-Shlomo, 2004]. Weight-related behaviors begin to develop early in life and, once established, are difficult to modify [Dietz, 1994; Birch & Doub, 2014]. Second, autonomy over diet and physical activity increases during adolescence and the transition to adulthood [Dietz, 1994; Elder, 1998; Gordon-Larsen et al., 2004; Harris et al., 2009; Wells et al., 2010]. Third, diet and physical (in)activity patterns in adolescence have been shown to track into adulthood [Gordon-Larsen et al., 2004; Nelson et al., 2005; Biddle et al., 2010]. Fourth, the incidence of obesity increases rapidly in adolescence and strongly predicts adult obesity [Ogden et al., 2014]. Since poor neighborhoods often have

greater access to unhealthy foods and reduced access to safe recreational spaces [Boone-Heinonen et al., 2013], unhealthy eating and physical activity habits may become ingrained in individuals whose adolescent development occurs under these conditions. Researchers have hypothesized that the association between neighborhood socioeconomic deprivation and weight is stronger in adolescents compared to younger children because this age group likely has more interaction with their environment [Schwartz et al., 2011]. A pattern of stronger, positive associations between neighborhood socioeconomic disadvantage and obesity was observed in the National Longitudinal Survey of Youth, Children and Young Adults [Alvarado, 2016]. Based on a "critical periods" model of the social environment and health, we hypothesized that exposure to neighborhood poverty during adolescence will be associated with a greater risk of obesity in adulthood independent of adult neighborhood poverty status (Hypothesis #4).

2.9 Conclusion

A growing body of literature suggests residing in poor neighborhoods is associated with an increased risk of obesity, but findings have been mixed and mechanisms remain poorly understood. The effects of timing, duration, and patterns of exposure—common themes in life course research—remains a critical knowledge gap in this area of research. The present study applies life course models of the social environment to the study of neighborhood poverty and adult obesity in order to address this gap. Through an efficient secondary analysis of data from Add Health, this study will shed light on the role neighborhoods play in shaping adult health. This study addresses important limitations in the extant literature on neighborhood poverty and obesity by utilizing longitudinal data and by grounding the research in well-established theories of the social environment and health.

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CHAPTER 3: PRELIMINARY STUDIES

The relationships among neighborhood poverty, weight-related behaviors, and weight status have been the subject of numerous prior studies using data from Add Health. This chapter summarizes findings from previous longitudinal studies and presents new data establishing associations between neighborhood poverty and obesity risk in this cohort.

3.1 Neighborhood poverty and the social environment in Add Health

Indicators of neighborhood cohesion, integration, quality, and safety were assessed during the Wave I parent and adolescent in-home interviews as well as the interviewer remarks at each survey wave (perceived safety only). The prevalence of agreement with statements regarding the neighborhood social environment were compared by level of neighborhood poverty (<20%, 20-39%, and ≥40% families at or below the federal poverty). Rao-Scott Chi-square tests are performed to assess the statistical significance of group differences for each construct.

Self-reported neighborhood safety and indicators of neighborhood disorder were associated with level of neighborhood poverty. The percentage of parents and adolescents who were concerned about the safety of their neighborhood increased with level of neighborhood poverty (Table 1). Additionally, parents living in the highest poverty neighborhoods were more than 8 times as likely to report litter being a major issue compared to parents in the lowest poverty neighborhoods, and 7 times as likely to report that drugs were a major issue. Compared to 8.7% of adolescents in neighborhoods with the least

poverty, 28.0% of adolescents in neighborhoods with the highest poverty level reported feeling unsafe.

Add Health interviewers were asked to report their perceptions of respondents' residential neighborhoods in a post-interview questionnaire at each wave including: "How safe did you feel when you were in the sample member's/respondent's neighborhood?" Consistent with the trend observed in parents' and adolescents' perceptions of neighborhood safety, interviewers reported feeling somewhat or very unsafe more often in neighborhoods with higher levels of family poverty and this pattern persisted across survey waves (Figure 7). Additionally, there was a temporal trend toward lower perceived safety in high poverty neighborhoods: the percentage of interviewers reportedly feeling unsafe in these neighborhoods increased from 3.2% at Wave I (1994-1995) to 35.9% at Wave IV (2008-2009).

3.2 Neighborhood poverty, weight-related behaviors, and obesity in Add Health

The association between physical activity environment and individual-level physical activity behaviors has been previously established in Add Health. Moderate-to-vigorous physical activity (number of occasions per week) was associated with number of physical activity facilities within a 3 km buffer and street connectivity within a 1 km buffer of the respondents' home; however, these associations varied by geographic and demographic characteristics [Boone-Heinonen et al., 2010]. Additionally, intersection density was associated with physical activity in areas of low urbanicity. In general, associations between the physical activity environment were more consistent in males compared to females. Frequency of moderate-to-vigorous physical activity was positively associated with the number of physical activity pay facilities in males and negatively associated with crime rates in both males and females.

Several studies have reported significant, positive associations between neighborhood socioeconomic disadvantage and BMI/obesity in the Add Health cohort. Burdette and Needham (2012) found that adolescent exposure to neighborhood disadvantage was positively associated with adolescent BMI (both measured at Wave II) among white males, and a faster rate of change in BMI from adolescence to adulthood (measured at Wave IV) for white females. Nicholson and Browning (2012) observed a curvilinear association between neighborhood disadvantage and the odds of becoming obese for adolescent females such that both low and high levels of disadvantage were associated with greater obesity risk; this relationship differed between non-Hispanic whites and Hispanics. On the other hand, neighborhood disadvantage was not associated with an increase odds of obesity for males.

3.3 Neighborhood selection in Add Health

Parental agreement with statements regarding neighborhood choice and satisfaction was associated with level of neighborhood poverty. With the exception of affordability and the presence of children of similar ages, reasons for neighborhood selection differed significantly at the 0.05-level for all indicators of neighborhood selection (Table 2). In general, parental respondents living in neighborhoods with the highest level of family poverty (≥40%) were less likely to agree with any statement regarding their decision to move into the neighborhood than parents living in neighborhoods with <20% or 20-39% of family poverty. Top reasons for neighborhood selection reported by parents in these high poverty neighborhoods were affordability (43.9%) and proximity to friends and relatives (43.0%). On the other hand, the majority of parents living in neighborhoods with the lowest level of family poverty reported affordability (51.1%), less crime (64.1%), less illegal activity by adolescents (58.9%), and better schools (53.8%) as influencing their decision-making. Parents' and adolescents' level of dissatisfaction with their neighborhood steadily increased

with increasing neighborhood poverty. Not surprisingly, then, both parents and adolescents in high poverty neighborhoods were more likely to report a desire to move away from their Wave I neighborhoods.

3.4 Conclusion

Preliminary analysis of the association between level of neighborhood poverty and perceived neighborhood environment reveal potential mechanisms linking poverty to obesity status. Neighborhood satisfaction and attachment was lower in poor neighborhoods. Perceived safety was inversely associated with level of neighborhood poverty for both neighborhood "insiders" (i.e. parents and adolescents) and "outsiders" (i.e. interviewers). Perceived lack of safety and weaker social attachment may adversely affect levels of physical activity for adolescents in high poverty neighborhoods. Consistent with the neighborhood selection literature [Sampson, 2008; Sampson & Sharkey, 2008; DeLuca et al., unpublished], the "choice" involved in neighborhood decisions appears to have been limited for parents living in high poverty neighborhoods. Since childhood neighborhood conditions are strongly correlated with adult neighborhood conditions, parents' neighborhood selection may influence adolescents' BMI in adulthood both through multiple pathways [Sharkey, 2008; Boone-Heinonen & Gordon-Larsen, 2012]. Lastly, previous findings suggest adolescent neighborhood poverty may be associated with higher BMI in adulthood. However, since these studies did not take into account the potential mediating effects of adult SES, it remains unclear whether this association operates independent of or through the accumulation of poverty in adulthood.

Table 1. Percent agreement with statements regarding neighborhood quality, safety, and satisfaction–National Longitudinal Study of Adolescent to Adult Health, 1994-1995.

	% poor families in census tract			
Neighborhood Mechanisms	**<20% (N=14,834)**	**20-39% (N=3,323)**	**≥40% (N=595)**	**X^2**
	<---------- % (standard Error) ---------->			
Neighborhood Quality and Safety				
Parent				
Litter or trash is a big problem	3.7 (0.38)	13.3 (1.22)	26.9 (4.42)	87.1 **
Drug dealers and drug users are a big problem	5.7 (0.49)	20.2 (2.23)	35.6 (5.22)	92.4 **
Adolescent				
Does not usually feel safe in current neighborhood	8.7 (0.67)	22.6 (1.74)	28.0 (4.12)	46.1 **
Neighborhood Satisfaction				
Parent				
Would very much like to move away from current neighborhood	13.1 (0.72)	24.8 (2.43)	36.9 (5.42)	65.1 **
Adolescent				
Happy to live in current neighborhood	70.9 (0.77)	60.8 (1.89)	53.7 (3.17)	53.3 **
Would be happy to move	17.3 (0.41)	22.7 (1.04)	28.8 (2.77)	64.2 **

**Significant at the P<0.01 level

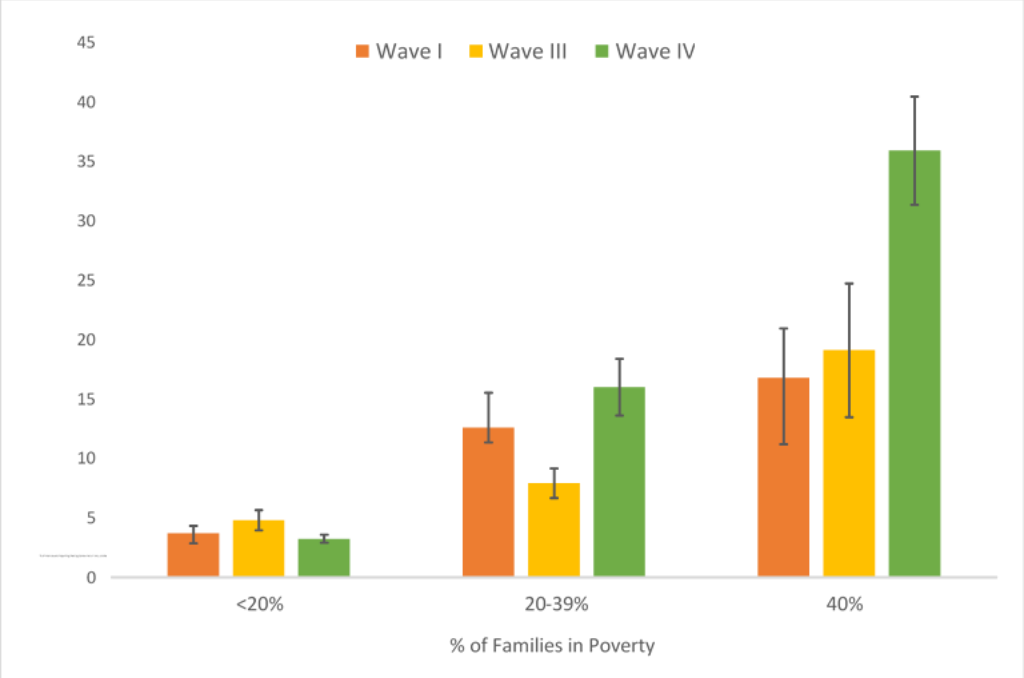

Figure 7. Percentage of Add Health interviewers reporting feel somewhat or very unsafe in the respondent's neighborhood by percent of family poverty in the census tract.

Table 2. Parental agreement with statements regarding reasons for neighborhood selection– National Longitudinal Study of Adolescent to Adult Health, 1994-1995.

	% poor families in census tract			
Reason	**<20% (N=14,834)**	**20-39% (N=3,323)**	**≥40% (N=595)**	**X^2**
	<---------- % (standard error) ---------->			
Close proximity to former workplace	23.8 (1.02)	25.9 (1.94)	16.6 (1.44)	5.9
Close proximity to current workplace	37.6 (1.27)	33.9 (1.68)	19.4 (1.94)	19.1 **
Outgrew previous housing	40.5 (1.19)	33.4 (1.60)	34.2 (3.66)	15.4 **
Affordable	51.1 (1.14)	53.4 (1.41)	43.9 (3.40)	5.2
Less crime	64.1 (1.11)	48.5 (1.88)	33.5 (3.06)	54.4 **
Less drug use and other illegal activity by adolescents	58.9 (0.84)	48.2 (2.15)	30.4 (3.01)	36.6 **
Close proximity to friends or relatives	40.1 (1.30)	49.9 (2.32)	43.0 (4.58)	15.3 **
Better schools	53.8 (2.09)	39.6 (3.32)	26.2 (3.78)	32.6 **
Children of similar ages as your own	30.6 (1.40)	27.3 (1.62)	26.1 (2.23)	5.7
You (or your spouse or partner) were born in this neighborhood	12.1 (0.97)	19.7 (4.20)	20.4 (5.17)	6.3 *

*Significant at the P<0.05 level **Significant at the P<0.01 level

CHAPTER 4: METHODOLOGICAL APPROACH

The goal of this study was to estimate the effect of exposure to neighborhood poverty across the life course, as conceptualized by three conceptual models, on adult weight status. This chapter details the research methodology including a description of the parent study, operational definitions of the exposure and outcome, and the modeling strategy. For each aim, the analysis proceeded in three general steps: 1) operationalizing and estimating the measure of exposure to neighborhood poverty as conceptualized by the life course model under study; 2) constructing statistical models to estimate the effect of neighborhood poverty on adult obesity risk; and 3) investigating modification of the exposure-outcome association by selected individual-level characteristics. Finally, as an exploratory aim, Steps #2 and Step #3 were replicated in samples stratified by neighborhood mobility status (i.e. mover versus non-mover).

4.1 Parent Study: National Longitudinal Study of Adolescent to Adult Health

The limited application of life course theories to the study of neighborhood disadvantage and obesity is largely due to the lack of contemporaneous data on residential neighborhood environments and weight status over time. The National Longitudinal Study of Adolescent to Adult Health (Add Health) is one of few nationally representative, population-based data sources for this information. The present research involves a secondary analysis of Add Health data collected in three of the four survey waves conducted between 1994 and 2008: Wave I (1994-1995); Wave III (2001-2002); and Wave IV (2008-2009). This section

provides a brief description of the design and data collection methods employed by Add Health (a detailed description is provided elsewhere; see Resnick et al., 1997 and Harris, 2009), and discusses its strengths for addressing the aims of this research.

Add Health employed a school-based sampling design to identify youth in grades 7-12 during the 1994-1995 academic year. A multi-stage, stratified random sample of 80 high schools was selected from an educational database administered by Quality Education Data, Incorporated [Harris et al., 2003]. These schools were chosen to be representative of all US high schools on the basis of region, urbanicity, number of students, type (e.g. private, public), % of white students, % of black students, grades offered, and curriculum. High schools were eligible if they enrolled a minimum of 30 students and offered 11^{th} grade. For each high school, one "feeder" school (i.e. schools that were expected to send at least 5 students to the selected high school in the following academic year) was also randomly selected to participate in the study. The feeder school's probability of selection was proportional to the number of students it sends to the high school in a typical academic year. Twenty high schools acted as their own feeder school and four schools had no feeder. A total of 144 schools in 80 communities across the US participated in the study.

There were four primary sources of baseline survey data: the administrator survey, the in-school student survey, the in-home student interview, and the parent interview. Administrators completed a mail survey to provide information on school characteristics such as student and teacher demographic composition and curriculum and services offered. Students present on the day of in-school survey administration completed a questionnaire that addressed a range of topics including individual behaviors and attitudes, parental characteristics, family dynamics, romantic partnerships, and friendship networks. Following

the in-school survey, a stratified random sample of students (73.1% in grades 9-12 and 26.9% in grades 7-8) chosen from the school roster to represent the school on the basis of grade and sex, and their parents, were selected to complete in-home interviews. Selected groups were oversampled to facilitate detailed analyses of these groups: Cubans, Puerto Ricans, Chinese, disabled, black students with at least one college-educated parent, siblings, and unrelated youth living in the same household. This in-home sample (also known as the Add Health Cohort) was re-interviewed in 1996, 2001-2002, and 2008-2009.

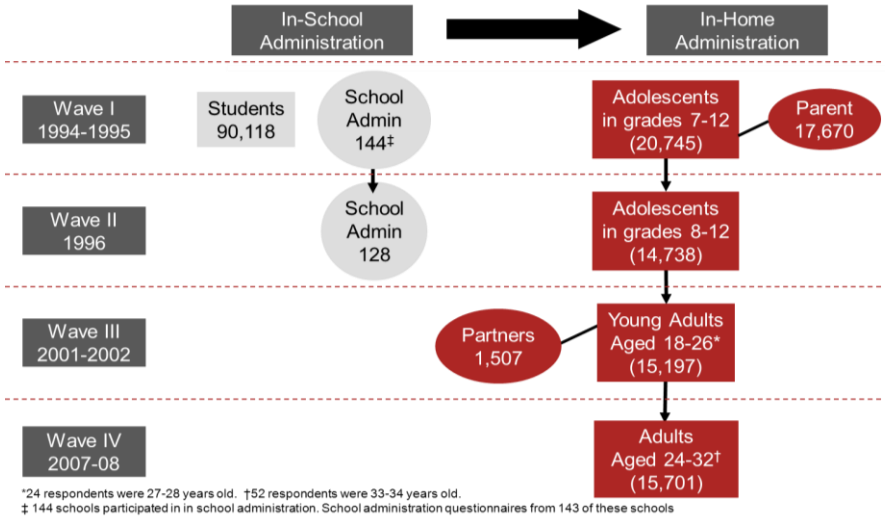

Figure 8. Longitudinal design of the National Longitudinal Study of Adolescent Health. (Source: http://www.cpc.unc.edu/projects/addhealth/design).

Figure 8 depicts the follow-up of the Add Health Cohort (n=20,745). With the exception of the disabled sample and most Wave I 12th graders, all students from this sample were eligible to participate in Wave II. Interviews were completed by 14,738 students for a participation rate of 88%. A second mail survey was also sent to school administrators who participated in Wave I and was completed by 128 out of 144 eligible administrators. In Wave III, all members of the Add Health Cohort aged 18 years or older were eligible to participate and 15,197 young adults completed in-home surveys for a participation rate of 73%. Finally,

the Add Health Cohort was re-interviewed in 2008-2009 (Wave IV). Successful contact was made with 93% of eligible study participants and 15,701 completed in-home interviews for a response rate of 80.3%.

Add Health has several strengths as it relates to the present research project. First, Add Health began at a time of high childhood obesity prevalence [Ogden et al., 2012] and, thus, represents the experiences of a generation that may be highly susceptible to obesogenic environments. Second, Add Health has maintained high retention rates and demographic diversity over the follow-up period thus reducing the potential for selection bias. Third, data on residential neighborhood environment and weight status was collected at each study wave, thus, capturing not only temporal changes in these measures, but variation across multiple stages of the life course. Unlike randomized studies, which can only be used to investigate a subset of neighborhood poverty trajectories (e.g. decreasing neighborhood poverty) for ethical reasons, Add Health is able to capture the full range of exposure patterns experienced in this cohort. Indeed, Swisher and colleagues (2013) describe substantial variation in the neighborhood poverty experiences of Add Health respondents from adolescence in 1994-1995 to early adulthood in 2008-2009. Fourth, since there is substantial heterogeneity in neighborhood poverty at each wave, our ability to detect a dose-response association between the exposure and outcome was enhanced (Figure 9). Fifth, the weighted design of Add Health makes it possible to generalize study findings to the entire source population. Lastly, Add Health collected information on a breadth of potential confounding or mediating variables including individual-level poverty status, parent's weight status, school environment, and self-reported measures of the neighborhood social environment.

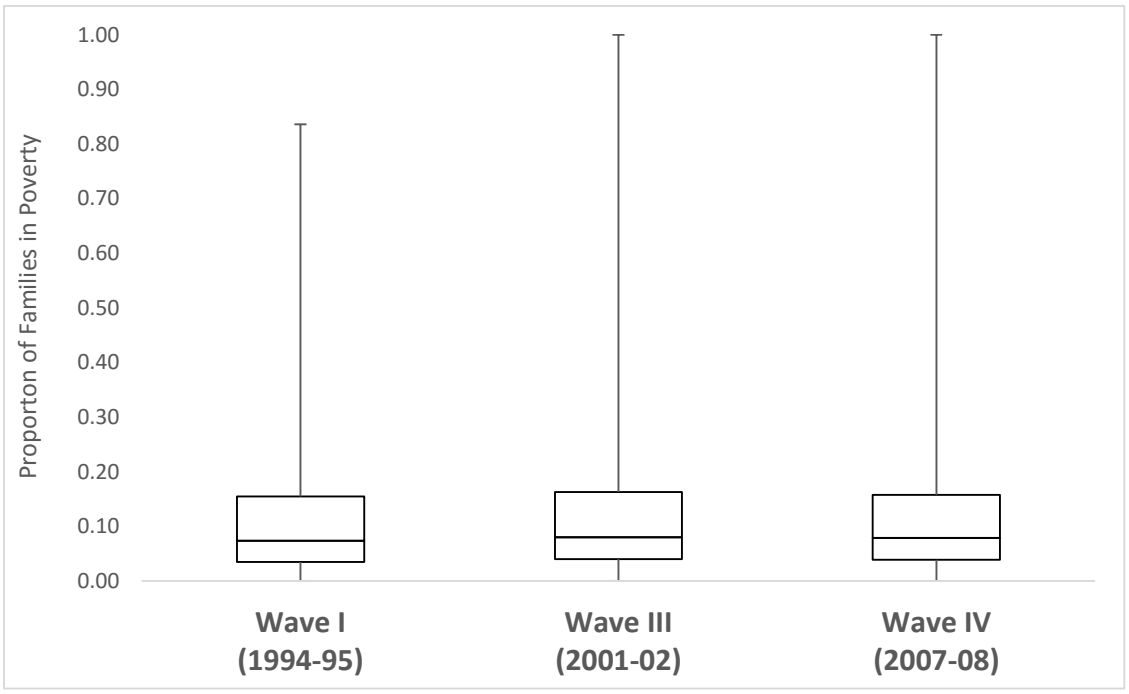

Figure 9. Distribution of family-level poverty in census tracts inhabited by National Adolescent Study of Adolescent to Adult Health (Add Health) Cohort members at Waves I, III, and IV.

4.2 Definition of Neighborhood

Respondents were assigned to a neighborhood at each study wave according to the census tract of their geocoded residential address at the time of in-home interview. A census tract is a geographic unit used by the federal government to subdivide the population in order to conduct a decennial census [US Census Bureau, 2012]. On average, census tracts contain about 4,000 persons, thus, in sparsely populated areas, the land area of tracts tend to be large compared to densely populated urban areas. Census tract boundaries have been widely used as proxies for neighborhoods in studies reporting positives associations between neighborhood environmental features and health. There were 2,865 census tracts represented in Add Health at baseline and an average of 3.4 respondents per tract. The number of tracts

represented in the data increased and the degree of geographic clustering decreased over time as respondents transitioned from the family home to independent living.

Table 3. Number of census tracts and mean respondents per census tract in Add Health by study wave.

Measure	Wave 1	Wave 3	Wave 4
# of unique tracts	2,865	4,454	5,974
# of respondents per tract			
Mean	3.4	2.2	1.6
Median	1.0	1.0	1.0
Range	1.0-87.0	1.0-85.0	1.0-65.0
Mode	1.0	1.0	1.0

4.3 Exposure Assessment

The proportion of residents within a census tract living at or below the federal poverty level (based on annual income, family size, and geographic location) is routinely reported by the decennial US Census Survey and the American Community Survey [US Census Bureau, 2015]. Tract-level family poverty is available at each wave of Add Health through prior linkage of respondents' geocoded residential locations with the 1990 and 2000 US Census Survey and the 2009 American Community Survey. Table 4 summarizes the sources of data on tract-level poverty available at each study wave. Using these data, exposure to neighborhood poverty was assessed at up to three time points (i.e. Wave I, Wave III, and Wave IV) for each member of the Add Health Cohort.

High poverty neighborhoods were defined as tracts where ≥20% of residents were poor. This measure has been widely used as an indicator of neighborhood socioeconomic disadvantage and found to predict a variety of health outcomes including body mass index

and weight status [Boardman et al., 2005; Ludwig et al. 2011; Kowaleski-Jones & Wen, 2013; McTigue et al., 2015].

Table 4. Source of data on proportion of families with annual incomes at or below poverty in the National Longitudinal Study of Adolescent to Adult Health.

Study Wave	**Data Source**
Wave I	1990 US Census
Wave II	1990 US Census
Wave III	2000 US Census
Wave IV	2009 American Community Survey

4.4 Outcome Assessment

The primary outcome of interest in this study is incident obesity. Obesity status at Wave IV (2008-2009), when all members of the Add Health Cohort had reached adulthood, was assessed using anthropometric measurements collected by trained interviewers. Per study protocol, height was measured in the Frankfort horizontal plane and weight was assessed using a high capacity (200 kg) digital bathroom scale. Height and weight were recorded to the nearest 0.5 cm and the nearest 0.1 kg, respectively. A short-term test/re-test study (N=100) found these measurements to be very reliable: the intra-class correlation coefficient was equal to 1.00 for weight and 0.98 for height [Hussey et al., 2015]. In the present study, these measurements were used to calculate BMI using the standard definition (height [kilograms] divided by weight [meters^2]) and, consistent with the WHO classification scheme, individuals with a BMI $\geq$30 kg/m^2 were classified as obese.

Obesity status at Wave I was also assessed in order to identify Add Health Cohort members with high body mass at baseline and exclude them from the study sample. Again, the standard equation was used to calculate BMI. However, since the WHO weight classes

are not appropriate for identifying obese individuals less than 18 years old, we used CDC growth percentiles for these respondents, which are the gold standard in childhood obesity research in US populations [Martin et al., 2012]. Since children and adolescents experience substantial physiological changes, and growth patterns vary greatly by age and sex, this schema uses age- and sex-specific percentiles instead of fixed values to define weight status. BMI values at or above the 95th percentile are classified as obese. In contrast to the protocol used for subsequent study waves, height and weight measurements were self-reported by the respondent at Wave I. Previous research estimates that females underreported their weight by an average of 0.86 kg, and the degree of underreporting increased with age [Clarke et al., 2014]. Evidence of underreporting weight was not found among males. However, obesity status estimated based on self-reported measures at Wave I consistently identified 96% of females identified as obese1 year later at Wave II [Kane et al., 2013]. In addition to exclusions made based on missing exposure data, participants eligible for analysis of Aims 1-3 had valid estimates of BMI at baseline (Wave I)—to eliminate cases of prevalent obesity—and at the time of outcome assessment (Wave IV).

4.5 Covariate Assessment

Add Health has amassed a wealth of individual and contextual data on each cohort member facilitating confounding control of the neighborhood poverty-obesity association in multivariable analysis. Table 5 summarizes variables (identified a priori) available in Add Health that were assessed as potential confounders and effect modifiers of the exposure-outcome associations under study.

Table 5. Sources of sociodemographic, parental, and household characteristics in the National Longitudinal Study of Adolescent to Adult Health.

Covariate	Wave I (1994-1995)		Wave III (2001-2002)	Wave IV (2008-2009)
	Parent Interview	Student Interview		
Sociodemographic Characteristics				
Age		X	X	X
Race/ethnicity		X	X	X
Sex		X	X	X
Educational attainment			X	X
Marital status			X	X
Parity		X	X	X
Pregnancy status (females only)		X	X	X
Parental Characteristics				
Race/ethnicity	X		X	X
Educational attainment	X			
Marital status	X			
Physical activity	X			
Body mass index	X			
Household Characteristics				
Income	X		X	X
Geographic region	X	X	X	X

4.6 Statistical Analysis

All analyses were conducted using STATA 14 (StataCorp LP, 2015, College Station, Texas).

4.6.1 Descriptive Analysis

Weighted descriptive statistics were calculated in order to describe the study population and provide estimates of exposure and outcome prevalence. Means and standard errors were calculated for continuous measures such as age. Prevalence estimates and standard errors were calculated for categorical variables (e.g. sex, race/ethnicity). T-tests or Rao-Scott Chi-square tests were performed to identify differences in obesity incidence and level of neighborhood poverty by individual characteristics.

4.6.2 Regression Modeling

Aims 1 and 2

The objective of the analysis was to estimate the association between accumulation of exposure to high neighborhood poverty from adolescence to adulthood (Aim 1) and patterns of exposure to high poverty neighborhoods (Aim 2) on the risk of obesity in adulthood. The directed acyclic graph (DAG) shown in Figure 10 was used to guide the modeling approach. Binomial regression models (i.e. identity link and binomial distribution) were constructed to estimate the risk difference of incident obesity (β_1) associated with life course exposure to neighborhood poverty. The general formula for models used to estimate this parameter is as follows:

$$\pi = \beta_0 + \beta_1 X + \beta_2 C$$

where π is the mean probability of becoming obese in the sample, X represents values of the neighborhood poverty measure, and C represents the set of confounders.

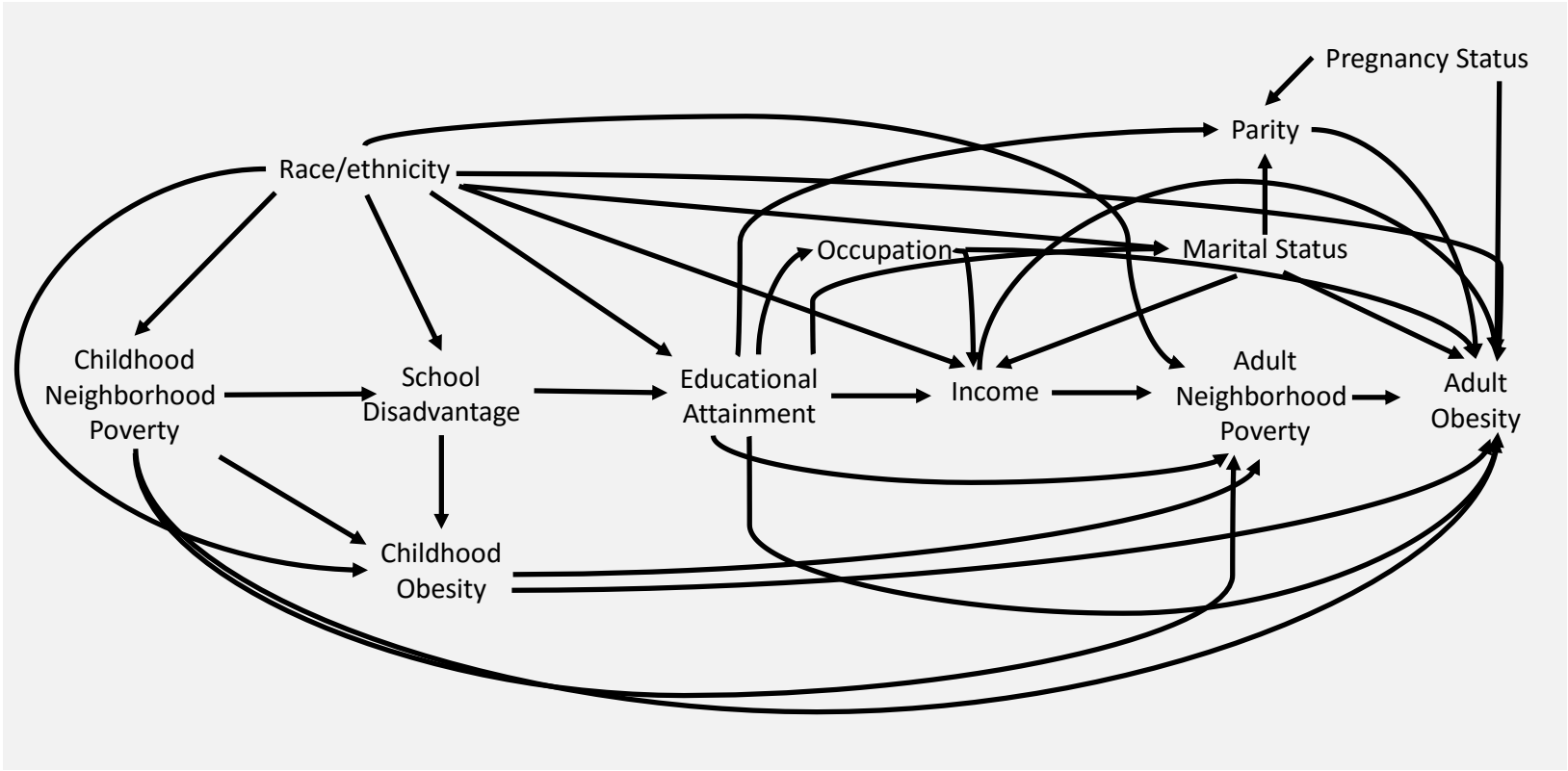

Figure 10. Directed acyclic graph of the hypothesized causal association between neighborhood poverty and obesity status in adulthood.

Aim 3

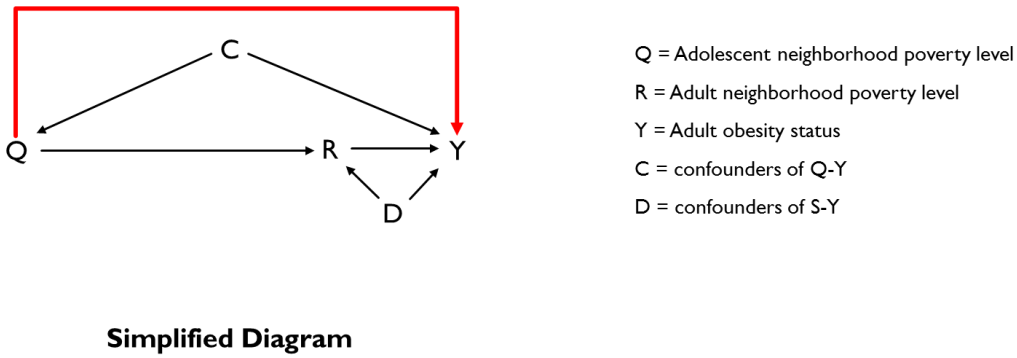

Figure 11. Simplified diagram of the controlled direct effect of adolescent neighborhood poverty on adult obesity.

Aim 3 investigated whether adolescence is a critical period for the obesogenic effects of exposure to neighborhood poverty. In order to test this hypothesis, we estimated the controlled direct effect of high neighborhood poverty on obesity in adulthood, independent of adult neighborhood poverty status, using a marginal structural modeling approach (Figure 11) [VanderWeele, 2015]. The controlled direct effect tells us the average change in the outcome associated with a 1-unit change in the exposure when the mediator (adult neighborhood poverty) is held constant across the population. Marginal structural models balance the distribution of measured confounders of the exposure-outcome and the mediator-outcome relationships across levels of the exposure using weights. This approach produces an unbiased estimate of the effect of the exposure on the outcome in the absence of (unmeasured) confounding of the exposure-outcome and mediator-outcome associations. The method proceeds in two stages: 1) calculation of inverse probability weights and 2) specifying regression models to estimate the exposure-outcome association.

Each individual in the analytic sample was assigned a weight based on their observed values of: the exposure (high neighborhood poverty during adolescence), the mediator

(neighborhood poverty status in adulthood), and confounders of both exposure-outcome and mediator-outcome relationships. First, an exposure weight (w_i^Q) was calculated for each individual as the marginal probability of exposure to neighborhood poverty during adolescence (Q) taking on an observed value (q) divided by this same probability conditional on observed values (c) of a set of confounders (C). Next, the numerator for the mediator weight (w_i^R) was calculated for each individual as the probability of adult neighborhood status (R) taking on an observed value (r) conditional on the probability of adolescent neighborhood status (Q) taking on the observed value (q). The denominator of mediator weight was calculated as the probability of adult neighborhood status (R) taking on an observed value (r) conditional on the probability of adolescent neighborhood status (Q) taking on the observed value (q), observed values (c) of a set of confounders of the exposure-outcome association (C), and observed values (d) of a set of confounders of the mediator-outcome association (D). Finally, the final weight for each individual (w_i) was calculated as the product of the exposure weight (w_i^Q), the mediator weight (w_i^R), and the survey sampling weight (sw) [Brumback et al., 2010].

$$w_i^Q = P(Q = q_i) \div P(Q = q_i \mid C = c_i)$$

$$w_i^R = P(R = r_i \mid Q = q_i) \div P(R = r_i \mid Q = q_i, C = c_i, D = d_i)$$

$$w_i = wi^Q \; x \; w_i^R \; X \; sw_i$$

Estimating the controlled direct effect of adolescent neighborhood poverty on adult obesity, then, proceeded in a similar manner as in Aims 1 and 2 with the exception only the exposure and mediator were included as independent variables in the model. Based on previous recommendations for constructing binomial marginal structural models, robust 95% confidence intervals were calculated [Robins et al., 2000; Stefanski & Boos, 2002].

Assessing Confounding and Effect Modification

Confounding and effect modification of the relationship between exposure to neighborhood poverty and adult obesity risk by the covariates found in Table 5 was assessed. Since an overarching aim of this research was to determine whether socioeconomically disadvantaged subgroups of the population are more vulnerable to the adverse effects of exposure to poor neighborhoods, we assessed interaction between the exposure and both race/ethnicity and sex. A threshold of $p<0.20$ was used to identify statistically significant effect modification. Then, each variable in Table 5 was assessed as a potential confounder of the exposure-outcome association by modeling the bivariate associations between the potential confounder and the exposure and the potential confounder and the outcome. Variables found to have a statistically significant association ($p<0.05$) with both the exposure and outcome (or the outcome alone) were retained in the full model.

4.7 Sensitivity Analyses

As an exploratory aim, we compared the estimates of effect produced in each aim between movers (i.e. respondents who changed neighborhoods) and non-movers (i.e. respondents who changed neighborhoods). A "neighborhood change" was defined as a residential location at any follow-up interview that was more than 5 miles from the respondents' residential location at Wave I (1994-1995). This step was used to validate the results from the main analyses because non-movers may be a lower SES group compared to movers (Table 6). Additionally, there is less ambiguity regarding the temporal ordering of exposure, mediators, and the outcome among the residentially immobile. Furthermore, since prior studies [Morris et al., 2015] have reported a positive association between frequent residential mobility and BMI in childhood, this step can help disentangle the effects of neighborhood socioeconomic mobility (i.e. experiencing varying levels of neighborhood

poverty across the life course) from the effects of neighborhood residential mobility (i.e. moving from one neighborhood to another), in general.

Table 6. Demographic characteristics of movers and non-movers in the National Longitudinal Study of Adolescent to Adult Health Cohort.

Characteristic	Movers (N=8,213)		Non-movers (N=3,944)	
	N	Weighted %	N	Weighted %
Mean census tract poverty**	8,213	13.6	3,944	16.7
Age, years (mean)	8,213	16.0	3,944	15.8
Sex				
Female	4,496	50.0	2,116	47.5
Male	3,717	50.0	1,828	52.5
Race/ethnicity**				
Non-Hispanic white	5,024	72.3	1,729	59.1
Non-Hispanic black	1,496	13.7	990	19.3
Hispanic	1,059	9.4	847	15.9
Non-Hispanic Asian	502	3.1	290	3.5
Non-Hispanic other race	127	1.5	86	2.2
Born in the US				
No	579	4.9	329	5.9
Yes	7,633	95.1	3,614	94.1
Parents' educational attainment**				
Less than high school	977	13.9	772	22.2
High school diploma or equivalent	1,994	31.4	1,059	34.8
Some college or technical school	2,177	30.2	913	27.3
College degree (Associate's or Bachelor's)	1,161	14.3	425	11.4
Advanced degree or training	843	10.1	195	4.4
Parent's marital status				
Married	5,617	77.7	2,563	76.0
Unmarried	1,562	22.3	815	24.0
Received public assistance income*				
No	6,648	91.9	3,018	87.9
Yes	501	8.1	357	12.1

*Distribution among movers and non-movers different at the $P<0.001$ level.
**Distribution among movers and non-movers different at the $P<0.0001$ level.

4.8 Conclusion

The study employed a novel approach to investigate the life course effects of neighborhood poverty on adult obesity using existing longitudinal data from a nationally representative, population-based cohort. By integrating theories from other disciplines, this study strengthens the conceptual basis for the hypothesized results. Second, this study brings methodological innovation by utilizing a marginal structural modeling approach to direct effect estimation. Lastly, by taking into consideration the distinct phenomenon that can give rise to change in one's neighborhood environment (i.e. residential mobility versus change in the neighborhood itself), this study provides even further insight into the types of policy interventions that may improve health outcomes for the most vulnerable populations.

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CHAPTER 5: AIM 1 AND AIM 2

5.1 Introduction

The neighborhood environment has been implicated in the development of obesity [McNeill et al., 2006; Lee et al., 2¬009; Carter & Dubois, 2010; Wells et al., 2010; Ding et al., 2011; Kimbro et al., 2011; Ludwig et al., 2011; De Vet et al., 2012; Ding & Gebel, 2012]. Residential neighborhoods help play a role in regulating access to material and social resources that can influence health behaviors and outcomes [Sharkey & Faber, 2014]. For example, relative to more affluent neighborhoods, poor neighborhoods tend to lack access to physical activity resources and healthful foods and have greater access to foods of low nutritional quality [Walker et al., 2010; Boone-Heinonen et al., 2013]. Additionally, poor neighborhoods often have higher crime rates, which may cause residents to feel less safe and socially connected to neighbors [Quillian, 2003]. This may discourage outdoor recreation and lead to a more sedentary lifestyle. Despite evidence of plausible mechanisms linking neighborhood poverty to weight-related outcomes, however, conflicting studies fuel debate over whether these associations represent causal effects or merely reflect the selection of people with similar backgrounds and health profiles into certain types of neighborhoods [Oakes, 2004; Black & Macinko, 2008].

The lack of integration of life course theory [Elder, 1998] and the predominance of cross-sectional study designs in the neighborhood effects literature limits our understanding of the obesogenic effects of neighborhood poverty. Disentangling neighborhood contextual

effects from the effects of neighborhood selection factors in a cross-sectional framework is difficult [Oakes, 2004]. Additionally, point-in-time measures of neighborhood exposures may be inadequate to capture the period(s) relevant to the development of obesity. Moreover, Americans are highly mobile and demonstrate considerable variation in their neighborhood environment over time [Quillan, 2003; Robert et al., 2010]. Therefore, ignoring neighborhood history or using a single measure to represent dynamic exposures may obscure the distribution of neighborhood experiences in a population and misestimate the effect of neighborhood poverty on obesity [Do et al., 2012]. Furthermore, viewing neighborhood effects through a cross-sectional lens precludes investigation into the role of exposure pattern and the accumulation of neighborhood poverty across the life course.

Understanding the health effects of life course neighborhood poverty can help identify protective factors capable of breaking the chain of risk before obesity develops [Evans et al., 2012]. Prior research demonstrated that long-term neighborhood poverty experiences were associated with weight gain over a 20-year period among women in an older adult cohort (45-84 years at baseline) [Murray et al., 2010]. Since weight-related behaviors are established early in life [Dietz, 1994; Harris & Bargh, 2009; Wells et al., 2010] and track into adulthood [Gordon-Larsen et al., 2004; Nelson et al., 2005; Biddle et al., 2010], similar studies are needed in younger cohorts to understand the effects of neighborhood poverty across the early life course on weight status. The present study adds to the current literature by investigating the neighborhood poverty-obesity relationship in the National Longitudinal Study of Adolescence to Adult Health (Add Health). Add Health followed a national cohort of school-attending youth in the US from 1994-1995, when most respondents were adolescents, to adulthood in 2008-2009. We hypothesized that the

accumulation of exposure to high poverty neighborhoods and neighborhood poverty trajectories characterized by persistent exposure to high poverty would be associated with an increased risk of becoming obese.

5.2 Methods

5.2.1 Sample

Add Health is an ongoing study of American youth enrolled in 7-12th grade during the 1994-1995 academic year. The study recruitment and data collection methods used in Add Health have been previously described [Resnick et al., 1997; Harris, 2010]. In brief, a stratified random sample of 80 schools was selected from an educational database to be representative of high schools in the US. Then, for each high school, one "feeder" school was selected proportional to the student contribution to the high school, resulting in a pair of schools in 80 communities. A core sample of students was chosen from the participating schools to be representative of each school based on grade level and sex with oversamples of certain demographic groups. This core sample of students (N=20,745), as well as a resident parent or guardian (93% of whom were the respondent's mother/step-mother), completed in-home interviews at baseline. Students were re-interviewed approximately 1 year (in 1996), 7 years (in 2001-2002), and 14 years (in 2008-2009) later. This analysis draws on data collected at the baseline (parent and student), Year 7, and Year 14 interviews. We excluded respondents who did not participate in each of the three study waves included in the analysis (N=8,457), had missing information on neighborhood poverty (N=382), or had an unknown weight status at the final interview (N=575). Additionally, we identified respondents who were already obese at the start of the study (N=1,488) based on self-reported height and weight, and limited the analysis to the non-obese. After these exclusions, there were 9,843 respondents eligible for the analysis.

5.2.2 Measures

Obesity

Trained interviewers collected anthropometric measures including weight and height at the Year 14 follow-up visit [Entzel et al., 2009]. BMI was calculated as height (in kg) divided by weight (in m2) and, since all respondents had reached adulthood by the time of the final interview, obesity was defined as a BMI ≥30 kg/m2 [WHO Expert Committee on Physical Status, 1995].

Life course neighborhood poverty

The percentage of residents in a census tract living at or below the federal poverty level has been widely used as an indicator of area-level socioeconomic disadvantage, and found to predict a variety of health outcomes including body mass index and weight status [Boardman et al., 2005; Ludwig et al. 2011; Kowaleski-Jones & Wen, 2013; McTigue et al., 2015]. Census tract poverty was available in Add Health through prior linkage of geocoded residential addresses to contextual data from administrative databases. Respondents' census tracts at the time of baseline and Year 7 interviews were linked to the 1990 and 2000 US Census Surveys, respectively. Year 14 census tracts were linked to geographic data from the 2005-2009 American Community Survey.

Prior research has demonstrated that individuals who resided in census tracts with ≥20% poverty were at a greater risk for poor health [Do and Finch, 2008]. Therefore, respondents' exposure to neighborhood poverty was classified as either high (≥20% residents living below the federal poverty level) or low (<20% residents below the federal poverty level) at each study wave using this threshold. Then, we constructed two life course measures of neighborhood poverty to represent respondents' 1) cumulative exposure to neighborhood

poverty and 2) pattern of exposure to neighborhood poverty from baseline to final interview (Year 14).

A cumulative neighborhood poverty score was calculated by summing the number of study waves a person was exposed to high neighborhood poverty. For example, respondents were assigned a score of 0 if they did not reside in a high poverty neighborhood at the time of any of the 3 interviews and were assigned a score of 1 if they resided in a high poverty neighborhood at only 1 of the 3 interviews. Preliminary analysis indicated that scores of 2 and 3 were similarly associated with the risk of obesity, therefore, these groups were collapsed into a single category (cumulative neighborhood poverty score ≥2) for the analysis.

Trajectories of neighborhood poverty were categorized based on respondents' combined neighborhood poverty experiences at the baseline and final interviews. A stable low trajectory indicated that the respondent lived in a low poverty neighborhood at both time points. Upwardly mobile and downwardly mobile trajectories include respondents going from high to low and low to high poverty neighborhoods, respectively. Finally, a stable high trajectory indicated that the respondent lived in a high poverty neighborhood at both time points.

Race/ethnicity and sex

Sex (male/female) and race/ethnicity were reported by respondents during the baseline interview. Race/ethnicity was categorized as white non-Hispanic, black non-Hispanic, Hispanic, Asian non-Hispanic, or other race non-Hispanic.

5.2.3 Covariates

Baseline characteristics of the student and parent respondent were included as covariates in multivariable regression models to control for selection factors that could influence both neighborhood poverty and obesity status. Age (continuous) was calculated

using the student's date of birth. Parents reported their highest level of schooling completed, the highest level of schooling completed by their resident spouse or partner (if applicable), and whether they received any public assistance income during the 1994 calendar year (yes/no). The educational attainment of the resident parent/guardian with the highest level of schooling was grouped into 5 categories (less than high school, high school diploma or equivalent, some college or trade/technical school, associate's or bachelor's degree, and advanced degree or training).

5.2.4 Statistical analysis

We calculated the weighted distribution of cohort members' demographic, household, and parental characteristics, and obesity status. The prevalence of exposure to neighborhood poverty (based on both cross-sectional and life course measures) was compared across sex and racial/ethnic groups. Binomial regression models were constructed to estimate adjusted risk differences of obesity associated with exposure to neighborhood poverty. Prior research has demonstrated that exposure to disadvantaged neighborhoods varies considerably across racial/ethnic groups in the US—even at similar levels of individual socioeconomic status [Messer et al., 2009]. Furthermore, the health effects of area-level poverty have been found to differ across both sex and racial/ethnic groups [Carson et al., 2007; Lemelin et al., 2009; Murray et al., 2010]. Therefore, we also investigated the neighborhood poverty-obesity association by sex and race/ethnicity.

We performed sensitivity analyses to evaluate the robustness of our findings. First, we investigated modification of the risk difference by respondents' neighborhood mobility status (whether the respondents' residential location at any follow-up interview was more than 5 miles from their residential location in 1994-1995). Second, we replicated the analysis

without exclusion of respondents who were already obese at baseline. These analyses provide insight into the potential biasing of the effect estimates due to the selection criteria.

All analyses were conducted using STATA 14 (StataCorp LP, College Station, TX). Standard errors were calculated using Taylor linearization to account for the complex survey design and sampling weights were used in all analyses (StataCorp, 2015).

5.3 Results

Respondents resided in 2,864 census tracts at baseline and 5,974 census tracts by the time of final interview14 years later and the number of respondents per tract averaged 3.4 and 1.6, respectively. The sample was fairly evenly distributed between urban (51.8%) and rural (48.2%) tracts. The mean level of neighborhood poverty in tracts where cohort members resided was 14.0% at baseline and remained relatively unchanged over the study period. However, the maximum level of neighborhood poverty increased from 76.5% at baseline to 93.2% in Year 7 and 100% in Year 14.

Table 7 summarizes the baseline characteristics of cohort members. The mean age at baseline was 16.0 years. Most respondents were born in the US (94.5%) and non-Hispanic whites made up 69.0% of the sample; 14.2% were non-Hispanic black; 11.6% were Hispanic; 3.7% were non-Hispanic Asian; and 1.5% were non-Hispanics from other racial groups. The majority of respondents (69.1%) changed neighborhoods at least once during the study period. Slightly more than half of respondents had a resident parent/guardian who completed schooling beyond a high school diploma or equivalent (53.1%), was married (78%), and reported no receipt of public assistance income during the 1994 calendar year (84%).

The 14-year incidence of obesity in this previously non-obese sample was 30.4%. Baseline factors associated with becoming obese were being female, older age, non-Hispanic black or Hispanic race/ethnicity, being born in the US, and lower socioeconomic status (i.e.

having a parent respondent who was unmarried or received public assistance income in 1994, or not having a resident parent/guardian with more than a high school education).

Most respondents resided in neighborhoods of relatively low poverty throughout the study period (Table 8). At the time of baseline and final (Year 14) interviews, less than a quarter of the sample lived in poor neighborhoods, and the majority had no exposure to neighborhood poverty over the entire 14-year period (55.3%). While most respondents had a stable low neighborhood poverty trajectory (63.6%), nearly 24% lived in impoverished neighborhoods at either the baseline or final interview. There were no sex differences in neighborhood poverty, but neighborhood poverty experiences varied drastically across racial/ethnic groups. Compared to non-Hispanic whites and non-Hispanic Asians, Hispanics and non-Hispanic blacks were more likely to reside in poor neighborhoods. Non-Hispanic Asians were the most likely to have never lived in a poor neighborhood (66.4%), followed by non-Hispanic whites (64.0%), Hispanics (43.4%), and non-Hispanic blacks (19.6%). Non-Hispanic blacks were the only group to be more likely to experience a stable high neighborhood poverty trajectory (37.1%) than a stable low trajectory (25.6%). Furthermore, a higher proportion of non-Hispanic blacks and Hispanics experienced (upward or downward) neighborhood poverty mobility over the 14-year study period.

Table 9 summarizes risk estimates of the association between cross-sectional and life course measures of exposure to neighborhood poverty and incident obesity. In general, estimates were slightly attenuated after adjustment for baseline characteristics. Living in an impoverished neighborhood at baseline was associated with 8 additional cases of obesity per 100 respondents 14 years later (risk difference [RD]: 0.08; 95% confidence interval [CI]: 0.06, 0.12) after controlling for baseline age, nativity, sex, and baseline socioeconomic

status. However, there was no association between neighborhood poverty status at the final interview and obesity (RD: 0.03; 95% CI: 0.00, 0.06). Multiple exposures to neighborhood poverty (i.e. score ≥2) was associated with an increased 14-year risk of obesity (RD: 0.07; 95% CI: 0.03, 0.10). Both upwardly mobile (RD: 0.08; 95% CI: 0.03, 0.12) and stable high (RD: 0.10; 95% CI: 0.05, 0.14) trajectories of neighborhood poverty were associated with similar increments in obesity risk.

Figure 12 illustrates adjusted risk difference estimates of the effect of cumulative neighborhood poverty on incident obesity by race/ethnicity. The association between life course neighborhood poverty and incident obesity was mostly limited to non-Hispanic whites. There was no evidence of an association between life course neighborhood poverty and obesity among non-Hispanic blacks and Hispanics in age-adjusted only models (data not shown), nor models accounting for age and baseline sociodemographic characteristics. Similarly, both upwardly mobile and stable high trajectories of exposure to neighborhood poverty were associated with increased risk of obesity among non-Hispanic whites, but not other racial groups or Hispanics (Figure 13). In general, there were no sex differences in the association between cumulative poverty and neighborhood poverty trajectory and the 14-year incidence of obesity (Table 10).

The results of our sensitivity analyses provide information about the association between life course neighborhood poverty and obesity. Respondents who did not change neighborhoods (non-movers) were more likely to live in high poverty neighborhoods at the time of baseline and final interviews, had more exposures to high poverty neighborhoods, and were more likely to have a stable high neighborhood poverty trajectory (Table 11). The observed trends between cumulative neighborhood poverty and neighborhood poverty

trajectory on the 14-year incidence of obesity were observed among movers (Table 12). However, there was no association between life course neighborhood poverty, neither cumulative dose nor trajectory, and obesity among non-movers. In the second set of sensitivity analyses, where we replicated the main analysis in a sample that included individuals who were already obese at baseline, the results remained largely unchanged (Figures 14 and Figure 15).

5.4 Discussion

We found evidence of an association between life course neighborhood poverty and obesity risk in this prospective cohort of American adolescents. Specifically, having a cumulative neighborhood poverty score ≥2 and both upwardly mobile and stable high trajectories of neighborhood poverty were associated with the 14-year risk of obesity among non-Hispanic whites. Furthermore, we showed that the association between neighborhood poverty and obesity was smaller (and non-significant in adjusted models) when both statuses were assessed at the final interview, when all respondents had reached adulthood. Instead, the risk of becoming obese was associated with respondents' baseline (in 1994-1995) and life course neighborhood poverty (from 1994-1995 to 2008-2009).

Nearly a quarter of respondents did not fall into one of the stable neighborhood poverty trajectory groups. This suggests that a single measure would do a poor job of representing exposure to neighborhood poverty in life course studies of area health effects. The extent of exposure misclassification would be even greater for non-Hispanic blacks and Hispanics who were more likely to experience upward or downward neighborhood socioeconomic mobility. This finding stands in contrast to previous research in older adults suggesting cross-sectional measures of neighborhood poverty might represent exposure history reasonably well [Murray et al., 2010].

We have extended prior research demonstrating that neighborhood disadvantage is associated with weight status by showing that the accumulation and pattern of residence in poor neighborhoods also matters. Cumulative "dose" of exposure to neighborhood poverty was associated with a greater risk of becoming obese with an apparent threshold effect at 2 or more exposures across the early life course. Additionally, both upwardly mobile and stable high trajectories of neighborhood poverty were associated with a greater 14-year risk of obesity. Since these trajectory groups include individuals who resided in high poverty neighborhoods around the time of adolescence, this finding may be indicative of a critical or sensitive period in the neighborhood poverty-obesity relationship. This hypothesis should be more thoroughly examined in future studies using mediation analysis techniques. Furthermore, given that critical periods for diet and eating habits occur as early as infancy [Dietz, 1994], similar research is needed in children to better understand the developmental origins of obesity.

Contrary to what was expected, we did not observe an association between neighborhood poverty and obesity incidence or prevalence among non-Hispanic blacks or Hispanics. These respondents were more likely to live in impoverished environments at baseline and throughout their life course, and previous studies have reported a higher baseline prevalence of obesity among non-Hispanic blacks and Hispanics [Nicholson & Browning, 2012]. Therefore, it is possible that factors occurring earlier in childhood play a more important role in adult weight status in these populations. Alternatively, other aspects of the neighborhood environment such as the availability of convenience stores [Lee, 2012], or other environmental contexts such as schools, may be more salient to health for these groups. Finally, there may be differential misclassification of exposure to neighborhood

poverty by race/ethnicity. We know from a well-established literature that a high degree of racial/ethnic segregation exists between neighborhoods across the US [Massey & Denton, 1993]. Therefore, it is possible that, even within the same low poverty census tract, non-Hispanic whites, non-Hispanic blacks, and Hispanics do not occupy the same space and the latter two groups are more likely to be surrounded by poverty.

Neighborhood poverty could influence obesity in non-Hispanic whites through several mechanisms. Prior studies have shown that residents of disadvantaged neighborhoods have greater access to convenience stores and lower access to recreational facilities [Richardson et al., 2014]; are more likely to have a poor quality diet [Rummo et al., 2015]; and are less physically active [Boone-Heinonen et al., 2011; Cubbin et al., 2006; Shishehbor et al., 2008]. Furthermore, disparities in access to food and physical activity resources, especially in early life when these behavioral preferences are being established, may potentially increase one's vulnerability to environmental obesogens. Future work should seek to illuminate the life course mechanisms linking neighborhood poverty to weight status, and understand why these mechanisms might operate differently across racial/ethnic groups.

As previously mentioned, limitations of this study include the potential for bias and misclassification. While we controlled for a number of baseline characteristics that may have influenced parents' choices and constraints on neighborhood selection as well as the respondents' adult obesity status, other factors (e.g. parents' food or recreation preferences) may not have been fully accounted for in this study. Thus, this study is not making causal claims about the role of neighborhood poverty on the incidence of subsequent obesity, but rather provides evidence that living in a poor neighborhood may influence adult health even before individuals are capable of choosing their own neighborhood. We attempted to reduce

the potential for reverse causality by excluding individuals who were already obese at the start of the study from the main analysis; however, life course neighborhood poverty experiences may have still been influenced by respondents' baseline BMI. On the other hand, we presumed that excluding respondents who were already obese at baseline may bias our estimates toward the null given that this group is more likely to include individuals exposed to extreme poverty or those most susceptible to the obesogenic effects of poor neighborhoods. However, the results of this analysis demonstrated that this aspect of the study design was not an important source of selection bias. In contrast, we found that the neighborhood poverty-obesity association differed between movers and non-movers. Jones (2015) previously reported that Add Health respondents who changed neighborhoods differed from those who did not move in ways that influence both neighborhood poverty experiences and obesity risk. That positive associations between neighborhood poverty and obesity were observed only among movers suggests residual confounding may be present. Indeed, our own analyses indicated that movers were more likely than non-movers to have some college education at the time of final interview (data not shown). Lastly, given the young age of the Add Health Cohort and the age of peak obesity prevalence in the US [Robinson et al., 2010], we may not have identified some cohort members who will eventually become obese later in adulthood.

This is the first study to measure the association between cumulative neighborhood poverty and trajectories of neighborhood poverty and obesity in the early life course. There are several strengths of this research including the use of a national, population-based sample; the high response rate at follow-up study waves; and the rich data available on

important confounders. Furthermore, the research questions under investigation were grounded in well-established theories of the life course social environment and health.

Among the main contributions of this study is the finding that neighborhood poverty experiences across the life course are associated with the risk of obesity among non-Hispanic whites in the US. This suggests efforts to curb the American obesity epidemic may require more interventions targeting the early life environment. Future research should aim to describe the neighborhood mechanisms underlying the associations described in the present study in order to improve our understanding of how to create and maintain healthier communities. The field would also benefit from research on protective factors that render individuals less vulnerable to obesogenic neighborhoods.

Table 7. Baseline characteristics of study sample, National Longitudinal Study of Adolescent to Adult Health Cohort, 1994-1995 (N=9,843).

Characteristic	N	Weighted %
Mean census tract poverty	2,864	14.0
Age, years (mean)	9,843	16.0
Sex		
Female	5,263	51.3
Male	4,580	48.7
Race/ethnicity		
Non-Hispanic white	5,592	69.0
Non-Hispanic black	1,891	14.2
Hispanic	1,521	11.6
Non-Hispanic Asian	679	3.7
Non-Hispanic other race	155	1.5
Born in the US		
No	790	5.5
Yes	9,053	94.5
Parents' educational attainment		
Less than high school		
High school diploma or equivalent	1,352	15.5
Some college or technical school	2,463	31.5
College degree (Associate's or	2,513	28.9
Bachelor's)	1,306	14.5
Advanced degree or training	886	9.7
Parent's marital status		
Married	6,672	78.0
Unmarried	1,884	22.0
Received public assistance income		
No	7,877	91.8
Yes	648	8.2
Mobility status		
Changed neighborhoods	6,752	69.1
Did not change neighborhoods	3,088	30.9

Table 8. Prevalence (95% confidence interval) of exposure to neighborhood poverty by sex and racial/ethnic group in the National Longitudinal Study of Adolescent to Adult Health Cohort (N=9,843).

Neighborhood poverty measure	All (N=9,843)	Sex		Race/ethnicity				
		Male (N=4,580)	Female (N=5,263)	NH white (N=5,592)	NH black (N=1,891)	Hispanic (N=1,521)	NH Asian (N=679)	NH other (N=155)
Lived in high poverty neighborhood at baseline	24.3 (18.8, 29.9)	23.9 (18.4, 29.4)	24.8 (18.9, 30.7)	15.3 (9.4, 21.1)	59.4 (50.0, 68.8)	38.7 (28.5, 49.0)	13.8 (5.4, 22.3)	23.9 (9.7, 38.2)
Lived in high poverty neighborhood at Year 14	24.6 (21.2, 28.0)	24.6 (21.1, 28.1)	24.6 (21.0, 28.3)	18.7 (15.3, 22.0)	52.0 (46.6, 57.4)	29.4 (23.5, 35.3)	14.0 (8.7, 19.2)	28.8 (17.0, 40.6)
Cumulative neighborhood poverty score								
0	55.3 (50.1, 60.6)	55.2 (50.0, 60.4)	55.5 (49.8, 61.1)	64.0 (58.4, 69.6)	19.6 (14.4, 24.8)	43.4 (34.1, 52.7)	66.4 (55.4, 77.4)	61.4 (47.8, 75.0)
1	24.4 (22.1, 26.7)	25.0 (22.3, 27.6)	23.8 (21.2, 26.4)	24.2 (21.3, 27.2)	26.8 (21.9, 31.8)	24.5 (20.8, 28.2)	19.8 (12.7, 26.8)	18.7 (10.0, 27.4)
≥2	20.3 (16.0, 24.6)	19.8 (15.6, 24.0)	20.7 (16.1, 25.3)	11.8 (7.9, 15.7)	53.5 (44.8, 62.2)	32.2 (23.5, 40.9)	13.8 (6.6, 21.1)	19.9 (7.3, 32.6)
Neighborhood poverty trajectory group								
Stable low	63.6 (58.2, 69.0)	63.9 (58.5, 69.3)	63.3 (57.4, 69.1)	72.8 (67.1, 78.6)	25.6 (19.0, 32.3)	51.0 (41.3, 60.8)	77.2 (68.5, 86.0)	64.1 (50.5, 77.7)
Upwardly mobile	11.8 (9.1, 14.4)	11.4 (8.7, 14.2)	12.1 (9.2, 15.0)	8.6 (5.5, 11.7)	22.2 (18.9, 25.5)	19.5 (14.3, 24.8)	8.8 (2.8, 14.8)	7.1 (2.4, 11.7)
Downwardly mobile	12.0 (10.5, 13.5)	12.1 (10.3, 13.9)	11.9 (10.2, 13.6)	11.8 (10.1, 13.6)	15.0 (11.1, 18.9)	10.2 (7.8, 12.6)	8.9 (5.4, 12.4)	11.9 (3.9, 20.0)
Stable high	12.6 (9.2, 16.0)	12.5 (9.1, 16.0)	12.7 (9.2, 16.2)	6.8 (3.5, 10.1)	37.1 (29.3, 45.0)	19.3 (13.2, 25.4)	5.1 (0.7, 9.4)	16.9 (5.0, 28.8)

Abbreviations: NH, non-Hispanic.

Table 9. Risk differences of obesity associated with exposure to neighborhood poverty over a 14-year period in the National Longitudinal Study of Adolescent to Adult Health Cohort (N=9,843).

Measure of neighborhood poverty	Crude RD	95% CI	Adj RD[a]	95% CI
High neighborhood poverty in 1994-1995	0.10	0.07, 0.13	0.08	0.06, 0.12
High neighborhood poverty in 2008-2009	0.05	0.02, 0.07	0.03	0.00, 0.06
Cumulative neighborhood poverty score				
0	Referent	--	Referent	--
1	0.01	-0.02, 0.04	0.01	-0.02, 0.04
≥2	0.09	0.05, 0.12	0.07	0.03, 0.10
Neighborhood poverty trajectory				
Stable low	Referent	--	Referent	--
Upwardly mobile	0.10	0.06, 0.14	0.08	0.03, 0.12
Downwardly mobile	0.01	-0.02, 0.05	0.00	-0.04, 0.04
Stable high	0.10	0.06, 0.15	0.10	0.05, 0.14

Abbreviations: Adj, adjusted; CI, confidence interval; RD, risk difference.
[a]Models adjusted for age, sex, nativity, and baseline socioeconomic position (parent's education, marital status, and receipt of public assistance income).

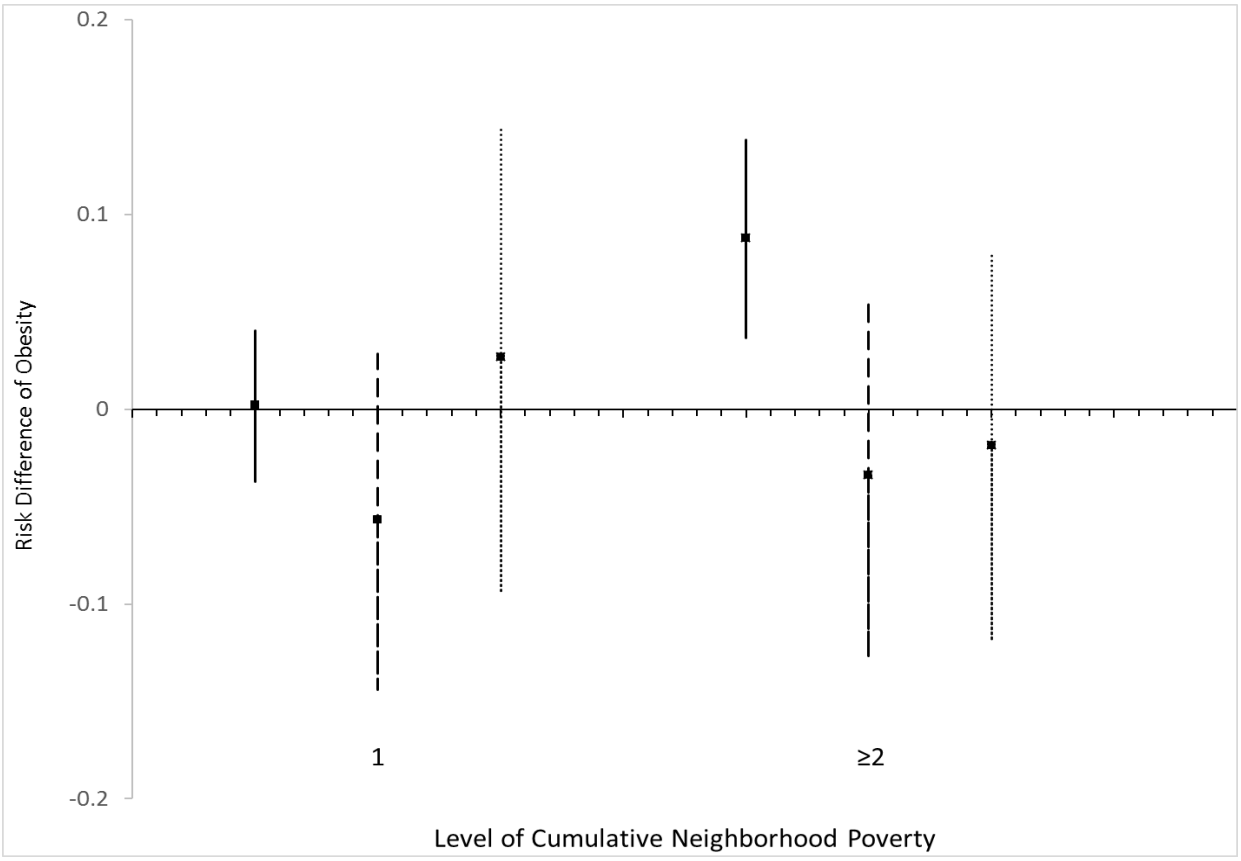

Figure 12. Adjusted risk differences of obesity, and 95% confidence intervals, for level of cumulative neighborhood poverty compared to no exposure. Models adjusted for age, sex, nativity, and baseline socioeconomic position (parent's education, marital status, and receipt of public assistance income). Solid line: non-Hispanic whites; dashed line: non-Hispanic blacks; dotted line: Hispanics.

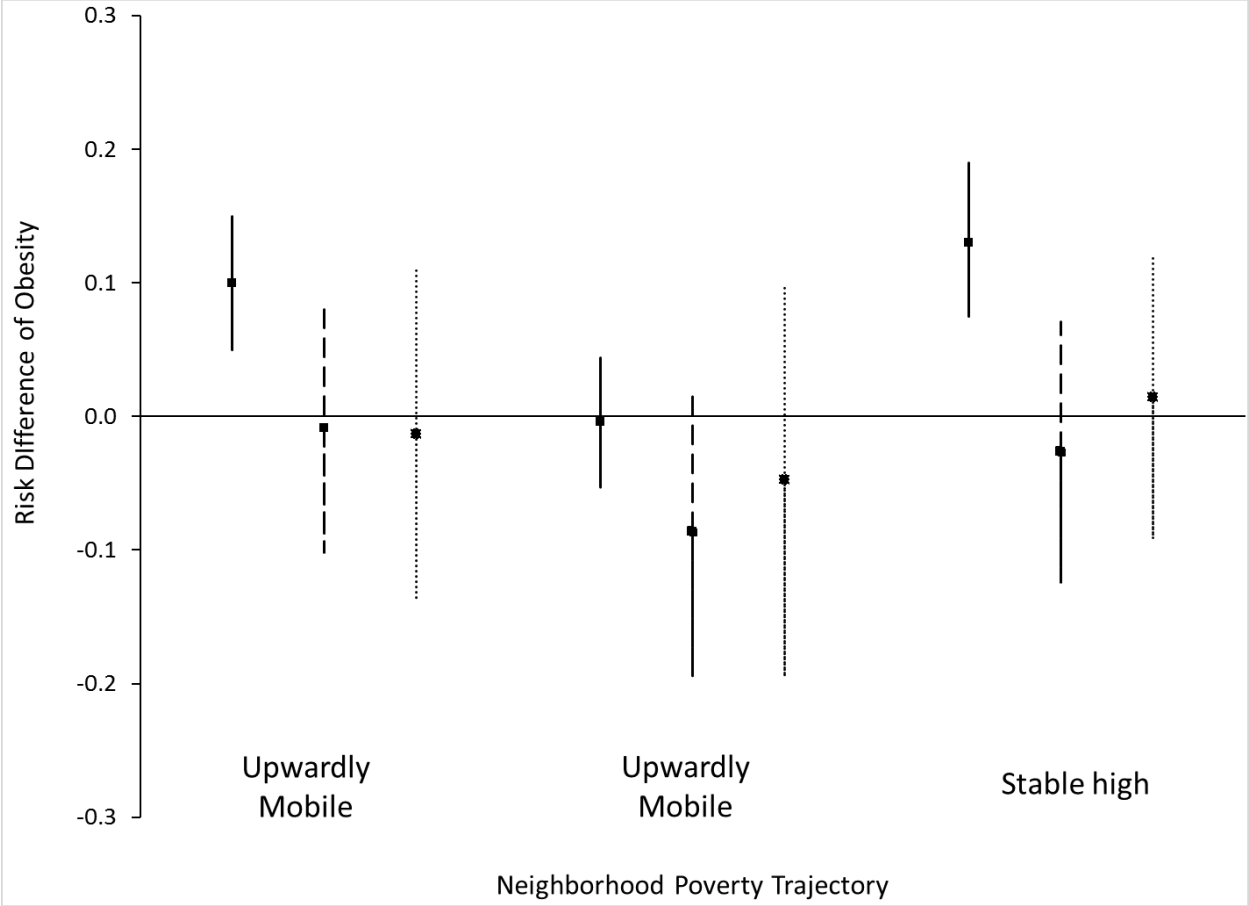

Figure 13. Adjusted risk differences of obesity, and 95% confidence intervals, for neighborhood poverty trajectory group compared to stable low trajectory. Models adjusted for age, sex, nativity, and baseline socioeconomic position (parent's education, marital status, and receipt of public assistance income). Solid line: non-Hispanic whites; dashed line: non-Hispanic blacks; dotted line: Hispanics.

Table 10. Adjusted[a] risk differences of obesity (and 95% confidence intervals) associated with exposure to high neighborhood poverty by sex in the National Longitudinal Study of Adolescent to Adult Health Cohort (N=9,843).

	Measure of Neighborhood Poverty	Male		Female	
Non-Hispanic whites	**Cumulative neighborhood poverty score**				
	0	Referent	--	Referent	--
	1	-0.01	-0.06, 0.05	0.01	-0.04, 0.06
	≥2	0.07	0.00, 0.14	0.11	0.02, 0.20
	Neighborhood poverty trajectory				
	Stable low	Referent	--	Referent	--
	Upwardly mobile	0.14	0.05, 0.23	0.07	0.00, 0.15
	Downwardly mobile	-0.04	-0.10, 0.03	0.03	-0.05, 0.11
	Stable high	0.10	0.02, 0.18	0.16	0.07, 0.26
Non-Hispanic whites	**Cumulative neighborhood poverty score**				
	0			Referent	--
	1			0.05	-0.08, 0.18
	≥2			0.06	-0.08, 0.20
	Neighborhood poverty trajectory				
	Stable low			Referent	--
	Upwardly mobile			0.06	-0.08, 0.19
	Downwardly mobile			-0.06	-0.19, 0.07
	Stable high			0.03	-0.11, 0.16
Hispanics	**Cumulative neighborhood poverty score**				
	0	Referent	--	Referent	--
	1	0.00	-0.15, 0.16	0.04	-0.11, 0.20
	≥2	0.04	-0.09, 0.18	-0.10	-0.23, 0.04
	Neighborhood poverty trajectory				
	Stable low	Referent	--	Referent	--
	Upwardly mobile	0.02	-0.18, 0.21	-0.04	-0.22, 0.13
	Downwardly mobile	-0.18	-0.39, 0.03	0.05	-0.12, 0.23
	Stable high	0.07	-0.05, 0.19	-0.06	-0.23, 0.11

Abbreviations: Adj, adjusted; CI, confidence interval; RD, risk difference.
[a]Models adjusted for age, sex, nativity, and baseline socioeconomic position (parent's education, marital status, and receipt of public assistance income).

Table 11. Prevalence (95% confidence interval) of exposure to high neighborhood poverty among movers (N=6,752) and non-movers (N=3,088) in the National Longitudinal Study of Adolescent to Adult Health Cohort.*

Measure of Neighborhood Poverty	Movers (N=6,752)	Non-movers (N=3,088)
Lived in high poverty neighborhood in 1994-1995	20.6 (15.5, 25.7)	32.6 (25.9, 39.4)
Lived in high poverty neighborhood in 2008-2009	20.2 (17.9, 22.5)	34.4 (28.1, 40.7)
Cumulative neighborhood poverty score		
0	56.5 (51.9, 61.1)	52.8 (45.4, 60.2)
1	27.8 (25.4, 30.2)	16.9 (13.4, 20.4)
≥2	15.7 (12.4, 19.1)	30.3 (23.7, 37.0)
Neighborhood poverty trajectory group		
Stable low	67.0 (62.2, 71.8)	56.1 (49.0, 63.2)
Upwardly mobile	12.8 (9.7, 15.9)	9.4 (6.5, 12.3)
Downwardly mobile	12.4 (10.9, 13.9)	11.2 (8.7, 13.7)
Stable high	7.8 (5.5, 10.2)	23.2 (17.3, 29.1)

*Neighborhood mobility status defined as a move >5 from the residential location in 1994-1995.

Table 12. Risk differences of obesity associated with neighborhood poverty among movers (N=6,752) and non-movers (N=3,088) in the National Longitudinal Study of Adolescent to Adult Health Cohort.

Measure of neighborhood poverty	Movers (N=6,752)	Non-movers (N=3,088)
Cumulative neighborhood poverty score		
0	Referent	Referent
1	0.01 (-0.02, 0.04)	0.04 (-0.03, 0.12)
≥2	0.08 (0.04, 0.24)	0.05 (-0.01, 0.11)
Neighborhood poverty trajectory		
Stable low	Referent	Referent
Upwardly mobile	0.11 (0.07, 0.16)	0.06 (-0.04, 0.16)
Downwardly mobile	0.02 (-0.03, 0.06)	0.00 (-0.07, 0.08)
Stable high	0.14 (0.10, 0.19)	0.04 (-0.02, 0.11)

Abbreviations: CI, confidence interval.

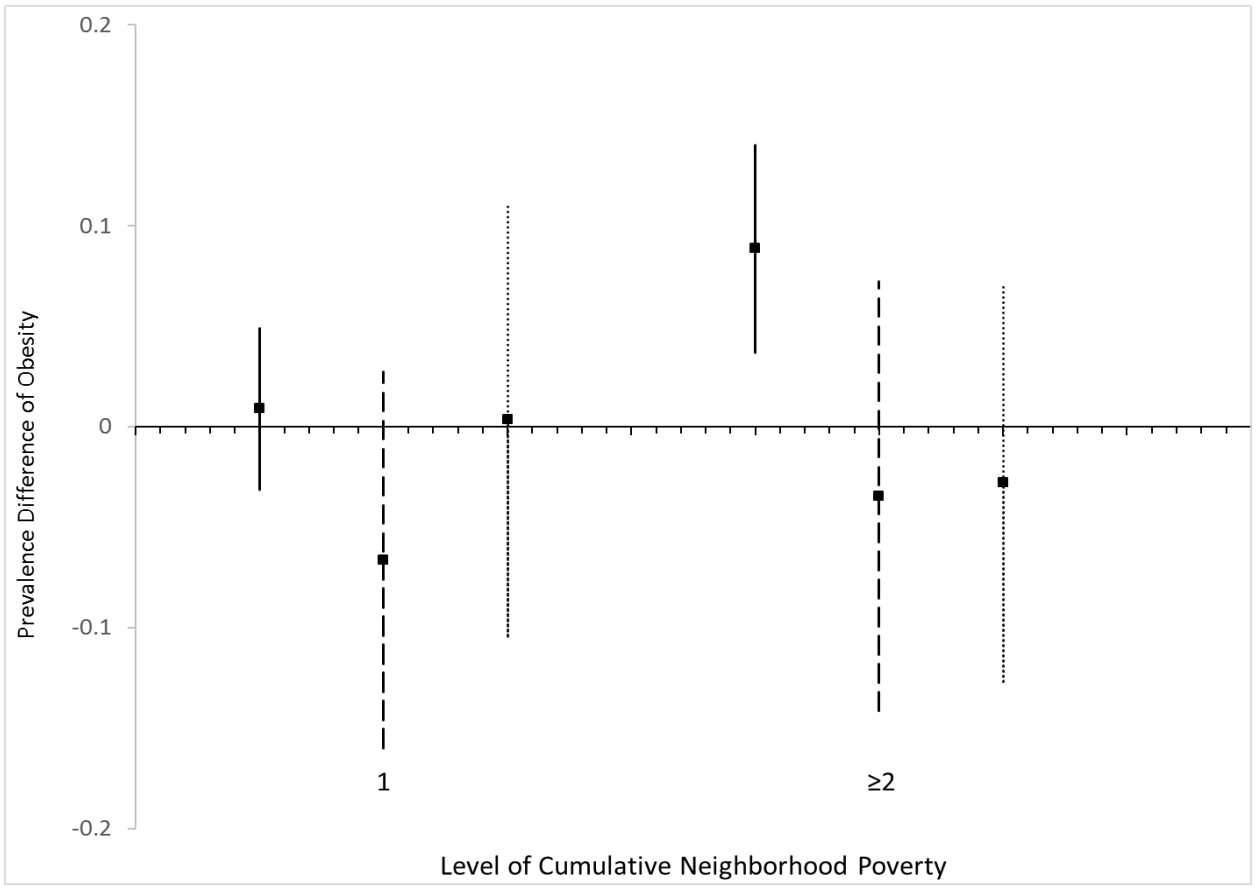

Figure 14. Adjusted prevalence differences of obesity, and 95% confidence intervals, for level of cumulative neighborhood poverty compared to no exposure. Models adjusted for age, sex, nativity, and baseline socioeconomic position (parent's education, marital status, and receipt of public assistance income). Solid line: non-Hispanic whites; dashed line: non-Hispanic blacks; dotted line: Hispanics.

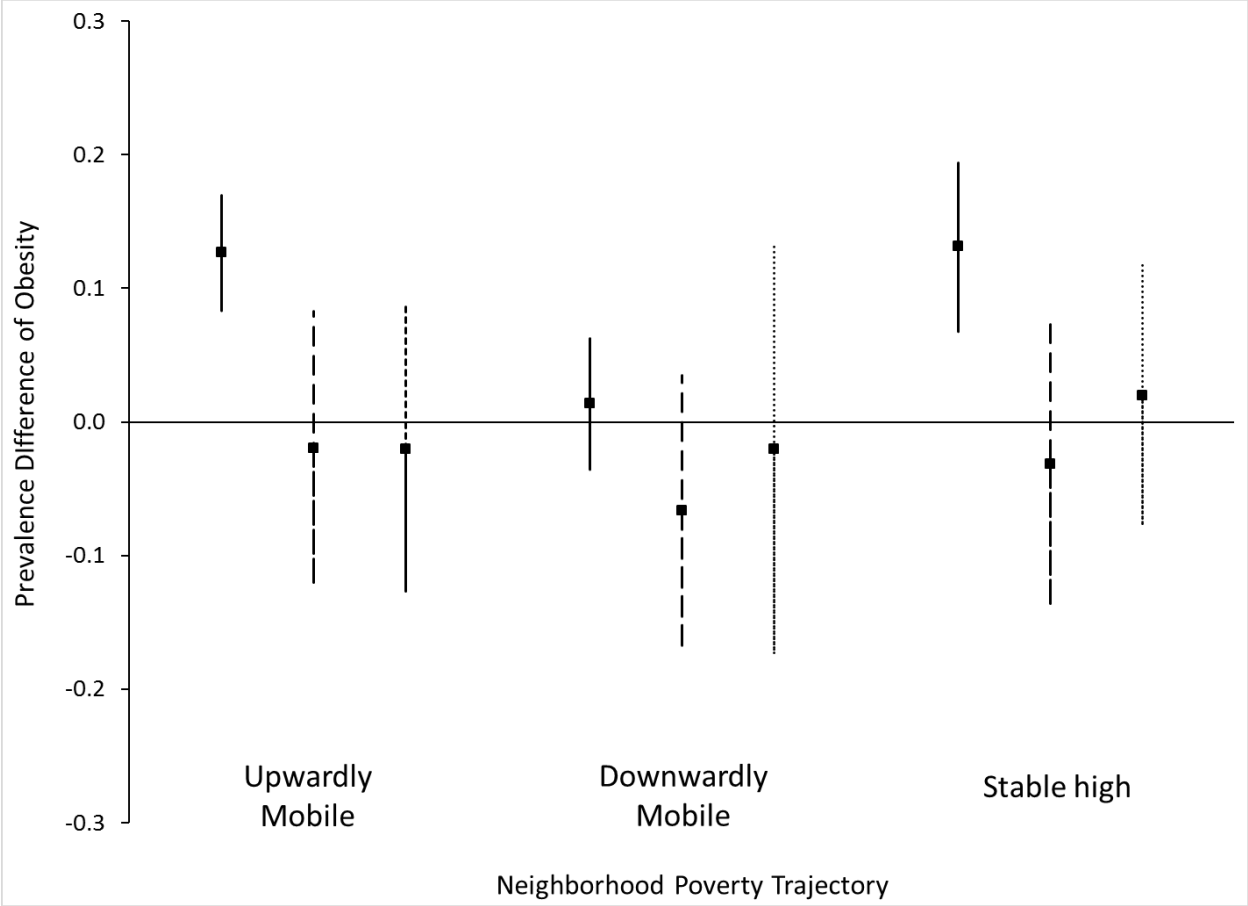

Figure 15. Adjusted prevalence differences of obesity, and 95% confidence intervals, for neighborhood poverty trajectory group compared to stable low trajectory. Models adjusted for age, sex, nativity, and baseline socioeconomic position (parent's education, marital status, and receipt of public assistance income). Solid line: non-Hispanic whites; dashed line: non-Hispanic blacks; dotted line: Hispanic.

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CHAPTER 6: AIM 3

6.1 Introduction

Adverse early life experiences can have enduring consequences for health including effects that manifest later in life. For example, early life residence in poor neighborhoods has been associated with high body mass index (BMI) in adulthood [Lee et al., 2009; Burdette & Needham, 2012]. However, previous studies of the relationship between childhood neighborhood disadvantage and adult weight status have not taken into account the potential mediating effects of the adult neighborhood environment. Therefore, it remains unclear whether this link is due to a direct effect—consistent with a sensitive or critical periods model of the life course [Berkman, 2009]—or whether the effects of early life neighborhood poverty on adult obesity operates solely through adult neighborhood attainment.

Weight-related behaviors begin developing early in childhood and become more established over time [Dietz, 1994; Birch & Doub, 2014]. During adolescence, the establishment of diet and physical activity preferences intersects with greater independence, in general, and more autonomy over these behaviors [Dietz, 1994; Harris & Bargh, 2009; Wells et al., 2010]. Not surprisingly, diet and physical (in)activity patterns in adolescence have been shown to track into adulthood [Gordon-Larsen et al., 2004; Nelson et al., 2005; Biddle et al., 2010]. Since poor neighborhoods often have greater access to unhealthy foods and reduced access to safe recreational spaces [Boone-Heinonen et al., 2013], unhealthy eating and physical activity habits may become ingrained in individuals whose adolescent

development occurs under these conditions. Therefore, adolescence may be a vulnerable period for the obesogenic effects of neighborhood poverty resulting in an increased risk of obesity later in life regardless of adult neighborhood conditions.

Alternatively, the effects of adolescent neighborhood poverty on the risk of obesity later in life may operate through neighborhood poverty status in adulthood. Prior research has shown that the accumulation of neighborhood poverty experiences, as well as neighborhood trajectories characterized by persistent or increasing poverty over the life course, is associated with incident obesity [Murray et al., 2010; Miles et al., unpublished]. Additionally, findings from an experimental study suggests that poor children living in impoverished neighborhoods after age 13 years tend to remain poor, and live in high poverty neighborhoods in adulthood [Chetty et al., 2015]. Therefore, the adolescent neighborhood environment may be indirectly linked to adult health due to the relative lack of social mobility from adolescence to adulthood.

The goal of this study was to determine whether residing in a poor neighborhood during adolescence increases the risk of becoming obese in adulthood independent of adult neighborhood socioeconomic status (SES). To this end, we estimated the direct effect of adolescent neighborhood poverty on obesity using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Understanding the role of timing in the neighborhood poverty-obesity relationship can help inform prevention efforts by identifying the developmental period(s) where this chain of risk is amenable to intervention. If neighborhood conditions in both adolescence and adulthood influence adult obesity, targeting intervention efforts across the life course would be warranted. However, if the obesogenic

effects of neighborhood poverty are determined prior to adulthood, obesity prevention efforts should be focused on youth populations.

6.2 Methods

6.2.1 Study sample

Add Health is an ongoing study of American youth enrolled in the 7 through 12th grade during the 1994-1995 academic year. The study recruitment and data collection methods of Add Health have been previously described [Harris, 2010; Harris, 2011; Resnick et al., 1997]. In brief, a stratified random sample of schools was selected from an educational database to be representative of high schools in the US. Then, for each high school, one "feeder" school expected to contribute students to the high school in the following academic year was selected (proportional to the student contribution to the high school) for participation in the study resulting in a pair of schools in 80 communities. A core sample of students was randomly selected from the school rosters of the participating schools to be representative of their respective schools on the basis of grade level and sex with oversampling from selected demographic groups. This core sample of students (N=20,745) as well as a resident parent or guardian (93% of whom were the mother or stepmother of the core sample respondent) completed in-home interviews at baseline (i.e. Wave I). Student respondents were re-interviewed approximately 1 year (i.e. Wave II, conducted in 1996), 7 years (i.e. Wave III, conducted in 2001-2002), and 14 years (i.e. Wave IV, conducted in 2008-2009) later.

This analysis draws on data collected at the Wave I (1994-1995) and Wave IV (2008-2009) interviews. Respondents without valid measures of both neighborhood poverty and weight status (e.g. females pregnant at the time of interview) collected in adolescence (12-18 years old) and adulthood (>25 years old) were not eligible for the study. Also excluded were

respondents with an age- and sex-specific BMI above the 95th percentile at the start of the study (Wave I, 1994-1995), which left 8,293 respondents for the analysis.

6.2.2 Measures

Obesity

Trained interviewers collected measures of weight and height at the Wave IV (2008-2009) follow-up visit. BMI was calculated as weight (in kilograms) divided by height (in meters2) and obesity was defined as a BMI ≥30 kg/m2 [WHO Expert Committee on Physical Status, 1995].

Life course neighborhood poverty

Census tract poverty was available in Add Health through prior linkage of geocoded residential addresses to geographic data from administrative databases. Respondents' census tract of residence at Wave I (adolescence) and Wave IV (adulthood) were linked to data from the 1990 US Census Survey and the 2005-2009 American Community Survey, respectively. We classified respondents' level of adolescent and adult neighborhood poverty as either high (≥20% residents living below the federal poverty level) or low (<20% residents below the federal poverty level) based on prior research demonstrating that census tract poverty above a 20% threshold is associated with a greater risk for poor health [Do & Finch, 2008] and mortality [Do, Wang, & Elliott, 2013].

Covariates

Characteristics of the respondent that could influence their neighborhood poverty status and obesity status were assessed. Age (continuous) was calculated based on the respondent's self-reported date of birth. Sex and race/ethnicity were also reported by respondents during the baseline (Wave I, 1994-1995) interview. Sex was categorized as male or female, and race/ethnicity as white non-Hispanic, black non-Hispanic, Hispanic, or other

race non-Hispanic. Total number of live births was assessed for female respondents at the final interview (Wave IV, 2008-2009), and all males were assigned a value of 0 for this variable in the analysis.

Indicators of respondents' individual-level SES in adolescence and adulthood were also assessed. The following parental characteristics served as indicators of respondents' adolescent SES: parent's education, marital status, and receipt of public assistance. Parents reported their marital status (married vs. unmarried), highest level of schooling completed, the highest level of schooling completed by their resident spouse or partner (if applicable), and whether they received any public assistance income during the 1994 calendar year (yes vs. no). The educational attainment of the resident parent or guardian with the highest level of schooling was collapsed into 5 categories (less than high school, high school diploma or equivalent, some college or trade/technical school, associate's or bachelor's degree, and advanced degree or training). Respondents' own marital status (ever been married vs. never married), educational attainment, and household income at the time of final interview (Wave IV, 2008-2009) served as indicators of their adult SES.

6.2.3 Statistical analysis

The weighted distribution of cohort members' demographic, household, and parental characteristics, and obesity status were calculated. Guided by the DAG shown in Figure 11, we constructed binomial regression models to estimate on the risk difference scale, the controlled direct effect (controlled direct effect) of high adolescent neighborhood poverty (exposure) on obesity in adulthood through pathways not mediated by adult neighborhood poverty status (mediator). For comparison, we also constructed models to estimate the total effect (total effect) of high adolescent neighborhood poverty on adult obesity and the total effect of high adult neighborhood poverty on adult obesity.

Whereas the total effect of high adolescent neighborhood poverty represents the average effect interventions preventing this exposure would have on the risk of obesity in adulthood, the controlled direct effect tells us the effect of interventions that would set adult neighborhood poverty to a certain level (i.e. low or high) for all people, regardless of their adolescent neighborhood poverty experience [VanderWeele, 2015]. In the absence of evidence suggesting interaction between adolescent and adult neighborhood poverty on the risk of obesity at the 0.2-significance level, we report only controlled direct effect estimates from models with no product interaction term between these variables and where adult neighborhood poverty is set to low for everyone in the sample—since this effect is of greatest public health interest.

Stabilized inverse probability weights were calculated to adjust for confounding of the total and direct effects being estimated. The technical details for constructing stabilized inverse probability weighted marginal structural models for effect estimation have been provided elsewhere [Nandi et al., 2010]. In brief, this method creates a pseudo-population with a balanced distribution of measured confounders (and, in the case of the controlled direct effect, the mediator) across levels of the exposure of interest. For total effect estimation, inverse probability weights were calculated as the marginal probability of exposure divided by the conditional probability of exposure given observed confounders. Final weights were calculated as the product of the stabilized inverse probability weight and the survey sampling weight [Brumback et al., 2010]. For controlled direct effect estimation, we calculated an additional set of stabilized inverse probability weights for the mediator (adult neighborhood poverty). Mediator weights were calculated as the marginal probability of exposure to high neighborhood poverty in adulthood divided by the conditional probability

of the same given the observed values of adolescent neighborhood poverty and measured confounders. The final weight was calculated as the product of the exposure, mediator, and sampling weights [Brumback et al., 2010].

Prior research has demonstrated that exposure to disadvantaged neighborhoods varies considerably across racial/ethnic groups in the US—even at similar levels of individual SES [Messer et al., 2010]. Furthermore, the health effects of area-level poverty may differ across sex and racial/ethnic boundaries [Carson et al., 2007; Lemelin et al., 2009; Murray et al., 2010]. Therefore, despite the lack of empirical evidence for effect measure modification by sex ($t=1.07$; $p=0.3$) or race/ethnicity ($t=-0.8$; $p=0.4$) in this study, we report stratum-specific effect estimates for the three largest racial/ethnic groups (non-Hispanic whites, non-Hispanic blacks, and Hispanics) and for males and females.

All analyses were conducted using STATA 14 (StataCorp LP, College Station, TX). Standard errors were calculated using Taylor linearization to account for the complex survey design and weights [StataCorp, 2015].

6.3 Results

The weighted distributions of selected characteristics of the analytic sample are shown in Table 13. The mean age of the sample at baseline was 15.3 years. Females made up half of the sample, and the majority race/ethnicity was non-Hispanic white (69.9%). Most respondents had a parent who reported being married at the time of baseline interview (78.0%) and not having received public assistance income in calendar year 1994 (91.4%). At the time of final interview (Wave IV, 2008-2009), 47.0% of respondents were currently married or had ever been married, 34.2% had obtained a college degree or higher, and more than half (56.3%) had an annual household income of at least $50,000.

The prevalence of high neighborhood poverty remained stable over time: 24.3% of respondents lived in high poverty neighborhoods as adolescents and 24.4% lived in high poverty neighborhoods as adults. Among individuals exposed to high poverty neighborhoods as adolescents, 51.8% lived in high poverty neighborhoods in adulthood. In contrast, only 15.9% of respondents residing in low poverty neighborhoods during adolescence resided in high poverty neighborhoods in adulthood. There were no sex differences in the prevalence of exposure to high adolescent neighborhood poverty (males, 23.6%; females, 25.0%) or high adult neighborhood poverty (males, 24.0%; females, 24.8%). However, there were dramatic racial/ethnic differences in both life stages. The proportion of non-Hispanic whites residing in high poverty neighborhoods increased slightly from 15.3% in adolescence to 18.7% in adulthood. Non-Hispanic blacks and Hispanics showed declines in high poverty neighborhood experiences over the life course from 61.4% to 52.4% and from 38.4% to 28.5%, respectively.

The incidence of obesity between adolescence and adulthood was 30.2%. Marginal structural model estimates of the total effect of adolescent and adult neighborhood poverty on the risk of obesity in adulthood are summarized in Table 14. The risk of obesity among individuals exposed to high neighborhood poverty during adolescence was 11.5 percentage points greater (95% confidence interval [CI]=8.10, 14.86) than the risk among individuals exposed to low adolescent neighborhood poverty. The total effect of high adolescent neighborhood poverty on the risk of adult obesity among females (risk difference [RD]: 16.0; 95% CI: 10.54, 21.51) was double that of males (RD: 7.8; 95% CI: 2.47, 13.12). Of the three racial/ethnic groups, the total effect of high adolescent neighborhood poverty on obesity was greatest among non-Hispanic whites (RD: 12.3; 95% CI: 7.65, 16.96). With the exception of

females, for whom the risk of obesity in adulthood was 12.4 percentage points greater (95% CI=4.49, 20.26) among individuals exposed to high versus low neighborhood poverty during adolescence, the effect of adult neighborhood poverty on obesity was non-significant in the overall sample and within subgroups.

High adolescent neighborhood poverty was associated with a 12.2 percentage point (95% CI: 7.96, 16.41) increase in the risk of adult obesity through pathways not mediated by adult neighborhood poverty (Table 15). When adult neighborhood was set to low, the estimated controlled direct effect of high adolescent neighborhood poverty on adult obesity was similar in magnitude to the estimated total effect for males (RD=11.8; 95% CI: 6.15, 17.44), females (RD=12.4; 95% CI: 5.72, 19.06), and non-Hispanic whites (RD=12.5; 95% CI: 7.07, 18.00). Differences in controlled direct effect estimates when setting adult neighborhood poverty to high versus low were non-significant (Table 16, Figures 16-19).

6.4 Discussion

In this study, we found evidence of a direct effect of adolescent neighborhood poverty on adult obesity, independent of adult neighborhood poverty, in a sample of previously non-obese youth in the US. Because childhood and adult SES are strongly related, and conventional regression methods to direct effect estimation may be more susceptible to bias due to unmeasured confounding [VanderWeele, 2015], we used marginal structural modeling to account for the potential mediating effects of adult neighborhood poverty and time-varying confounding by SES. This approach balances the distribution of the mediator of interest and confounders of both the exposure-outcome and mediator-outcome relationship across levels of the exposure using stabilized inverse probability weights.

Estimates of both the total and direct effects of high adolescent neighborhood poverty on obesity were generally larger than the effect of high adult neighborhood poverty overall.

This be might suggestive of a greater importance of early life neighborhood conditions on the development of obesity than adult conditions. These findings are consistent with prevailing theories on critical periods in the life course for obesity development [Dietz, 1994; Birch & Doub, 2014] and health-related behavior development during adolescence [Kuh & Ben-Shlomo, 2004].

Distinct patterns in the effects of adolescent neighborhood poverty on adult obesity risk emerged within groups. The TEs of high neighborhood poverty in adolescence and adulthood on obesity were larger for males than females, whereas the estimated direct effect of high adolescent neighborhood poverty was similar across groups. That both adolescence and adulthood seem to be periods of susceptibility for the obesogenic effects of neighborhood poverty among females suggests an accumulation model of the life course [Berkman, 2009]. In contrast, results for males and non-Hispanic whites were more consistent with a critical periods model: the risk of obesity was greater among people with high adolescent neighborhood poverty exposure whereas high adult neighborhood poverty did not increase the risk of obesity.

There are several limitations to our study that should be considered when interpreting these findings. First, while marginal structural model estimates produced using inverse probability weights allow for time-varying confounders, they are still susceptible to bias due to residual confounding by unmeasured time-invariant and time-varying factors that influence one's neighborhood conditions and adult weight status. This common limitation may be especially salient to our study given the numerous challenges of attempting to control for the influence people have on their neighborhood environment in order to isolate and measure "neighborhood effects" [Oakes, 2014; Oakes et al., 2015]. We explored the potential

for unmeasured confounding by other early life risk factors for obesity not included in our main analysis: intrauterine growth restriction (birth weight, low vs. normal, used as a proxy) and parent's obesity status (i.e. whether either biological parent was obese at the time of baseline interview). These factors have been associated with adult health including weight status and can plausibly influence parents' choices or constraints regarding neighborhood selection. The TE was attenuated and the CDE relatively unchanged when accounting for these additional variables (data not shown). Second, measured risk factors may be measured with error (e.g. parent's marital status, receipt of public assistance, and educational attainment may not fully capture aspects of one's childhood SES) and, thus, our estimates may be subject to residual confounding. Third, to be included in our sample, respondents had to participate in the Wave IV interview (conducted in 2008-2009) and could not be obese at baseline. The exclusion of these individuals from the study may underestimate the true effect of adolescent neighborhood poverty on obesity since Add Health cohort members who were lost to follow-up were more likely to 1) belong to a low SES group; 2) reside in a high poverty neighborhood; and 3) be obese at the start of the study.

Nevertheless, our findings are buttressed by an established literature demonstrating a link between individual socioeconomic position in early life and adult weight status [Hardy et al., 2000; Power et al., 2005; Ball & Mishra, 2006; Bennett et al., 2007; Giskes et al, 2008; Lee et al., 2009], and a growing literature that suggests the early life neighborhood SES environment may influence adult weight status as well [Lee et al., 2009]. If confirmed in future studies, these findings suggest that adolescence may be a critical time for tailored obesity prevention efforts, especially for youth residing in poor neighborhoods. Future research should seek to identify specific mechanisms at the neighborhood level, and in

related contexts such as the school and family, linking neighborhood poverty to obesity during this adult life stage.

Table 13. Demographic characteristics of sample, National Longitudinal Study of Adolescent to Adult Health Cohort, 1994-2009 (N=8,293).

Characteristic	N	%
Age, years (mean)	8,293	15.3
Sex		
Female	4,479	49.5
Male	3,814	50.5
Race/ethnicity		
Non-Hispanic white	4,790	69.9
Non-Hispanic black	1,595	13.5
Hispanic	1,219	11.3
Non-Hispanic Asian	547	3.6
Non-Hispanic other race	139	1.6
US-born		
Yes	7,707	95.0
No	586	5.0
Parent's marital status in 1994-1995		
Married	5,754	78.0
Unmarried	1,636	22.0
Parent's educational attainment in 1994-1995		
Less than high school	1,119	15.0
High school diploma or equivalent	2,111	31.2
Some college or technical school	2,170	28.9
College degree (Associate's or Bachelor's)	1,168	14.9
Advanced degree or training	790	9.9
Parent received public assistance income in 1994	6,788	91.4
No	578	8.6
Yes		
Marital status in 2008-2009		
Ever married	3,947	47.0
Never married	4,336	53.0
Educational attainment in 2008-2009		
Less than college degree	5,285	65.8
College degree or higher	3,006	34.2
Household income in 2008-2009		
<$10,000	322	4.2
$10,000-$14,999	237	3.3
$15,000-$19,999	248	3.2
$20,000-$29,999	735	10.0
$30,000-$39,999	833	11.1
$40,000-$49,999	903	11.9
$50,000-$74,999	1,959	25.0
$75,000+	2,567	31.3

Table 14. Risk difference estimates of the total effects of high adolescent neighborhood and high adult neighborhood poverty on adult obesity incidence.

	Risk Difference of Obesity per 100 (95% CI)	
	Adolescence	**Adult**
All	11.5 (8.10, 14.86)	2.9 (-1.10, 6.91)
Males	7.8 (2.47, 13.12)	-2.5 (-7.96, 3.00)
Females	16.0 (10.54, 21.51)	12.4 (4.49, 20.26)
Non-Hispanic whites	12.3 (7.65, 16.96)	1.7 (-3.93, 7.27)
Non-Hispanic blacks	4.7 (-7.84, 17.16)	-1.0 (-12.08, 9.98)
Hispanics	-2.1 (-12.89, 8.77)	-6.5 (-27.83, 14.76)

Table 15. Risk difference estimates of the controlled direct effect of high adolescent neighborhood poverty on adult obesity incidence.

	Risk Difference of Obesity per 100 (95% CI)
All	12.2 (7.96, 16.41)
Males	11.8 (6.15, 17.44)
Females	12.4 (5.72, 19.06)
Non-Hispanic whites	12.5 (7.07, 18.00)
Non-Hispanic blacks	8.5 (-3.32, 20.34)
Hispanics	-9.5 (-29.56, 10.48)

Table 16. Estimated controlled direct effect (risk difference, 95% CI) of adolescent neighborhood poverty on adult obesity when setting adult neighborhood poverty to low versus high.

	Risk Difference of Obesity per 100 (95% CI)	
	when adult neighborhood poverty set to low	**when adult neighborhood poverty set to high**
All	10.9 (4.96, 16.92)	14.6 (7.69, 21.56)
Males	9.8 (1.92, 17.67)	15.0 (6.31, 23.74)
Females	11.7 (4.19, 19.17)	14.4 (-0.53, 29.43)
Non-Hispanic whites	10.9 (3.89, 17.89)	16.3 (6.48, 26.08)
Non-Hispanic blacks	8.6 (-7.15, 24.36)	8.4 (-8.49, 25.28)
Hispanics	-12.1 (-35.92, 11.78)	7.9 (-11.45, 27.20)

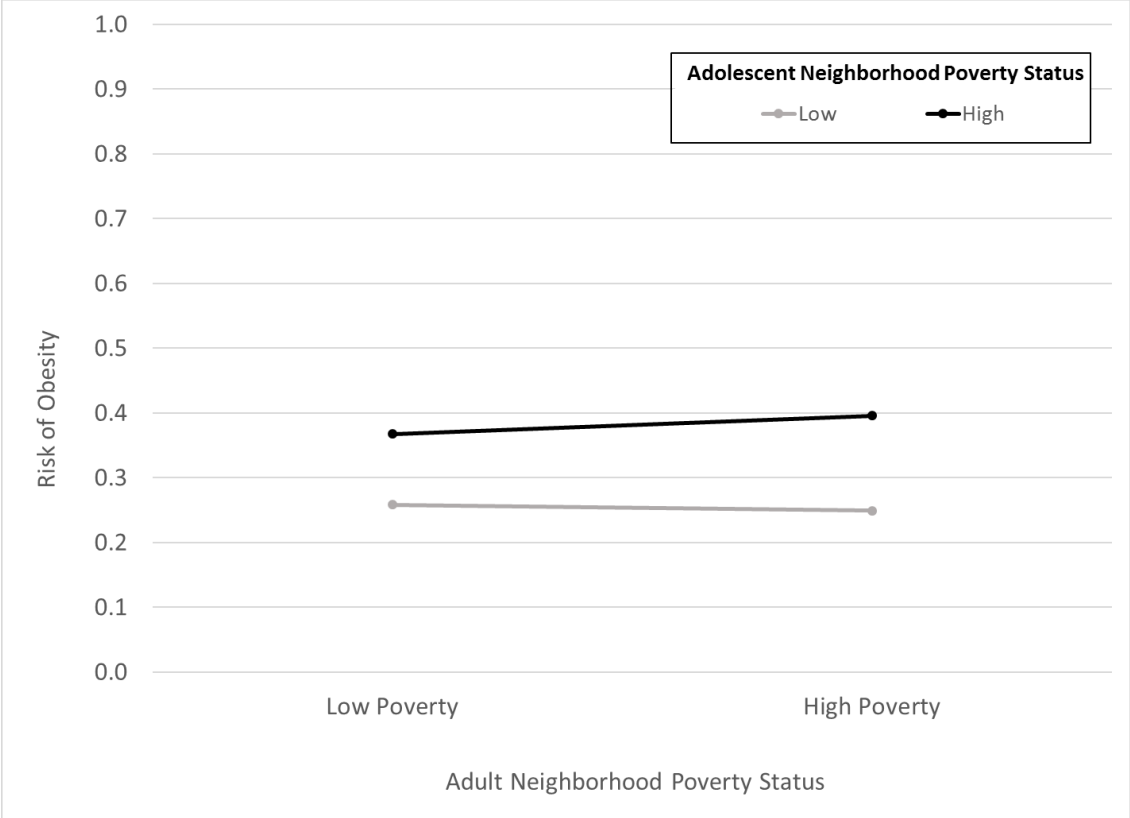
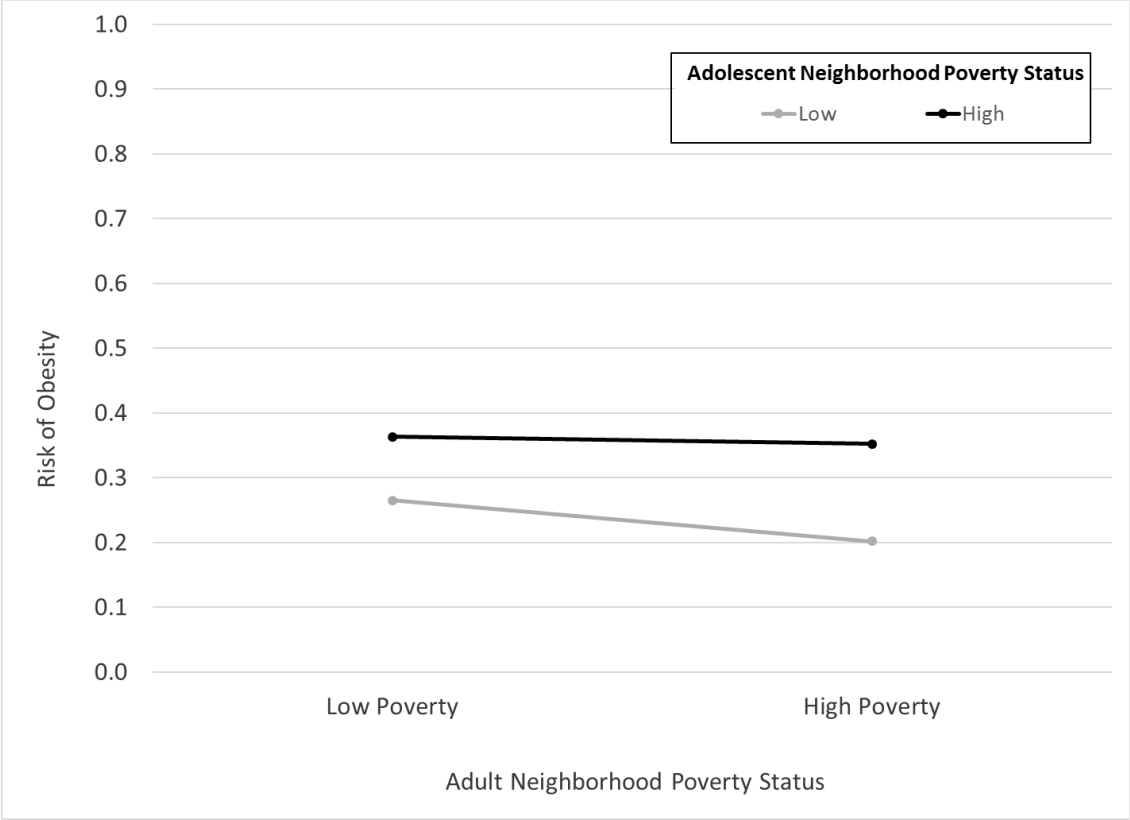


Figure 16. Estimated risk of obesity among individuals exposed to high (≥20% poverty) versus low (<20%) neighborhood poverty during adolescence when adult neighborhood poverty set to low and high for the entire sample (N=8,392).

A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Risk of Obesity
Adolescent Neighborhood Poverty Status
Low
High
Low Poverty
High Poverty
Adult Neighborhood Poverty Status

B

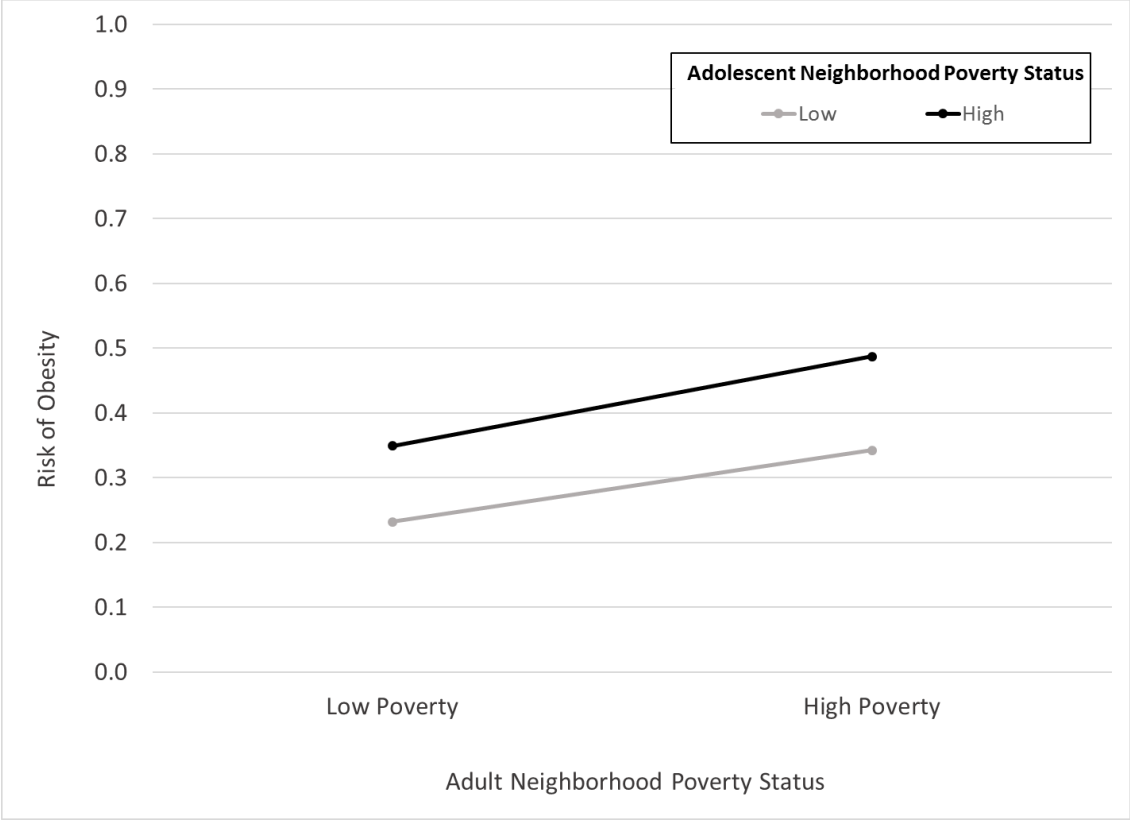
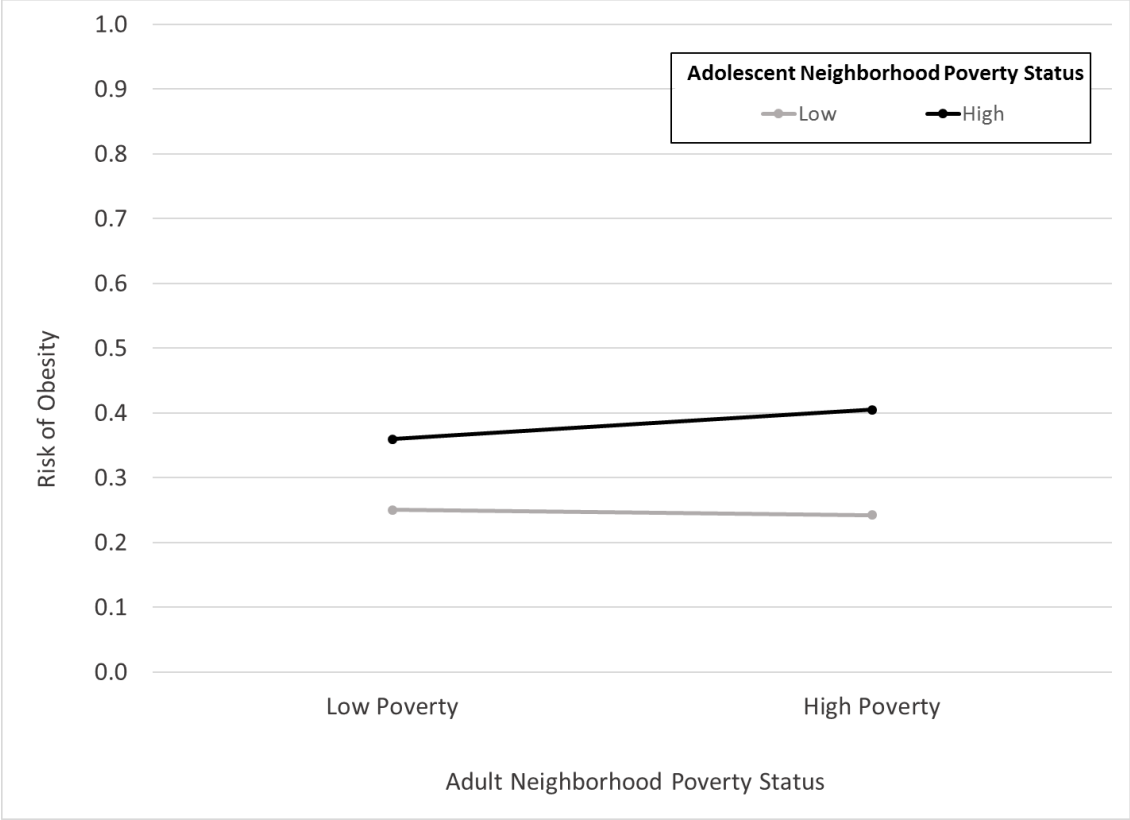


Figure 17. Estimated risk of obesity for A) males and B) females exposed to high (≥20% poverty) versus low (<20% poverty) neighborhood poverty during adolescence when adult neighborhood poverty set to low and high for the entire sample (N=8,392).

A


1.0
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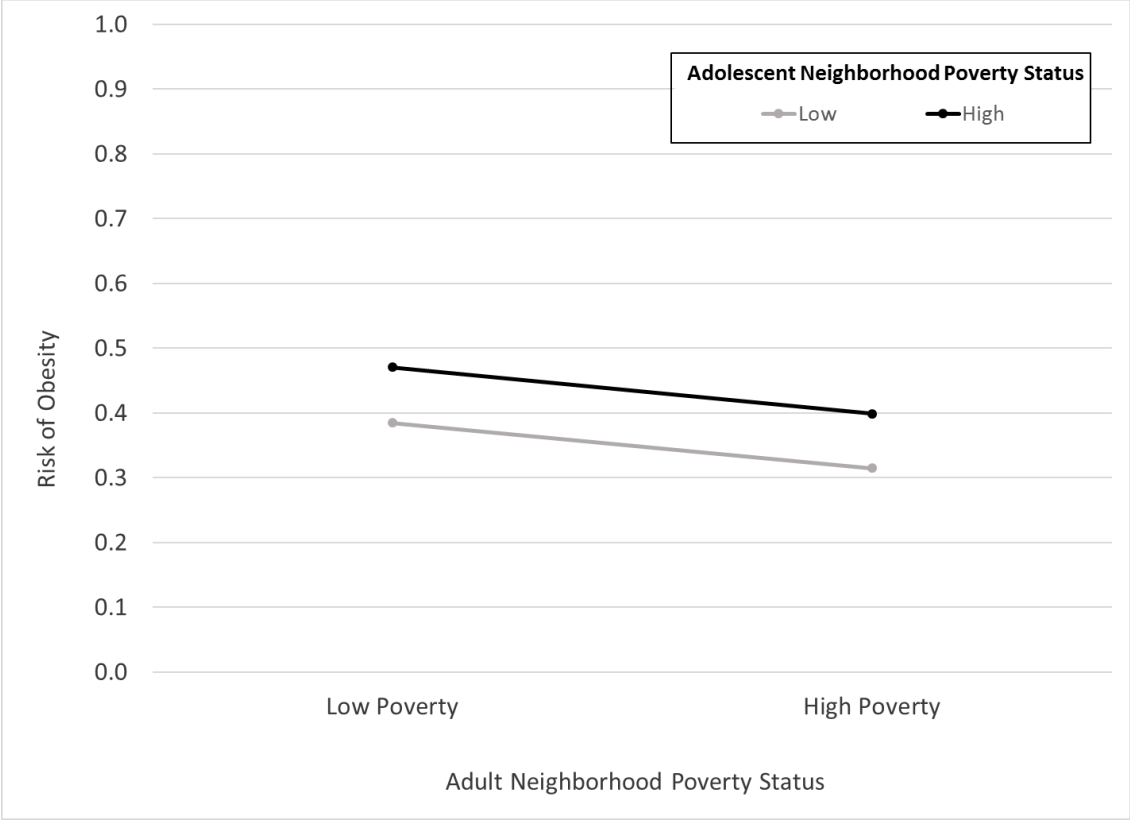
Risk of Obesity
Adolescent Neighborhood Poverty Status
Low
High
Low Poverty
High Poverty
Adult Neighborhood Poverty Status

B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Risk of Obesity
Adolescent Neighborhood Poverty Status
Low
High
Low Poverty
High Poverty
Adult Neighborhood Poverty Status

C

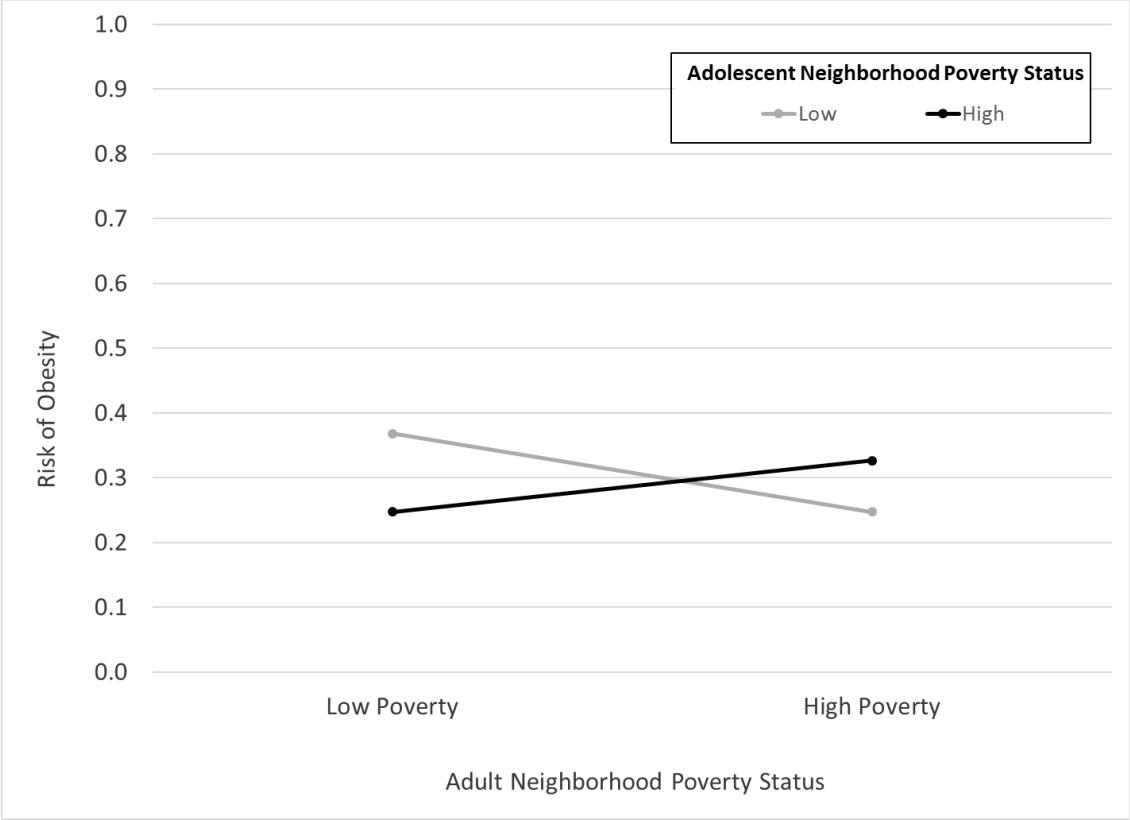
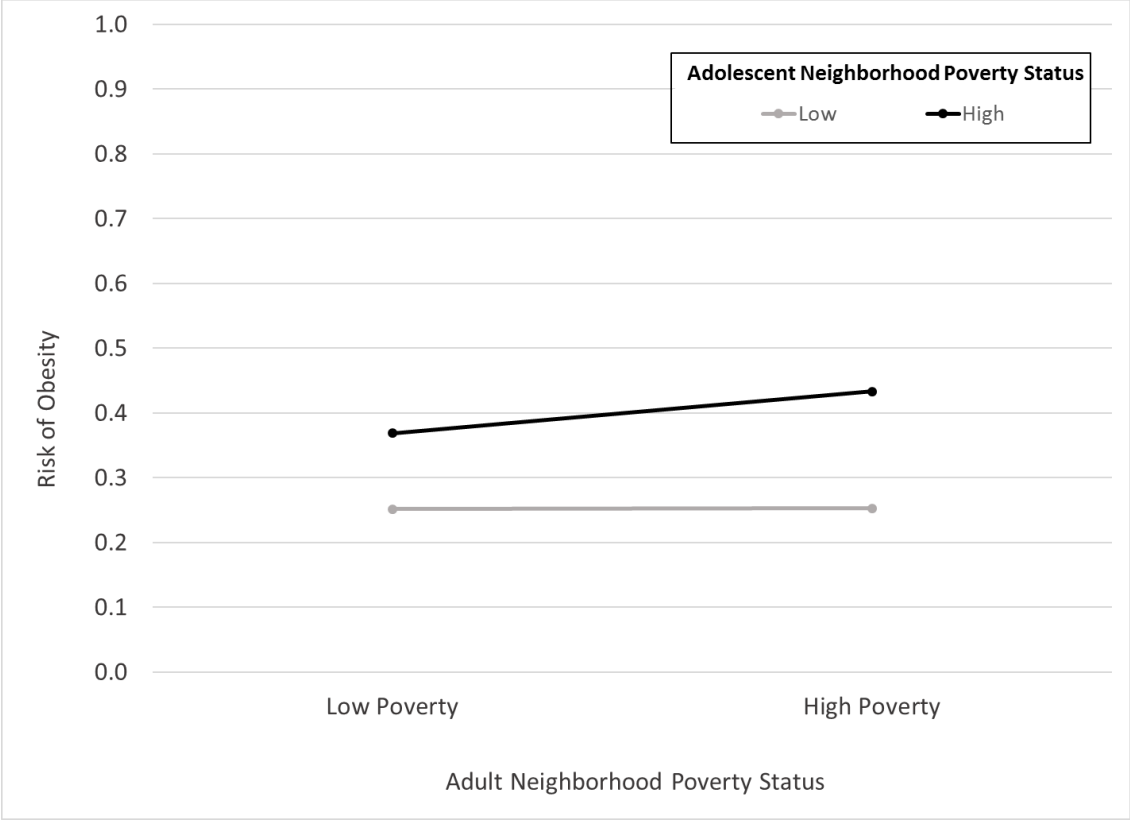


Figure 18. Estimated risk of obesity for A) non-Hispanic whites, B) non-Hispanic blacks, and C) Hispanics exposed to high (≥20% poverty) versus low (<20% poverty) neighborhood poverty during adolescence when adult neighborhood poverty set to low and high for the entire sample (N=8,392).

A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Risk of Obesity
Adolescent Neighborhood Poverty Status
Low
High
Low Poverty
High Poverty
Adult Neighborhood Poverty Status

B

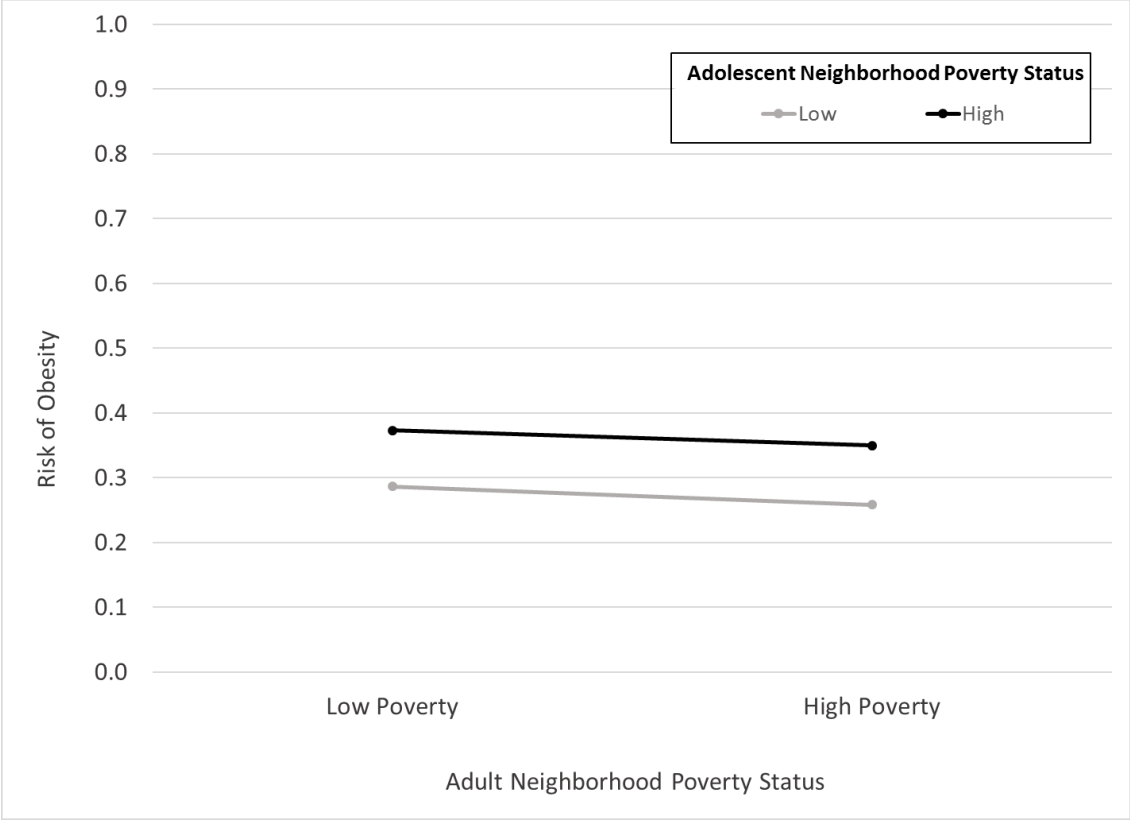

Figure 19. Estimated risk of obesity for A) movers and B) non-movers exposed to high (≥20% poverty) versus low (<20% poverty) neighborhood poverty during adolescence when adult neighborhood poverty set to low and high for the entire sample (N=8,392).

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CHAPTER 7: CONCLUSION

This research project investigated the effects of life course neighborhood poverty on obesity in an American cohort of non-obese, school-attending youth followed from adolescence in the mid-1990s to adulthood in 2008-2009. Specifically, we applied and tested three models of the life course social environment and health: the social trajectories model (Research Aim #1), the accumulation model (Research Aim #2), and the critical periods model (Research Aim #3). These models postulate distinct roles of the early life and adult social environment on adult health [Berkman, 2009]. Briefly, both the social trajectories model and the accumulation model propose the adult social environment has a causal effect on adult health. The distinction between the two models arises from the proposed role of the early life social environment in shaping adult health. The social trajectories model hypothesizes that the early life social environment influences adult health only through its impact on one's adult social environment. The accumulation model, on the other hand, hypothesizes that the early life social environment influences adult health through pathways mediated by the adult social environment and through pathways not mediated by the adult social environment. Finally, the critical periods model postulates that the early life social environment influences adult health through pathways not mediated by the adult social environment, and that the adult social environment does not influence adult health.

In Research Aim #1, we observed considerable variability in neighborhood poverty experiences between adolescence and adulthood: nearly a quarter of respondents did not fall

into one of the stable trajectory groups (i.e. stable low poverty or stable high poverty). Similar to prior research examining neighborhood trajectories from adolescence through the transition to adulthood [Swisher et al., 2013], we observed stark racial/ethnic differences in adolescent-adult neighborhood poverty trajectories. Non-Hispanic blacks and Hispanics were more likely to fall into one of the unstable neighborhood poverty trajectory groups. These findings demonstrate that a single measure would have done a poor job of representing exposure to neighborhood poverty, and that the degree of misclassification would be greater in populations at an increased risk for obesity. This finding stands in contrast to previous research in older adults suggesting cross-sectional measures of neighborhood poverty might represent exposure history reasonably well [Murray et al., 2010]. However, multivariable models indicated that the association between neighborhood poverty trajectory and incident obesity is limited to non-Hispanic whites. In this group, individuals experiencing decreasing neighborhood poverty (i.e. were upwardly mobile) or a stable high neighborhood poverty trajectory had a greater risk of obesity in early adulthood than those who experienced a stable low neighborhood poverty trajectory.

In Research Aim #2, we found evidence of an association between the accumulation of neighborhood poverty experiences and obesity. Specifically, individuals residing in high poverty neighborhoods (i.e. neighborhoods where ≥20% of the residents had a family income below the federal poverty level) at 2 or more study waves were at an increased risk of becoming obese by early adulthood. Again, in subgroup analyses, we only found evidence of an association among non-Hispanic whites.

Finally, in Research Aim #3, we attempted to account for bias due to time-varying SES by employing a marginal structural modeling approach to mediation analysis in order to

investigate whether neighborhood poverty experiences in early life (adolescence) influenced the risk of obesity in adulthood through pathways not mediated by adult neighborhood poverty. Given the increasing autonomy over weight-related behaviors [Dietz, 1994; Harris & Bargh, 2009; Wells et al., 2010] and the tracking of these behaviors into adulthood [Gordon-Larsen et al., 2004; Nelson et al., 2005; Biddle et al., 2010], we hypothesized that adolescence may be a critical period in development for the obesogenic effects of poor neighborhoods. We found evidence in support of this hypothesis, but findings varied across groups. There was a direct effect of high adolescent neighborhood poverty on the risk of obesity among males, females, and non-Hispanic whites through pathways not mediated by adult neighborhood poverty. However, the effect of high adult neighborhood poverty on obesity varied. Consistent with a critical periods model of the life course, there was no estimated effect of adult neighborhood poverty on adult obesity for males and non-Hispanic whites. However, findings for females were more consistent with an accumulation model: in addition to the direct effect of high adolescent neighborhood poverty on obesity, the risk of obesity was also higher for individuals experiencing high neighborhood poverty in adulthood.

This is the first study to report evidence of life course effects of neighborhood poverty from adolescence to adulthood on the risk of obesity. However, our findings are consistent with previous studies reporting life course effects of individual-level poverty on adult weight status [Giskes et al., 2008; Lee et al., 2009; Smith et al., 2016]. Lee, Harris, and Gordon-Larsen (2009) previously reported a longitudinal association between neighborhood poverty in adolescence and staying obese (but not becoming obese) between adolescence and the transition to adulthood (ages 18-25 years old). Also using Add Health data, Burdette and

Needham (2012) found that adolescent neighborhood disadvantage was associated with higher adolescent body mass index and a more rapid increase in BMI from adolescence to adulthood**.** Murray and colleagues (2010) found that trajectories of neighborhood poverty were associated with differing levels of subclinical atherosclerosis risk. In particular, stable trajectories of medium to high neighborhood poverty over a 20-year period were associated with a greater risk of positive change in BMI among females.

This research also responds to calls for a more nuanced understanding of the ways in which neighborhoods affect health across the life course [Sharkey & Faber, 2014]. In general, we estimated greater effects of life course neighborhood poverty on obesity for non-Hispanic whites compared to non-Hispanic blacks and Hispanics and for females compared to males. It has become widely accepted that females are more vulnerable to the obesogenic effects of neighborhoods [Stafford et al., 2005]. There is less consistency in the literature over racial/ethnic differences in the effects of neighborhood disadvantage on obesity. It is clear, however, that racial/ethnic minorities are more likely to be exposed to disadvantaged neighborhoods—a findings confirmed in the present study—as well as associated environmental hazards [Northridge et al., 2003]. Therefore, even small effects of neighborhood poverty on obesity in these populations may have public health significance.

This research does have important caveats that should be considered when interpreting its findings. First, causal interpretations of these findings are based on the assumption of no unmeasured confounding of the exposure-outcome relationships being investigated. For Research Aim #3, this assumption is even stronger because we must also assume that we have accurately measured and controlled for all confounders of the mediator-outcome relationship. Confounding assumptions are particularly challenging to test in

neighborhood effects research since factors associated with neighborhood selection are not well understood [DeLuca et al., unpublished; Gustafsson et al., 2013]. Even proponents of neighborhood effects research recognize the social complexities of neighborhoods and the limitations of traditional epidemiologic methods and causal frameworks for studying their effects on health. Cummins et al. (2007) warns neighborhood researchers to "avoid the false dualism of context and composition by recognizing that there is a mutually reinforcing and reciprocal relationship between people and place." Therefore, it may be impossible to truly isolate and measure "neighborhood effects" in observational settings [Oakes, 2014; Oakes et al., 2015]. This issue is further complicated by the longitudinal nature of our research question. While assessing neighborhood environment and obesity status prospectively from adolescence—when respondents presumably had less influence over housing choices—and limiting our analyses to individuals who were non-obese at baseline limits the possibility of reverse causality [Jokela, 2014], the potential for confounding by parental characteristics that influenced both neighborhood selection in early life and adult obesity status still remains. Nevertheless, to the extent that neighborhood selection represents a complex interplay of constrained choices, especially for low SES groups, neighborhood effects may represent an important contributor to obesity and obesity-related disparities.

Finally, this study is susceptible to bias due to differential loss to follow-up in Add Health. The percentage of females, individuals of higher SES, and those who identified as non-Hispanic white race/ethnicity increased over time in the Add Health sample [Brownstein et al., unpublished]. Given that sex, SES, and race/ethnicity are associated with neighborhood poverty and/or obesity in this sample, we may have inadvertently introduced bias into our effect estimates by selecting individuals who were present at follow-up study waves.

However, Add Health survey weights account for nonresponse stratified by factors such as sex and race. Furthermore, inverse probability weighting—the approach we employ in Research Aim #3—controls for selection bias when the variables used to calculate the conditional probability of exposure adequately capture factors that predict selection [Miguel et al., 2005]. Previous studies suggest, however, that selection bias due to differential attrition in longitudinal studies may be minimal even without controlling for selection factors [Carter et al., 2012].

This study advances the current knowledge of the health effects of neighborhood socioeconomic disadvantage by describing the life course effects of neighborhood poverty on obesity within population subgroups. These findings point to the importance of neighborhood context in the development of obesity and the need for early intervention on the more distal determinants of weight and weight gain. Future research should similarly be grounded in theory and seek to understand the social and neighborhood mechanisms linking life course neighborhood socioeconomic experiences to adult health. As Morenoff and Lynch (2004) stated regarding the apparent disconnect between the epidemiology and social science literatures on neighborhood effects:

> "…whereas the sociological literature on neighborhood effects has taken a 'process turn' in recent years and begun to focus on the mechanisms that explain *why* neighborhoods matter…most research on the neighborhood context of health is still attempting to establish *that* context matters."

Considering the selection and identification challenges that plague research on neighborhood health effects and the unfeasibility of randomized trials, this field would benefit from studies capitalizing on policy interventions and other exogenous neighborhood changes that lend

themselves to quasi-experimental study designs.

In closing, it is time to move beyond cross-sectional studies of neighborhood-obesity associations toward designs that offer stronger evidence for causal inference and can yield information useful for guiding public health programs and policies. Given the burden of obesity and its associated consequences on the American population for the past three decades, and the little progress that has been made toward reversing these trends, it is critically important that epidemiologists generate sound research that can provide evidence-based solutions to the greatest public health problem of our generation.

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