The indelible weight of place: Childhood neighborhood disadvantage, timing of exposure, and obesity across adulthood

Steven Elías Alvarado¹
Cornell University

Abstract:

I use 28 (1986-2014) years of restricted geocoded NLSY data and find positive associations between exposure to childhood neighborhood disadvantage and adult obesity and BMI among individuals growing up and entering adulthood during the rise of obesity in the United States. Sibling fixed effects and cousin fixed effects models partially address unobserved confounding nested in the nuclear as well as extended family. Furthermore, exposure to neighborhood disadvantage in adolescence is most salient, providing insight into when policy intervention may be most effective. Results are robust to alternative specifications for neighborhood disadvantage, ages of exposure, and to alternative sampling strategies.

Keywords:

Neighborhood disadvantage, obesity, fixed effects

Funding:

There are no funding agencies to report.

¹ 336 Uris Hall, Ithaca, NY 14850; alvarado@cornell.edu; 607.255.6289 (phone); 607.255.8473 (fax)

1. Introduction

Obesity threatens the well-being of over 93 million individuals in the United States because it increases the risk of weight-related morbidity such as heart disease, stroke, type 2 diabetes and certain cancers (Diez-Roux and Mair, 2010; National Center for Health Statistics, 2015; OECD, 2014). Efforts to eradicate obesity in the U.S. have been generally unsuccessful (Zylke and Bauchner, 2016), with obesity currently afflicting 40 percent of adults (Flegal et al., 2016; Ogden et al., 2016). Its geographic concentration amidst pockets of poverty has also made it a central feature of contemporary inequality research. Indeed, scholars have found that where one resides can in fact predict the chances of becoming obese (Black and Macinko, 2008, 2010), suggesting that variation in resources in the local residential context can predict unhealthy weight. What remains underdeveloped, however, is an understanding of the long-term association between residential context and obesity across the life course (Kravitz-Wirtz, 2016). Specifically, we know little about how childhood neighborhood context and adult obesity are associated and how variation in exposure across developmental stages may moderate this association.

The rise in obesity has acutely affected individuals from low-socioeconomic backgrounds (Lochner, 2011; Ogden et al., 2010; Sobal and Stunkard, 1989). Accordingly, place-based research on obesity and related health outcomes has proliferated (Black and Macinko, 2008; Diez-Roux and Mair, 2010; Sampson, 2003a, 2003b). Given the negative consequences associated with obesity on educational attainment, wages, employment, and medical care costs (Cawley, 2015), it is imperative that scholars and policymakers gain a clearer understanding of how neighborhoods contribute to obesity. Furthermore, of special interest to sociological theory

about neighborhood effects and policy is an understanding of how and when neighborhood influence manifests (Ellen and Turner, 1997; Sharkey and Faber, 2014).

The current analysis asks two main questions: (1) Is childhood neighborhood disadvantage associated with adult obesity and (2) does the timing of exposure moderate this association? This paper capitalizes on 28 years of restricted geocoded data (1986 – 2014) from the National Longitudinal Survey of Youth, 1979 (NLSY79) and the NLSY Children and Young Adults cohort (NLSY:CYA) that, to my knowledge, have never been used before to assess the association between childhood neighborhood disadvantage and adult obesity. These data are well-suited to study this topic because they extend across the exact same time period when both income segregation and obesity expanded in the United States and because they allow for fixed effects models that control for unobserved confounders.

2. Concentrated disadvantage

The geographic concentration of disadvantage limits individuals' life chances by restricting access to essential social and economic resources (Firebaugh and Farrell, 2015; Massey and Denton, 1993; Sharkey, 2013; Sharkey and Elwert, 2011; Wilson, 1987). The trend in segregation by socioeconomic status (SES), in particular, has had enormous impact on the ability of residents of disadvantaged neighborhoods to improve their life-chances (Reardon and Bischoff, 2011; Reardon et al., 2015; Wilson, 1987, 1991). Moreover, unhealthy environments are often passed down from one generation to the next (Sharkey, 2013).

Neighborhoods need special attention as one source of the obesity epidemic because of their ability to encapsulate the “obesogenic” conditions, which include prevailing norms, social connectedness, conditions of the built environment, sources of food, and safe places to exercise,

that surround individuals and may be associated with weight (Lipek et al., 2015; Swinburn et al., 1999).

Critical, however, to estimating neighborhood effects on obesity is addressing the problem of unobserved selection (e.g., the bias introduced by imbalances of underlying time-constant family characteristics that affect neighborhood selection and health such as motivation, genes, behaviors, and other attributes that often go unmeasured in observational data) that could render seemingly statistically significant effects spurious (Diez-Roux and Mair, 2010). Beyond obesity and other health outcomes, selection bias challenges research that attempts to estimate all contextual effects (Durlauf, 2004; Fleischer and Roux, 2008; Hauser, 1970, 1974; Oakes et al., 2015). One method to address unobserved selection is by using fixed effects. Building on previous neighborhood scholars that have used fixed effects (Aaronson, 1998; Vartanian and Houser, 2012; Yang and South, 2018), I use both sibling and cousin fixed effects to control for unobserved confounders nested at the level of the nuclear and extended family, respectively. Fixed effect models adjust for anything that may be unobserved and invariant between family members that could impact neighborhood selection and outcomes and therefore bias results (Allison, 2009; Gangl, 2010; Greene, 2008; Halaby, 2004; Johnston and DiNardo, 1997; Oakes, 2004; Wooldridge, 2002). The major weakness of the FE approach is that unobserved characteristics that differ between family members, such as parents' psychological and emotional states, could impact respondent's differently and therefore bias results. Still, any psychological dispositions, such as those that are genetically rooted and pose problems in the sibling FE models, should be at least partially addressed in the cousin FE models.

Along with addressing issues of endogeneity, assessing the long-term consequences of neighborhood disadvantage using panel data is an equally pertinent research goal (Boone-

Heinonen and Gordon-Larsen, 2012; Entwisle, 2007; Rodgers GP and Collins FS, 2012).

Studying the long-term effects of exposure to disadvantaged neighborhoods on obesity across the life course can shed light on the lingering impact of childhood context on health.

2.1 Neighborhood SES and weight

The social and economic profiles of neighborhood residents mold the environment in which behaviors and attitudes can become normative and can influence weight outcomes (Crane, 1991; Jencks and Mayer, 1990; Kawachi and Berkman, 2003; Sampson, 2003a). To my knowledge, only three studies have examined the influence of childhood neighborhood conditions on adult obesity. Kravitz-Wirtz (2016) used Panel Study of Income Dynamics (PSID) data and found that childhood exposure, especially during adolescence, to neighborhood disadvantage increased the likelihood of obesity in adulthood. The Kravitz-Wirtz paper provides essential foundational knowledge about the relationship between childhood neighborhood disadvantage and adult obesity. Key differences are that the Kravitz-Wirtz paper only examined obesity between 18 and 30 while the current paper examines obesity through age 42, Kravitz-Wirtz do not control for unobserved confounders, the sample size of the current paper is roughly 5.5 times that of the Kravitz-Wirtz paper, and the current paper examines all races and ethnicities (rather than simply black and white respondents in the Kravitz-Wirtz paper). Therefore, the current paper builds off of the Kravitz-Wirtz paper and extends our knowledge about this process by examining obesity across a longer period of adulthood, controlling for unobserved confounders, and including a much larger and diverse sample of respondents. Critically, controlling for unobserved selection bias may yield stronger inferences about the relationship between childhood neighborhood disadvantage and adult obesity. Lippert (2016) found that

neighborhood poverty in childhood was positively associated with obesity in adulthood. This study did not separate childhood and adult neighborhood conditions and did not account for unobserved selection bias. Vartanian and Houser (2012) used sibling fixed effects and found that socioeconomically advantaged neighborhoods amplified the positive influence of childhood Supplemental Nutrition Assistance Program (SNAP) usage on BMI in adulthood. These authors also found differential effects for the interaction between SNAP and neighborhood quality by developmental periods.

Other scholars have focused on the influence of adult neighborhood SES on weight in adulthood. For instance, Ruel and colleagues (2010) and Robert and Reither (2004) found that neighborhood disadvantage was positively associated with adults' BMI. The former study was limited in its ability to even partially account for unobserved selection bias through quasi-experimental techniques and also only examined women. The latter study was similarly limited in its ability to account for unobserved selection bias and was also limited because it only examined cross-sectional data. Harrington and Elliot (2009) found similar results among a cross-section of Canadian adults, but were also limited by their ability to account for unobserved selection bias. Yang and South (2018) use NLSY data – from the 1979 cohort only – and find that adult neighborhood poverty is associated with adult BMI among whites, but not among blacks or Latinos. They also control for unobserved confounders by using within-person fixed effects. Experimental evidence from the Moving to Opportunity (MTO) project demonstrates that the offer to move to a lower-poverty neighborhood can have beneficial long-term effects on obesity and weight (Kling et al., 2007; Ludwig et al., 2011; Sanbonmatsu et al., 2012). However, the specialized sample of families living in public housing in high poverty neighborhoods in only 5 cities, among other elements, largely limit the generalizability of MTO findings (Clampet-

Lundquist and Massey, 2008). Rarely have scholars been able to examine the long-term association between childhood neighborhoods and adult obesity.

2.2 Life-course

Exposure to socioeconomically disadvantaged environments can have a lasting detrimental effect on one's health (Diez-Roux and Mair, 2010; Kawachi and Berkman, 2003), and scholars are beginning to gain a clearer understanding of the pathways that facilitate how neighborhoods “get under the skin” across the early life-course (Hertzman and Boyce, 2010). Generally, scholars have explained the association between neighborhood SES and health outcomes in two main ways: (1) the influence that the neighborhood physical built environment has on health and (2) the influence that the neighborhood social environment has on health (Diez-Roux and Mair, 2010). I will discuss the literature on these two pathways in turn before discussing a third mechanism that has received much less attention; epigenetics. Although I discuss potential pathways to provide a conceptual idea of underlying processes, I lack the data to formally test these mechanisms.

Access to physical activity and the local food environment may affect obesity and BMI (Black and Macinko, 2008; Black et al., 2010; Larson et al., 2009; Papas et al., 2007). Previous studies also suggest that food insecurity may increase the likelihood of being obese (Beaulac et al., 2009; Boone-Heinonen et al., 2011; Medicine and Council, 2009). While some residents of disadvantaged communities travel substantial distances to purchase food (Hillier et al., 2011), disadvantaged communities remain largely deficient in terms of nutritious food options. Yet, scholars have found that adults' dietary behavior or obesity are little affected by adding a grocery store to a “food desert” (Cummins et al., 2014).

Aspects of the social environment such as prevailing norms, social stressors, and social cohesion can also explain the association between neighborhood SES and health outcomes. Some studies have examined neighborhood social environments and weight, generally finding mixed evidence (Burdette et al., 2006; Cohen et al., 2006; Glass et al., 2006; Mujahid et al., 2008). Nevertheless, growing up in neighborhoods where residents are not only afforded the opportunity to safely access aspects of the physical environment but are also surrounded by peers, adults, and institutions that value healthy and active lifestyles may imprint on children and mold their motivations for pursuing the activities that will lead to healthy weight in adulthood.

A life-course approach that incorporates early-life processes on disease outcomes in adulthood and stresses the embeddedness of individuals in a life-long process of time-dependent social roles and relationships nested within evolving ecological settings represents a theoretically compelling but empirically challenging mandate of research (Diez-Roux and Mair, 2010; Elder et al., 2015; Kuh and Ben-Shlomo, 2004). Indeed, attention to the idea of long-term neighborhood effects coheres to the recent focus that medical researchers have placed on life-course health development, which stresses a dynamic model of health trajectories nested in genetic, biological, behavioral, social, and economic contexts that evolve over time (Halfon and Hochstein, 2002; Halfon et al., 2014). Early exposure to neighborhood disadvantage may increase the likelihood of being obese in adulthood vis à vis encumbrances on healthy social, economic, psychological, and physical development.

Embedded within the life-course perspective is the insight that constitutes the theoretical foundation of the current study: Neighborhood effects may linger over time. However, beyond correlational studies (Fletcher and McLaughlin, 2015), very little is known regarding the association between neighborhood conditions in childhood on obesity in adulthood.

Lippert (2016) found that sustained exposure to neighborhood poverty throughout adolescence and early adulthood increased the likelihood of women, but not men, becoming obese in early adulthood compared to those who never lived in poor neighborhoods, net of observed adjustments. Still, the Lippert (2016) study only examined neighborhood poverty, ignoring other factors that can contribute to an obesogenic environment. Moreover, it did not adjust for adult exposure, which may explain away any childhood neighborhood effects, or for unobserved confounders.

An epigenetic (Hertzman and Boyce, 2010) framework suggests that changes in the environmental conditions during sensitive developmental epochs may explain differences in gene-expression that can lead to differences in obesity and other health outcomes. The effects of the built and, in particular, social environment could very well play a role in gene expression. The epigenetic model may help improve our understanding of the pathway linking childhood neighborhoods and adult obesity because it combines the built environment, social development, and genetic processes. That is, differences in gene-by-environment interactions may drive differences in obesity outcomes if siblings, who share parental gene stems, are exposed to different prenatal or postnatal neighborhood conditions. This approach to conceptualizing disease is embedded within the larger framework provided by the developmental origins of health and disease approach, which stresses how events that take place at the earliest possible stages of development can have lasting effects on health throughout the life-course (Gillman, 2005; Mcmillen and Robinson, 2005; Oken and Gillman, 2003). In particular, the fetal origins hypothesis (Barker et al., 2006) suggests that in utero disruptions such as stressful neighborhood conditions could influence fetal nutritional development and could potentially affect the likelihood of obesity. Moreover, sociologists are beginning to combine knowledge from biology

and genetics to inform long-held understandings of social behavior (Freese, 2008). Epigenetics is therefore a potentially fruitful framework to augment traditional understandings of how neighborhoods affect health.

2.4 Timing of exposure

Children's sensitivity to their environment may change as they pass through different stages of development (Aber et al., 1997; Elder et al., 2015; Hertzman and Boyce, 2010). Epidemiologic and developmental literatures both stress the importance of critical ages when children are most vulnerable to disease risk. For instance, the sensitive period model (Ben-Shlomo and Kuh, 2002) stresses that exposure to deleterious conditions during developmentally susceptible stages may have a stronger effect on disease risk than it does at other times during development.

Scholars have argued that neighborhood contexts may have stronger effects during adolescence because of the increased autonomy and interaction with neighborhood peers and institutions during this stage of development (Aber et al., 1997; Ellen and Turner, 1997). Indeed, researchers have begun to find more pronounced neighborhood effects during adolescence for childhood developmental and obesity outcomes (Alvarado, 2016a, 2016b), teen childbearing (Wodtke, 2013), and high school attainment (Wodtke et al., 2016). If neighborhood effects work through engagement with neighborhood peers, institutions, adults (Jencks and Mayer, 1990; Kawachi et al., 2008; Sampson and Raudenbush, 1999), or through aspects of the built environment (Feng et al., 2010), then the influence of neighborhood ecology could change as children become more engaged with that ecology as they mature.

As children age, their social connection outside of their immediate families begin to expand (Berk, 2003). Although there are no strict age demarcation separating early childhood from adolescence in the literature on developmental sensitivity to neighborhood ecology, scholars often rely on transition points that coincide with key stages of the early life-course (Ellen and Turner, 1997). These include educational shifts such as the transition from elementary school to middle school, which represents a period of rapid physical, emotional, and social development (Aber et al., 1997). Furthermore, in light of the previous discussion of epigenetics, biological development during adolescent (e.g., puberty, growth spurts, and brain maturation) may increase vulnerability for epigenetic changes (Aylwin et al., 2019; Mychasiuk and Metz, 2016), although research in this area is nascent and ongoing (Fisher et al., 2018). Scholars studying heterogeneous neighborhood effects by age define childhood as approximately ages 10 and younger and adolescence as approximately 11 and older and use this cutoff as the approximate age when the gradual shift away from the family as the center of influence and toward neighborhood peers and institutions begins for many children (Aber et al., 1997; Vartanian and Buck, 2005).

2. 5 Contributions

This study contributes to the larger neighborhood effects literature in five ways: (1) it applies life-course concepts and methods regarding developmental stages; (2) it examines the effects of neighborhood disadvantage in youth on adult obesity; (3) it overcomes place and time-dependent constraints to understanding neighborhood effects by using 28 years of longitudinal data at the national level; (4) it addresses the thorny methodological challenge of selection bias by using fixed effects models that adjust for unobserved confounders nested within the family;

and (5) it introduces a new data set to the study of neighborhood effects on adult obesity. Here, I expect that childhood neighborhood disadvantage will be positively associated with adult obesity and that adolescent exposure will be more strongly associated with obesity than early childhood exposure.

3. Methods

3.1 Data

I analyze two cohorts of restricted geocoded (tract-level) data from 15 waves (1986 – 2014) of the National Longitudinal Survey of Youth: the 1979 cohort of mothers (NLSY79) and the Child and Young Adult cohort (NLSY:CYA). The Bureau of Labor Statistics (BLS) has deemed these data containing Census tract identifiers of the mother as highly sensitive and therefore requires a federal clearance to access them on-site in Washington, DC. Only female respondents who have ever had children are linked to the NLSY:CYA cohort. Yet, family background measures remain mostly intact because I include key household information that captures contributions from both parents.

The NLSY:CYA is composed of biennial data on all children born to the NLSY79 female respondents and represents children born to a nationally representative sample of women aged 21-28 years on January 1, 1986.¹ That is, the NLSY:CYA captures data for every child that was born by 1986, and all children who were subsequently born after 1986 to female NLSY79 respondents. These data provide comprehensive information about parents and children as well as capture all sibling pairs within families throughout childhood and into adulthood. By 2014, 10,503 of the children of the NLSY79 females had been interviewed at least once, the oldest child being 42 years old. On average, NLSY79 women have had about two children spaced 3.5

years apart. Although the number of respondents with siblings changes from wave to wave, in 2014 there were 4,537 young adults over age 14 (out of 5,735 young adults [79%]) who had a sibling in the sample. I imputed to correct for missingness using the “ice” command in Stata (Allison, 2002; von Hippel, 2007).

I followed previous neighborhood effects researchers by operationalizing neighborhoods using tracts (Brooks-Gunn et al., 1993; Vartanian and Buck, 2005; Wodtke et al., 2016) and linearly interpolating for inter-Census waves. I merged neighborhood data from the Geolytics Neighborhood Change database to the geocoded NLSY data. Geolytics provides data on tract socioeconomic characteristics for 1980, 1990, and 2000 using the Census long form, normalized to 2000 tract boundaries, and it also provides data from the 2005-2009 American Communities Survey (ACS) that is also normalized to 2000 tract boundaries. The 2010 ACS is no longer normalized to 2000 so it is incompatible with the NLSY79 geocode data (which is only normalized to 2000 and before). All neighborhood characteristic data are from Geolytics.

3.2 Neighborhood disadvantage

I define neighborhood disadvantage as a constellation of factors that collectively proxy the multidimensional underlying contextual disadvantage present in a neighborhood. First, I created a scale which represents the mean of the following seven variables measured in each wave when the child was between 0 and 18 and was presumably living with the mother: percent of residents at or below 100% of the poverty threshold as defined by the U.S. Census Bureau, the percent of residents who are unemployed, the percent of residents out of the labor force, the percent who have at least a Bachelor’s degree (reverse coded), the percent of managers and professionals in the neighborhood (reverse coded), median income (reverse coded), and the

median housing value (reverse coded). Collectively, these variables cohered into a single approximation of neighborhood disadvantage ($\alpha = .88$). Second, I divided the scale into quintiles (1 = the least disadvantaged neighborhood and 5 = the most disadvantaged neighborhood) and assigned a quintile score to each respondent at each of the 15 waves. Finally, I calculated the mean quintile score for each respondent across the following age ranges: (1) 0 – 18; (2) 0 – 10; and (3) 11 – 18. This continuous quintile score serves as the main independent variable.

3.3 Obesity

I created a dichotomous outcome to indicate whether the NLSY:CYA respondents ever experienced obesity at any point as adults (i.e., between ages 19 – 42). I first used the self-reported measure of height and weight in adult (over 18) wave T to calculate body mass index (BMI) in wave T for NLSY:CYA respondents.² Second, I created an obesity indicator (i.e., 1 = having a BMI ≥ 30 and 0 = not) in wave T of adulthood so that a given respondent had as many indicators of obesity as they had waves of observation. If respondents ever had 1 on any of the per-wave indicators of obesity over the age of 18, then they received a 1 on the final obesity dummy; otherwise 0. I also examine mean adult BMI (which varies per wave) as an outcome to complement the obesity indicator.

3.4 Observed adjustments

Table 1 provides a full summary (means and standard deviations) of the observed variables that I adjust for in this analysis. The BLS measured each variable at each of the 15 waves spanning childhood and adulthood. These include both time-varying and time-invariant

adjustments and all of the mother specific variables are limited to when the child was between 0 and 18 years of age.

[Table 1 about here]

These adjustments include whether the respondent's mother was ever obese and whether the respondent was ever obese in childhood (0 – 18). I also include a subjective measure of adult neighborhood disadvantage for two reasons. First, geocodes are only tied to the NLSY79 respondents, not the NLSY:CYA respondents – so I do not reasonably know the census tract residential locations of NLSY:CYA respondents once they become adults and presumably leave the home. Second, debate continues to surround the proper definition of a neighborhood, with many scholars arguing that tracts and other administrative units do not properly or completely reflect individuals' geographic contexts of influence (Cummins et al., 2007; Diez-Roux, 2001; Matthews et al., 2005). These subjective assessments of the neighborhood characteristics that respondents lived in as adults allow the respondent to define what s/he means by a “neighborhood.” The adult neighborhood disadvantage scale is a composite of NLSY:CYA respondents' concern that the following conditions are a problem in their neighborhoods as adults in each wave: 1) people do not respect rules/laws; 2) crime and violence; 3) abandoned/run-down buildings; 4) parents do not supervise their children; 5) people do not care/keep to themselves; and 6) people cannot find jobs. The Chronbach's α is 0.81 for adult subjective neighborhood disadvantage.

3.5 Statistical analysis

I begin by analyzing bivariate pooled logistic models to get a sense of the zero-order association between childhood neighborhood disadvantage and adult obesity. Next I include all observed adjustments in multivariate pooled logistic models to assess how much of the association is explained by observed adjustments. Finally, I incorporate sibling fixed effects that improve upon the logistic models by addressing unobserved “hidden” confounding. There may be some selection bias based on cultural and class sorting engrained in the FE models because of the requirement that families must contain at least two children to be included in this analysis. In addition, the sibling FE analyses may only generalize to families with siblings. Fortunately, most (60%) families have at least two children while many have even more (e.g., 28% have three or more and 10% have four or more). The FE model capitalizes on variation in neighborhood conditions that result from 1) older and younger siblings experiencing different neighborhood conditions due to moving and 2) neighborhood conditions changing around separate siblings over time.³ It is the spacing of siblings (mean is 3.5 years between siblings), however, that facilitates the estimation of sibling FE models from the NLSY:CYA data.

Following Allison (2009), the multivariate logistic sibling FE model where the outcome is a dichotomous variable for adult obesity for child i in family f takes the following form:

$$\log\left(\frac{p_{if}}{1 - p_{if}}\right) = \mu_t + \beta \mathbf{x}_{if} + \gamma \mathbf{z}_i + a_i, \quad f = 1, 2, \dots, F$$

where p_{if} is the probability that the child ever experiences obesity in adulthood is equal to 1, where $\mathbf{x}_{if}$ represents a vector of within-family varying predictors and $\mathbf{z}_i$ represents a vector of within-family invariant predictors, and where a_i represents the combined effects of all unobserved variables that are constant within families. Unobserved constant variables may include the degree of love and warmth in the home, genes, parent’s emotional and mental

stability, and other salient characteristics of the home environment that are not measured. In the logistic fixed effects approach, a_i is assumed random and is able to freely associate with $\mathbf{x}_{if}$ in any way. The logistic sibling FE model uses conditional maximum likelihood, which causes the constant, μ_f , to go unreported because the constant is conditioned out of the likelihood function.

I supplement these models with *cousin* FE models that further adjust for unobserved confounders nested at the level of the grandparent and that flow downstream across three generations. That is, the cousin FE models account for any unobserved legacies of disadvantage that are passed down from the parents of the NLSY79 female respondents to their grandchildren, the NLSY:CYA respondents. The cousin FE models only control for unobserved confounders for the maternal side of the family. However, unlike the sibling FE models, the cousin FE models allow for the inclusion of respondents that a) did not have siblings and/or b) did not experience within-family variation in neighborhood conditions or the outcome.

4. Results

4.1 Main results

I present results from analyses of pooled bivariate, pooled multivariate, and multivariate fixed effects models that examine the association between childhood neighborhood disadvantage and obesity in adulthood. Figure 1 depicts the bivariate association between childhood exposure to neighborhood disadvantage and obesity across adulthood – demonstrating that growing up in disadvantaged neighborhood increases obesity in adulthood. Specifically, a one-standard deviation increase in neighborhood disadvantage quintile between ages 0 and 18 increases the odds of being obese in adulthood by 10 percent ($e^{[0.41]*0.23} = 1.10$); a one-standard deviation increase in neighborhood disadvantage quintile between ages 0 and 10 increases the odds of

being obese in adulthood by 13 percent ($e^{[0.37]*0.34} = 1.13$); and a one-standard deviation increase in neighborhood disadvantage quintile between ages 11 and 18 increases the odds of being obese in adulthood by 14 percent ($e^{[0.35]*0.37} = 1.14$). All of the bivariate coefficients are statistically significant at the 5 percent level. Although suggestive, these baseline results are limited in inference and could be spurious.

[Figure 1 about here]

The next series of models, summarized in Table 2, use logistic regression and introduce adjustments for observed and unobserved confounders that may explain the zero-order results in Figure 1. One should note that Models 4 and 8 adjust for childhood exposure while models 3 and 7 do not.

[Table 2 about here]

In models 1 – 4 of Table 2, the results suggest that exposure to neighborhood disadvantage at any point in childhood has a positive association with obesity in adulthood, net of observed adjustments. Models 2 and 3 suggest equivalent positive associations for early childhood and adolescent exposure to neighborhood disadvantage. Model 4 demonstrates that the coefficient for adolescent exposure decreases after adjusting for early childhood exposure. Yet, a post hoc test of mean difference (see equation 4 in Paternoster et al., 1998) shows that the difference between the coefficients in models 2 and 4 is not statistically significant, suggesting that early childhood exposure to neighborhood disadvantage and adolescent exposure may be equivalent in magnitude, net of observed adjustments.

Models 5 – 8 introduce sibling fixed effects to adjust for unobserved differences between siblings that could bias results from the traditional logistic regression in models 1 – 4. The sibling FE results, also depicted in Figure 2, suggest that youth exposure to neighborhood disadvantage continues to be positively associated with obesity in adulthood, net of observed and unobserved potential confounders. Specifically, the sibling FE results suggest that a one-standard deviation increase in neighborhood disadvantage quintile at any point between the ages of 0 and 18 (model 5) increases the odds of being obese in adulthood by 31 percent ($e^{[1.19]*0.23} = 1.31$), net of observed and unobserved confounders. A one-standard deviation increase in neighborhood disadvantage quintile between ages 0 and 10 (model 6) increases the odds of being obese in adulthood by 13 percent ($e^{[0.35]*0.34} = 1.13$), net of observed and unobserved confounders. Finally, a one-standard deviation increase in neighborhood disadvantage quintile between ages 11 – 18 (model 8) increases the odds of being obese in adulthood by 29 percent ($e^{[0.69]*0.37} = 1.29$), net of observed and unobserved confounders and net of early childhood exposure. Moreover, the difference between early childhood and adolescent exposure is statistically significant after testing for difference between these two coefficients (Paternoster et al., 1998), suggesting that adolescent exposure to neighborhood disadvantage is more salient than early childhood exposure for adult obesity. I also run non-FE analyses on the sibling sample to check if the sibling sample is driving these results (see online Appendix A). These results demonstrate agreement with the findings in Table 2, suggesting that the sibling sample is not driving the results.

[Figure 2 about here]

4.2 Alternative specification of timing of exposure

Although the previous analyses were based on developmental theory in regards to the timing of exposure to neighborhood disadvantage, there is no standardized method of defining “early childhood” or “adolescence.” Therefore, I created alternative specifications of the timing of exposure to neighborhood disadvantage. For instance, I defined three age periods as “early childhood” that spanned ages 0 – 7, “middle childhood” that spanned ages 8 – 12, and “adolescence” that spanned ages 13 – 18.

Table 3 summarizes these results from sibling FE logistic regression models and suggests that youth neighborhood disadvantage has statistically a significant positive association with adult obesity and that this association grows in magnitude over developmental stages so that they are most acute in adolescence. In fact, exposure during the earliest stage of childhood does not exert any statistically significant association with adult obesity. The results in Table 3 suggest that a one-standard deviation increase in neighborhood disadvantage quintile in adolescence (model 3) increases the odds of being obese in adulthood by 16 percent ($e^{[0.76]*0.20} = 1.16$), net of observed and unobserved confounders. These alternative results are in line with the main results in Table 2 and add credibility to the finding that youth exposure to neighborhood disadvantage has a negative association with healthy weight in adulthood. Online Appendix B shows sibling FE results for the analysis of a four-age-segment version of timing of exposure (0-5; 6-10; 11-14; and 15-18) and these results show once again that (1) childhood neighborhood disadvantage is positively associated with adult obesity and (2) that adolescent exposure is most salient, adding further support for the findings in Table 2.

There remains the possibility that including child-level predictors measured during adulthood could mediate and confound childhood neighborhood effects. Education, income, and (perceived) neighborhood disadvantage may be especially likely to explain the association

between childhood neighborhood disadvantage and adult obesity. Therefore, in Appendix C, I summarize results for sibling fixed effects logistic models that remove these adult covariates. The results are in line with the results from Models 5 – 8 in Table 2 and suggest that 1) childhood neighborhood disadvantage is positively associated with adult obesity; 2) adolescent exposure is more salient than early childhood exposure; and 3) the results from Table 2 are likely underestimates of the true effect of childhood neighborhood disadvantage. Moreover, the adult covariates present in the models in Table 2 do not explain away the effects of childhood neighborhood disadvantage on adult obesity.

[Table 3 about here]

4. 3 Alternative neighborhood disadvantage specifications

The previous results are based on a continuous specification of neighborhood disadvantage (i.e., the mean of the neighborhood disadvantage quintile scores across 0 – 18, 0 – 10, and 11 – 18). However, an alternative specification that compares respondents who grew up, on average, in the most severely disadvantaged neighborhoods to those who grew up in less disadvantaged (i.e., more advantaged) neighborhoods could alter the story further. That is, I created a dichotomous variable to indicate whether the respondent grew up in a neighborhood above the 60th percentile of disadvantage (coded as 1) or below the 60th percentile (coded as 0), on average across all waves within specific ages ranges (0 – 18, 0 – 10, and 11 – 18).

Table 4 demonstrates the sibling FE logistic regression results for exposure to the more severe specification of neighborhood disadvantage on obesity across adulthood (quintiles 1 – 3 versus quintiles 4 and 5). The results suggest that youth exposure to neighborhood disadvantage

continues to have a statistically significant positive association with adult obesity, in accord with the findings in Table 2. For instance, a one-standard deviation increase in exposure to severe neighborhood disadvantage between the ages of 11 – 18 (model 4) increases the odds of being obese in adulthood by 11 percent ($e^{[0.64]*0.16} = 1.11$), net of observed and unobserved confounders and net of early childhood exposure. Despite the large difference in the coefficient sizes between early childhood and adolescent exposure in Table 4, they are technically statistically equivalent. These results would likely align with the results in Table 2 in terms of the salience of adolescent exposure were it not for the relatively extreme (and rare) comparisons being made between siblings' neighborhood contexts in Table 4 and the larger standard errors that this produces.

[Table 4 about here]

As an additional robustness check, I also created another continuous neighborhood disadvantage score. Instead of using the mean of the quintiles, however, I created the mean of the raw neighborhood scale scores in waves across the different age periods and then created the quintiles for the age periods. Here, each respondent has a quintile value across the various age periods that is based on the mean of the raw scale scores across waves (and therefore preserves more variation in neighborhood context). Table 5 demonstrates that, when using the alternative continuous measure of neighborhood disadvantage, the general conclusions remain the same as those in Table 2. That is, youth exposure to neighborhood disadvantage is positively associated with adult obesity; and adolescence is the most salient stage of development when these effects take hold, net of observed and unobserved potential confounders. For instance, a one-standard deviation increase in neighborhood disadvantage quintile between the ages of 11 – 18 (model 4)

increases the odds of being obese in adulthood by 24 percent ($e^{[0.41]*0.53} = 1.24$), net of observed and unobserved confounders and net of early childhood exposure. These results align with the main findings in Table 2. In fact, the sibling FE results for adolescent exposure in the original specification in Table 2 and in the alternative specification in Table 5 are quite similar, increasing the odds of adult obesity by 29 percent and 24 percent, respectively. Finally, I simply used the raw average index score (proportion). The results, summarized in Appendix D, also align with the sibling FE (models 5 – 8) results in Table 2 – that is, childhood neighborhood disadvantage is positively associated with adult obesity and adolescent exposure is more salient than early childhood exposure. This provides further confidence in the main findings of this paper.

[Table 5 about here]

4.4 Cousin fixed effects

Recent findings suggest that where one lives reflects inequality rooted in previous generations (Sharkey, 2013; Sharkey and Elwert, 2011). The legacy of disadvantage that is embedded in previous generations may also impact one's outcomes. Therefore, to partially account for disadvantage that cascades down from the grandparents' generation to the grandchildren, I examine cousin FE models.

Table 6 displays results from the cousin FE logistic regression models for the main continuous measure of neighborhood disadvantage and suggests that, similar to the analysis in Table 2, neighborhood disadvantage in childhood and adolescence is positively associated with obesity in adulthood, net of observed and unobserved confounders nested in the grandparent

generation. A one-standard deviation increase in neighborhood disadvantage quintile between the ages of 11 – 18 (model 4) increases the odds of being obese in adulthood by 31 percent ($e^{[0.62]*0.44} = 1.31$), net of observed and unobserved confounders and net of early childhood exposure. However, despite the fact that the coefficient for adolescent exposure is larger, adolescent exposure is not technically statistically different from early childhood exposure in the cousin FE models.

[Table 6 about here]

4.5 Body mass index

I supplemented the obesity findings with analyses of adult BMI to gain a more comprehensive understanding of how childhood neighborhood disadvantage affects adult weight. Table 7 shows results from sibling FE linear regression models and suggests that adolescent exposure (model 4) has a statistically significant positive association with BMI in adulthood, but early childhood exposure (model 2) does not. These BMI results for the main continuous specification of neighborhood disadvantage are in line with the results for obesity in Table 2.

[Table 7 about here]

5. Conclusions

Obesity scholars have emphasized the need for more long-term studies that uncover the connection between residential context and weight at different stages of the life-course (Boone-Heinonen and Gordon-Larsen, 2012; Rodgers GP and Collins FS, 2012). Using restricted geocoded NLSY data that spans both the rise in socioeconomic segregation and obesity in the

United States, I find that childhood exposure to neighborhood disadvantage increases the odds of obesity in adulthood, net of observed and unobserved confounders, and that adolescent exposure may be particularly salient.

This paper contributes to the understanding of the association between neighborhood disadvantage during childhood and adult obesity by using novel data and a life-course theoretical framework which stresses the developmental sensitivity of children to ecological influences. Earlier findings from the five city MTO suggest that the opportunity to move to less-poor neighborhoods reduces obesity among a select sample of poor adults (Kling et al., 2007; Ludwig et al., 2011; Sanbonmatsu et al., 2012). Cohering to the national-level findings of the current paper, Kravitz-Wirtz (2016) used PSID data and found that adolescent exposure to neighborhood disadvantage was particularly salient in predicting obesity between ages 18 and 30. An analysis of national Add Health data also found that neighborhood poverty increased obesity during the transition to adulthood for women (Lippert, 2016). Further, a multiplicative sibling FE analysis of PSID data found that receiving SNAP in childhood and growing up in an advantaged neighborhood combined to increase adult BMI (Vartanian and Houser, 2012).⁴

Building off of this previous research, the sibling fixed effects analyses (Table 2) in this paper reveal that exposure to childhood neighborhood disadvantage is positively associated with obesity in adulthood, net of observed and unobserved confounders. This finding aligns with the earlier finding by Lippert (2016) after adjusting for adult exposure and unobserved confounders nested within the nuclear family. Moreover, the results in Table 2 may represent conservative estimates of childhood neighborhood effects on adult obesity, given that inclusion of adult covariates may suppress the true size of the childhood neighborhood effect (as suggested by the models that exclude these adult covariates in Appendix C). Cousin fixed effects models that

further adjust for unobserved legacies of disadvantage that cascade across three generations (Table 6) reinforce this finding for neighborhood effects on adult obesity. Moreover, the current study expands upon earlier findings from MTO and Add Health by examining characteristics of neighborhoods beyond simply the percentage of poor residents, addressing unobserved confounding for a national sample, and adjusting for adult neighborhood disadvantage. The finding here answer the first research question in the affirmative and is consistent throughout the various modeling strategies. However, the sibling FE models also reveal that the timing of exposure may be a relevant factor, as previous theories of neighborhood effects suggest.

The analyses here suggest that exposure to neighborhood disadvantage during adolescence yields stronger associations with adult obesity compared to exposure during early childhood, a finding that coheres to both prior theory on ecological sensitivity across stages of development (Aber et al., 1997; Ellen and Turner, 1997) and to prior work on neighborhoods and obesity in youth (Alvarado, 2016b) and adulthood (Kravitz-Wirtz, 2016). The earlier finding from Kravitz-Wirtz (2016), however, was limited in that it only examined obesity between ages 18 – 30 and did not account for unobserved confounders. This finding also answers the second research question in the affirmative. These findings suggest that it may behoove place-based policies aimed at curbing obesity in adulthood to focus more on processes in adolescence, when children’s engagement with their environments may be growing and their absorption of norms, behaviors, and food may be highest. Alternative models where I use different age cutoffs (Table 3 and Appendix B), where I use more severe forms of neighborhood disadvantage (Table 4), or where I increase the variation in the measurement of neighborhood disadvantage (Table 5) all demonstrates that youth neighborhood disadvantage is positively associated with adult obesity and that adolescence is a particularly acute developmental stage where the associations are the

strongest. Finally, examining BMI as an outcome (Table 7) not only reinforces the featured effect of childhood neighborhoods on adult obesity, but also supports the finding that adolescent exposure is most salient. The findings for BMI contradict the null findings from PSID (Vartanian and Houser, 2012) – whom also use sibling FE. Possible explanations for the difference in BMI findings between the current and previous studies include the fact that the NLSY:CYA represents a more recent cohort of respondents that came of age during the rise of obesity and the fact that the NLSY:CYA covers a more expansive adult age range compared to the PSID.

Despite the fact that the current study has applied FE models to estimate neighborhood effects using a novel longitudinal dataset, there remain important limitations to this study. First, the sibling FE model, while accounting for time-invariant unobserved confounders, does not account for unobserved confounders that differ between siblings and is therefore no panacea for the threat of unobserved selection bias. Second, the current study does not address the possibility of cumulative and lagged effects, which could provide even more nuanced insights. Third, there are alternative specifications to unhealthy weight (e.g., waist circumference) which researchers can explore to contrast with the current specifications. Unfortunately, the NLSY is limited by only offering height and weight data. Fourth, the chemical environment and other aspects of the ecological health of neighborhoods are relevant and, unfortunately, missing from these analyses due to data constraints. Future researchers who either have access to genetic data across the life course or can collect it themselves could further investigate epigenetic pathways. The interactions between the built environment, the social environment, and genes could help to account for how neighborhood disadvantage during the critically important adolescent years when social and biological change is happening rapidly can have lasting influence on health outcomes. Fifth, school contexts could be an important missing variable given that students,

especially ones from disadvantaged households, may rely on schools for nutrition. However, despite some correlational evidence that school nutrition policies can mitigate obesity (Veugelers and Fitzgerald, 2005), other more rigorous studies have shown that school socioeconomic quality does little to mediate the effects of neighborhood socioeconomic quality on children's outcomes (Owens, 2010; Wodtke and Parbst, 2017). Unfortunately, the NLSY does not contain school identifiers so I cannot explicitly model both contexts simultaneously.

These limitations notwithstanding, the current analysis contributes new evidence for the long-term association between childhood neighborhood disadvantage and obesity and BMI in adulthood. Further, the current findings illuminate potential stages of development when policy may be most effective at curbing obesity. This study provides evidence to support interventions that are directed at adolescent youth given that exposure to normative and behavioral influences in the neighborhood may have the strongest effects at this stage of development.

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Table 1. Descriptive statistics for analytical sample of NLSY79 mothers & NLSY:CYA youth

	Mean	Standard deviation (between)
Mother & household characteristics		
In poverty	0.25	0.44
Weeks unemployed	3.70	16.28
Number of children in household	1.35	1.24
Household income (logged 2014 dollars)	10.63	1.07
Single parent	0.46	0.50
Parental education: less than high school degree	0.16	0.37
Parental education: high school degree	0.45	0.50
Parental education: some college	0.19	0.39
Parental education: Bachelor's or higher	0.21	0.40
Foreign born	0.07	0.25
Armed Forces Qualifying Test	39843.75	28959.46
Obese	0.55	0.47
Child characteristics		
White	0.52	0.50
Black	0.27	0.44
Latino	0.15	0.36
Female	0.49	0.50
Ever moved during childhood	0.43	0.14
Obese in childhood	0.43	0.32
Birthweight (ounces)	115.92	19.03
Childhood neighborhood		
Neighborhood disadvantage quintile (1 = most advantaged; 5 = most disadvantaged)		
Ages 0 - 18	3.14	1.09
Ages 0 - 10	3.31	1.13
Ages 11 - 18	2.94	1.20
Outcome		
Obese in adulthood	0.66	0.40

Source: NLSY 1979 - 2014; NLSY CYA 1986 - 2014

Table 2. Logistic regression results (without and with sibling FE) for the association between childhood neighborhood disadvantage and ever obese in adulthood (NLSY:CYA 1986-2014)

	Observed adjustments				Observed adjustments & sibling fixed effects			
	Exposure age				Exposure age			
	(1) 0-18	(2) 0-10	(3) 11-18	(4) 11-18	(5) 0-18	(6) 0-10	(7) 11-18	(8) 11-18
Neighborhood disadvantage	0.17*** (0.03)	0.16*** (0.03)	0.16*** (0.02)	0.11*** (0.03)	1.19*** (0.17)	0.35*** (0.10)	0.59*** (0.09)	0.69*** (0.10)
Constant	-2.00** (0.82)	-1.96** (0.82)	-1.98** (0.81)	-2.50** (0.83)	—	—	—	—
Observations	8,430	8,190	8,430	8,190	3,320	3,230	3,320	3,230

Notes: All models control for mother's poverty, mother's weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income. Additionally, Models 4 and 8 control for exposure to neighborhood disadvantage in early childhood.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for clustering.

** p < 0.01; ***p < 0.001

Table 3. Logistic regression results for the association between childhood neighborhood disadvantage and ever obese in adulthood by age of exposure: early childhood, middle childhood, and adolescence (NLSY:CYA 1986-2014)

	Observed adjustments & sibling fixed effects		
	Obesity across adulthood		
	Exposure age		
	(1)	(2)	(3)
	<u>0-7</u>	<u>8-12</u>	<u>13-18</u>
Neighborhood disadvantage	0.19	0.31*	0.76***
	(0.14)	(0.13)	(0.16)
Constant	—	—	—
	—	—	—
Observations	2,870	3,310	3,320

Notes: All models control for mother's poverty, mother's weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for clustering.

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Table 4. Logistic regression results for an alternative specification of childhood neighborhood disadvantage (quintiles 1 - 3 vs. 4 and 5) and ever obese in adulthood (NLSY:CYA 1986-2014)

	Observed adjustments & sibling fixed effects			
	Quintiles 1 - 3 versus 4 - 5			
	Exposure age			
	(1) <u>0-18</u>	(2) <u>0-10</u>	(3) <u>11-18</u>	(4) <u>11-18</u>
Neighborhood disadvantage	0.49** (0.20)	0.37** (0.15)	0.55*** (0.16)	0.64*** (0.17)
Constant	— —	— —	— —	— —
Observations	3,320	3,230	3,320	3,230

Notes: All models control for mother's poverty, mother' weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income. Additionally, Model 4 controls for exposure to neighborhood disadvantage in early childhood.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for
 ** p < 0.01; ***p < 0.001

Table 5. Logistic regression results for the association between childhood neighborhood disadvantage and ever obese in adulthood using specification that averages the standardized continuous index first, then quintiles these scores (instead of using an average of the quintiles) (NLSY:CYA 1986-2014)

	Observed adjustments & sibling fixed effects			
	Exposure age			
	(1) <u>0-18</u>	(2) <u>0-10</u>	(3) <u>11-18</u>	(4) <u>11-18</u>
Neighborhood disadvantage	0.51*** (0.10)	0.05 (0.07)	0.37*** (0.06)	0.41*** (0.07)
Constant	—	—	—	—
Observations	3,320	3,230	3,320	3,230

Notes: All models control for mother's poverty, mother's weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income. Additionally, Model 4 controls for exposure to neighborhood disadvantage in early childhood.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Table 6. Logistic regression results for the association between childhood neighborhood disadvantage and ever obese in adulthood - cousin fixed effects (NLSY:CYA 1986-2012)

	Observed adjustments & cousin fixed effects			
	Exposure age			
	(1) <u>0-18</u>	(2) <u>0-10</u>	(3) <u>11-18</u>	(4) <u>11-18</u>
Neighborhood disadvantage	0.32* (0.14)	0.52*** (0.14)	0.69*** (0.13)	0.62** (0.20)
Constant	—	—	—	—
Observations	860	859	860	859

Notes: All models are fully adjusted. Model 4 also controls for early childhood exposure.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for clustering.

** p < 0.01; ***p < 0.001

Table 7. OLS regression results for the association between childhood neighborhood disadvantage and mean adult body mass index (NLSY:CYA 1986-2014)

	Observed adjustments & sibling fixed effects			
	Exposure age			
	(1) 0-18	(2) 0-10	(3) 11-18	(4) 11-18
Neighborhood disadvantage	4.24*** (1.25)	0.74 (0.78)	2.04** (0.67)	2.28** (0.77)
Constant	-27.24 (22.24)	-1.34 (22.82)	-14.93 (21.38)	-18.24 (24.44)
Observations	3,980	3,950	3,980	3,950

Notes: All models control for mother's poverty, mother's weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income. Additionally, Model 4 controls for exposure to neighborhood disadvantage in early childhood.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for clustering.

** $p < 0.01$; *** $p < 0.001$

Online Appendix A. Logistic regression results for the association between childhood neighborhood disadvantage and ever obese in adulthood using only those respondents with siblings (NLSY:CYA 1986-2014)

	Observed adjustments			
	Exposure age			
	(1)	(2)	(3)	(4)
	<u>0-18</u>	<u>0-10</u>	<u>11-18</u>	<u>11-18</u>
Neighborhood disadvantage	0.08* (0.04)	0.08† (0.04)	0.10** (0.04)	0.11** (0.04)
Constant	-0.95 (1.20)	-0.88 (1.98)	-1.25 (1.19)	-1.45 (1.20)
Observations	3,320	3,230	3,320	3,230

Notes: All models control for mother's poverty, mother' weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income. Additionally, Model 4 controls for exposure to neighborhood disadvantage in early childhood.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for †p < .10; *p < .05; **p < .01; ***p < .001

Online Appendix B. Logistic regression results for the association between neighborhood disadvantage and adult obesity by age of exposure: 0-5; 6-10; 11-14; 15-18 (NLSY:CYA 1986-2012)

	Observed adjustments & sibling fixed effects			
	Obesity across adulthood			
	Exposure age			
	(1)	(2)	(3)	(4)
	<u>0-5</u>	<u>6-10</u>	<u>11-14</u>	<u>15-18</u>
Neighborhood disadvantage	0.15 (0.15)	0.52*** (0.13)	0.64*** (0.13)	0.80*** (0.14)
Constant	—	—	—	—
Observations	2,350	3,230	3,320	3,320

Notes: All models control for mother's poverty, mother's weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income. Additionally, Model 4 controls for exposure to neighborhood disadvantage in early childhood.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for clustering.

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

Online Appendix C. Sibling fixed effects logistic regression results for the association between childhood neighborhood disadvantage and ever obese in adulthood (NLSY:CYA 1986-2014)

	Observed adjustments & sibling fixed effects			
	Exposure age			
	(1) 0-18	(2) 0-10	(3) 11-18	(4) 11-18
Neighborhood disadvantage	1.42*** (0.15)	0.52*** (0.09)	.66*** (0.08)	.75*** (0.09)
Constant	—	—	—	—
Observations	3,510	3,400	3,510	3,400

Notes: All models control for mother's poverty, mother' weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, and child's birthweight. Additionally, Model 4 controls for exposure to neighborhood disadvantage in early childhood. Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for
 ** p < 0.01; ***p < 0.001

Online Appendix D. Logistic regression results (with sibling FE) for the association between childhood neighborhood disadvantage and ever obese in adulthood (NLSY:CYA 1986-2014): Raw scale score.

	Observed adjustments & sibling fixed effects			
	Exposure age			
	(1)	(2)	(3)	(4)
	0-18	0-10	11-18	11-18
Neighborhood disadvantage	1.88*** (0.34)	0.56** (0.20)	0.98*** (0.19)	1.15*** (0.20)
Constant	—	—	—	—
Observations	3,020	2,920	3,020	2,920

Notes: All models control for mother's poverty, mother' weeks unemployed, number of children in the household, mother's logged household income, single parent, mother's education, mother's foreign born status, mother's AFQT score, mother's obesity history, whether or not the mother's household ever moved, race/ethnicity, child's sex, child's obesity history, child's work limitations, child's highest year of schooling, neighborhood disadvantage in adulthood, child's birthweight, and child's adult household income. Additionally, Model 4 controls for exposure to neighborhood disadvantage in early childhood.

Coefficients are shown.

Robust bootstrapped standard errors (in parentheses) are corrected for
 ** p < 0.01; ***p < 0.001

Figure 1. Logistic regression results for the bivariate association between childhood neighborhood disadvantage and adult obesity.

Figure 2. Logistic regression results for the fully adjusted sibling fixed effects association between childhood neighborhood disadvantage and adult obesity.

Notes

¹ Like the PSID Child Development Supplement before it, the NLSY:CYA is not nationally representative. Rather, the PSID CDS and the NLSY:CYA both represent the children of nationally representative samples of the U.S. population in 1968 and in 1979, respectively.

² I used self-reported measures of height and weight to calculate BMI by dividing weight in pounds by height in inches squared times 703 using the following formula: $(\text{weight}[\text{lb}] / \text{height}[\text{in}]^2 * 703)$.

³ For example, 57 percent of the sample never moved and therefore exclusively experienced variation in neighborhood conditions exogenously through changes to their environment around them over time.

⁴ Vartanian and Houser also find differential effects for the interaction between SNAP and neighborhood quality based on timing of exposure. However, one clear difference between my study and theirs lies in the fact that I am not analyzing the interaction effects for receiving SNAP – or any other government aid program – and neighborhoods.