

Original Contribution

Associations of Continuity and Change in Early Neighborhood Poverty With Adult Cardiometabolic Biomarkers in the United States: Results From the National Longitudinal Study of Adolescent to Adult Health, 1995–2008

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Limitations of extant research on neighborhood disadvantage and health include general reliance on point-intime neighborhood measures and sensitivity to residential self-selection. Using data from the US Census and the 1995–2008 National Longitudinal Study of Adolescent to Adult Health, we applied conventional methods and coarsened exact matching to assess how cardiometabolic health varies among those entering, exiting, or remaining in poor and nonpoor neighborhoods. Within the full sample (n = 11,767), we found significantly higher systolic and diastolic blood pressures among those who entered or consistently lived in poor neighborhoods relative to those who never lived in poor neighborhoods. Obesity was similarly more common among those who originated from poor neighborhoods than among those who originated from nonpoor neighborhoods. Having exited poor neighborhoods was associated with lower systolic blood pressure than was consistent residence in low-income communities. Among the matched sample (n = 9,727), results adjusted for confounders and residential selfselection revealed fewer significant contrasts. Compared with peers who had no neighborhood poverty exposure, those who consistently lived in poor neighborhoods had 46% and 52% higher odds of being obese or hypertensive, respectively. Those who exited neighborhoods. These findings underscore the importance of past as well as current residential circumstances for cardiometabolic health.

biomarkers; coarsened exact matching; neighborhoods; transition to adulthood

Abbreviations: Add Health, National Longitudinal Study of Adolescent to Adult Health; CEM, coarsened exact matching; CI, confidence interval; MTO, Moving to Opportunity; OR, odds ratio.

Chronic health conditions are increasingly common among younger people. Approximately 1 in 5 American adolescents is obese (1), and more than 1 in 10 nonobese adolescents become obese by young adulthood (2, 3). Similar shifts have been seen for obesity-related conditions, such as diabetes (4, 5) and hypertension (6, 7). These conditions are major contributors to mortality and partially account for the lower life expectancy in the United States relative to peer nations (8, 9).

Neighborhood factors have been identified as contributors to such conditions. In past studies, investigators have drawn links between low neighborhood socioeconomic status and obesity (10-12), hypertension (13, 14), diabetes (15), and other associated risk factors, including poor diet (16-18),

infrequent exercise (19, 20), and sleep deficiencies (21). In most such studies, researchers relied on self-reported data from respondents, which may lead to bias. In response, some have used biomarkers to demonstrate positive associations of low neighborhood socioeconomic status with high blood pressure (22, 23), high glycosylated hemoglobin levels (24, 25), low serum carotenoid concentrations (26), and high triglyceride levels (23).

Because of a lack of longitudinal studies, it remains unclear how exiting, entering, or continuously residing in poor and nonpoor communities is associated with cardiometabolic health. This omission is less problematic for samples with older participants because residential mobility declines with age (27). However, this is not the case among younger people. Residential mobility peaks in early adulthood, with most adolescents transitioning from their parents' homes to independent residences as they enter adulthood. During this process, many will experience discontinuities in their residential environments. For instance, Sharkey (28) found that 60% of white adolescents and 33% of black adolescents from neighborhoods in the poorest quintile of American communities moved to less disadvantaged neighborhoods by young adulthood. Other studies provide similar results (29), although for many, neighborhood exposures are reproduced—not interrupted—during the transition to adulthood.

Three theoretical perspectives can be used to describe the health effects that may be associated with upward, downward, or lateral residential mobility. The sensitive periods model holds that negative health exposures have greater effects when they occur during developmentally vulnerable stages in life and affect later-life outcomes irrespective of future exposures (30). This model has been used to explain associations between early-life disadvantage and future outcomes, including risky health behaviors, impaired immune function, and decreased adult life expectancy (11, 31-33). Under this view, adolescents from poor neighborhoods are expected to have a higher risk of cardiometabolic disorders in adulthood than are those from nonpoor neighborhoods, irrespective of residential circumstances in adulthood.

The cumulative disadvantage theory (34–36) holds that harmful exposures at multiple stages in life operate jointly to produce additive effects on health. It draws support from studies in which gradients in the relationship between time exposed to poverty and risk for negative health outcomes were identified (37, 38). Accordingly, cardiometabolic health is expected to be worst among those who consistently live in poor neighborhoods, best among those who never live in poor neighborhoods, and somewhere in between for those who enter or exit neighborhood poverty.

A third perspective—the social mobility model (39)—holds that effects from earlier-life conditions can be modified by later circumstances. Consistent with this view, empirical evidence has shown that the risk of acute conditions, including myocardial infarction, is lower among the upwardly mobile than among the downwardly mobile (40). Accordingly, it is expected that youths from poor neighborhoods who move to nonpoor neighborhoods in adulthood will present with better cardiometabolic health than will their counterparts who move from nonpoor to poor communities.

Health studies in which repeated measures of neighborhoodlevel exposures are used are needed to test these models, although such studies are rare (41). The Moving to Opportunity (MTO) residential relocation experiment, which was designed to fill this void, has provided some evidence of the health benefits of upward residential mobility. For instance, adult women who were randomized to more affluent neighborhoods had a lower risk of severe obesity and diabetes than did controls who remained in low-income communities (42). The longterm benefits of upward residential mobility for adolescents were less clear, with higher adult educational attainment and earnings for treated versus controls (43) but poorer mental health and behavioral problems among adolescent males randomized to nonpoor neighborhoods, especially those with pretreatment health vulnerabilities or exposure to criminal victimization (44, 45).

A wave of longitudinal studies using nonexperimental population-based data followed, revealing associations between greater exposure to neighborhood disadvantage and higher risk of poor self-rated health (46), substance use (47, 48), obesity (49–51), diabetes (51), and mortality (52). Strengths of some such studies included adjustment for residential self-selection bias through inverse-probability of treatment weighting, whereas weaknesses included a focus on either baseline neighborhood poverty exposure or cumulative measures based on the average neighborhood poverty rate across multiple observations. This makes it unclear how entering or exiting disadvantaged neighborhoods—a common event during the transition to adulthood—is associated with health relative to continuous residence in poor or nonpoor communities.

Building on previous work, we used data from the US Census and multiple waves of the National Longitudinal Study of Adolescent to Adult Health (Add Health) to investigate 2 questions. 1) How is exiting, entering, or consistently living in poor or nonpoor neighborhoods associated with multiple cardiometabolic biomarkers? 2) Are these patterns robust to adjustment for residential selection bias using treatment weights derived from coarsened exact matching?

METHODS

Sample

Data were drawn from the restricted-use Add Health data set and the US Census. Add Health is an ongoing, nationally representative, school-based survey of adolescents in seventh through twelfth grades from 132 high schools and middle schools (53). In 1994, Add Health administered in-school questionnaires to students selected through a stratified random sample of all high schools in the United States (n = 90,118). A subsample of these students participated in home-based interviews between 1994 and 1995 (n = 20,745). Except for graduating high school seniors, baseline interviewees were re-interviewed in a second wave of data collection in 1996 (n = 14,738). Third and fourth waves of data were collected in 2001-2002 and 2008, respectively, from all wave-1 participants who underwent home interviews. A total of 12,284 respondents were interviewed in waves 1, 3, and 4. We retained respondents with GPS- or address-matched records in order to derive neighborhood-level measures, yielding a final sample of 11,767.

Contextual data appended to Add Health (54) were utilized to derive measures of neighborhood poverty in wave 1 (1994–1995) using 1990 Census data and in wave 4 using 2005–2009 American Community Survey estimates (55). Census tracts were used to approximate neighborhood boundaries, although supplementary models in which we used block groups produced similar results.

Although the data were mostly complete, 23.5% and 17.8% of the sample was missing household poverty data from waves 1 and 4, respectively. To address this, we used Stata's ICE program (StataCorp LP, College Station, Texas) to multiply impute missing values for all of variables (56). The resulting 5 sets of complete data were combined to

adjust for variance within and between imputed samples in order to calculate standard errors and coefficients (57).

Measures

Multiple dependent variables derived from the interview conducted when participants were young adults (i.e., wave 4) were assessed. Obesity was determined by calculating body mass index (dividing measured weight in kilograms by height in meters squared) and classifying those with a body mass index that met or exceeded a value of 30 as obese. High waist circumference was a dichotomous measure, with participants receiving a score of 1 if the measured circumference of the area of the midsection between the lowest rib and the superior border of the iliac crest at end expiration was greater than or equal to 102 cm for males or 88 cm for females. Diabetes was determined via measured glycosylated hemoglobin values $(\geq 6.5\%)$ or if the participant self-reported having diabetes or using antidiabetic medications. Systolic blood pressure and diastolic blood pressure were measured in millimeters of mercury by Add Health interviewers up to 3 times, with multiple measurements being used among 96% of the sample to construct average values. Baroreflex sensitivity-a marker for cardiovascular disease (58)-was measured in participants with nonmissing sampling weights and all 3 measures of systolic blood pressure and pulse rate (beats per minutes). Baroreflex sensitivity was recorded as ms/mm Hg, and details on baroreflex calculation are provided in Add Health documentation (59). Triglyceride, high-density lipoprotein, and lowdensity lipoprotein cholesterol levels were measured from blood spots collected via finger prick and reported in deciles by Add Health. Lipoproteins are protein-containing particles that transport lipids-including triglyceridesthroughout the blood stream, and their concentrations are associated with cardiovascular disease (60).

Neighborhood poverty change

Measures of neighborhood poverty change from wave 1 to wave 4 were constructed over the course of several steps. First, binary indicators of neighborhood poverty were based on the Census-derived percentages of individuals with incomes below the federal poverty line within respondents' wave-1 and wave-4 tracts. Research has shown nonlinearities in the relationship between neighborhood poverty and resident well-being, with a rate of 20% or greater being especially relevant to outcomes including high school dropout, exposure to community violence, and premature mortality (52, 61). Accordingly, we classified respondents in tracts with poverty rates of 20% or more as living in poor neighborhoods; all other respondents were classified as living in nonpoor neighborhoods.

Binary indicators were combined into a 4-category measure that indicated change in neighborhood poverty from wave 1 to wave 4: consistently lived in nonpoor neighborhoods (score of 0), moved from a nonpoor neighborhood in wave 1 to a poor neighborhood at wave 4 (score of 1), moved from a poor neighborhood in wave 1 to a nonpoor neighborhood at wave 4 (score of 2), or consistently lived in poor neighborhoods at each wave (score of 3). For simplicity, we refer to the middle 2 categories as entered and exited poor neighborhoods, respectively. Control variables included baseline measures of respondent age (in years), sex, race/ethnicity, highest educational attainment of the respondent's parents (less than high school (0), high school diploma or equivalent (1), or 4-year college degree or higher (2)), and duration of residence at wave 1 address (1 if \geq 5 years (i.e., the time of the 1990 Census), 0 otherwise).

Analysis

Descriptive statistics of the study sample are presented in Table 1. We fit dichotomous outcomes (i.e., obesity, high waist circumference, and diabetes) to logistic models and linear outcomes (i.e., blood pressure, baroreflex sensitivity, and blood lipid levels) to ordinary least squares models. Two models are shown for each outcome: an unadjusted model that included only the neighborhood poverty typology and a minimally adjusted model that included neighborhood poverty and baseline controls (Tables 2 and 3).

A second set of analyses were based on a subset of the sample matched on the neighborhood poverty typology using coarsened exact matching (CEM) (61). CEM is a matching method to reduce covariate imbalance between "treated" and "untreated" case patients in nonexperimental data. Matching with CEM is preferable for this study because it accommodates multicategory treatments, automatically restricts the sample to common support, and yields causal estimates with lower variance and bias than alternative methods (62, 63).

Because of our interest in multiple categories of neighborhood poverty exposures, we began by selecting a "prime" treatment (64), which in this case was the most severe: consistent residence in poor neighborhoods. All other neighborhood poverty exposures were treated as control groups by CEM. Next, we identified variables with theoretical importance to the probability of having received treatment. We used conceptual models of locational attainment-the process of attaining residence in neighborhoods of higher, lower, or equal socioeconomic standing as one's neighborhood of origin-which emphasize socioeconomic and demographic factors (29, 65). Variables based upon this framework are shown in Appendix Table 1. Measures that required further coarsening to optimize matching included income-to-poverty ratios in waves 1 and 4 (both coarsened into bins of 0.00-0.99, 1.00-1.99, 2.00-2.99, >3.00) and distance in kilometers separating respondents' wave-1 and wave-4 addresses (0.00–119.99 km or \geq 120.00 km).

Using the "cem" command in Stata, version 14 (66), treated and control groups were exactly matched across strata identified by temporarily coarsened variables, creating matched pairs of treated and control cases within strata. Strata with unmatched cases were removed (n = 2,751). For cases from matched strata (n = 9,727), weights were calculated based on the relative proportion of matched controls and treated within specific strata. All "prime" treatment cases received weights equal to "1," whereas controls received stratum-specific weights equal to the ratio of control observations to prime observations. Selection-adjusted models were then weighted to adjust for residential selection bias and other design features of Add Health. Using the language of CEM, we refer to those

Variable	Proportion (SE)	Mean (SE)
Neighborhood poverty exposure		
Never lived in poor neighborhoods	0.62 (0.01)	
Entered neighborhood poverty	0.13 (0.01)	
Exited neighborhood poverty	0.11 (0.01)	
Consistently lived in poor neighborhoods	0.14 (0.01)	
Baseline controls		
Age, years		15.40 (0.03)
Female	0.51 (0.01)	
Race/ethnicity		
White, non-Hispanic	0.67 (0.01)	
Black, non-Hispanic	0.15 (0.01)	
Hispanic	0.12 (0.01)	
Other race	0.07 (0.00)	
Parental educational level		
Less than high school	0.12 (0.01)	
High school diploma/equivalent	0.56 (0.01)	
Four-year college degree	0.32 (0.01)	
Resided at wave-1 address for \geq 5 years	0.56 (0.01)	
Outcome measures		
Systolic blood pressure, mm Hg		124.96 (0.18)
Diastolic blood pressure, mm Hg		79.36 (0.14)
Baroreflex sensitivity, ms/mm Hg		0.69 (0.03)
High-density lipoprotein cholesterol, mg/dL		5.57 (0.04)
Low-density lipoprotein cholesterol, mg/dL		5.45 (0.04)
Triglyceride level, mg/dL		5.57 (0.04)
Obese	0.37 (0.01)	
High waist circumference	0.51 (0.01)	
Diabetic	0.06 (0.00)	

Table 1. Description of the Study Sample, National Longitudinal Study of Adolescent to Adult Health (n = 11,767),1995–2008

Abbreviation: SE, standard error.

who entered or exited neighborhood poverty as having "abbreviated exposure" and those who never lived in poor neighborhoods as having "no exposure." Weight construction for each group used the following:

$$w_s^{AE} = \frac{m_{C_{Abbreviated Exposure}}}{m_T} \cdot \frac{m_T^s}{m_{C_{Abbreviated Exposure}}^s} \tag{1}$$

and

$$w_s^{NE} = \frac{m_{C_{No Exposure}}}{m_T} \cdot \frac{m_T^s}{m_{C_{No Exposure}}^s},\tag{2}$$

where m_C and m_T represent the matched observations in the control and treatment groups, respectively, and m_T^S and m_C^S represent observations within a specific stratum. w_S^{AE} and w_S^{NE} are stratum-specific weights for groups with abbreviated exposure to neighborhood poverty and no exposure, respectively.

Covariate imbalance between treated and control groups was measured before and after matching using the L_1 statistic (62) given below:

$$L_1(f,g) = \sum_{l_1...l_k} |f_{l_1...l_k} - g_{l_1...l_k}|, \qquad (3)$$

where *f* and *g* are relative frequencies of prime and control cases, respectively, obtained by discretizing and crosstabulating the variables used for coarsening; $l_1 ldots l_k$ are the number of bins used for discretizing coarsening variables. L_1 ranges from 0 to 1, where 0 constitutes perfect balance between "prime" and control cases and 1 represents perfect imbalance. Postmatching results indicated considerable reduction of imbalance across the coarsening variables (see Appendix Table 1).

Because Add Health respondents lived in different tracts over time, the data were not nested in a conventional way, and wave-4 tract clustering was low. Thus, neighborhood

						Neighborhood	Poverty Ex	posure					
0	Con	Consistently Lived in Poor Neighborhoods				Entered Neighborhood Poverty				Exited Neighborhood Poverty			
Outcome Measure	Model 1 ^a		Model 2 ^b		Model 1 ^a		Model 2 ^b		Model 1 ^a		Model 2 ^b		
	b°	95% CI	b°	95% CI	b ^c	95% CI	bc	95% CI	b°	95% CI	bc	95% CI	
Versus Never Lived in Poor Neighborhoods													
Systolic BP	2.53	1.42, 3.65	1.77	0.53, 3.02	1.40	0.36, 2.44	1.08	0.07, 2.10	0.17	-0.95, 1.28	-0.07	-1.15, 1.01	
Diastolic BP	1.96	1.13, 2.79	1.73	0.82, 2.64	1.16	0.38, 1.94	1.08	0.32, 1.85	0.80	-0.01, 1.61	0.76	-0.06, 1.57	
Baroreflex sensitivity	0.10	-0.07, 0.27	0.10	-0.08, 0.28	0.15	-0.03, 0.33	0.14	-0.04, 0.33	0.07	-0.09, 0.23	0.07	-0.11, 0.24	
HDL	-0.23	-0.48, 0.02	-0.09	-0.36, 0.19	-0.17	-0.42, 0.07	-0.13	-0.37, 0.12	-0.27	-0.52, -0.03	-0.19	-0.44, 0.06	
LDL	-0.23	-0.48, 0.03	-0.07	-0.35, 0.22	-0.14	-0.38, 0.09	-0.10	-0.34, 0.14	-0.12	-0.40, 0.15	-0.02	-0.30, 0.27	
Triglycerides	-0.26	-0.54, 0.02	0.09	-0.17, 0.35	-0.23	-0.45, 0.01	-0.08	-0.32, 0.15	-0.14	-0.38, 0.10	0.09	-0.16, 0.34	
Varsus Consistently Lived in Poor Neighborhoods													
Systolic BP				10,000 0	_1 14	-2 53 0 26	_0 69	-2 14 0 76	-2.37	-3.82 -0.92	_1 84	-3 24 -0 44	
					_0.80	_1 83 0 22	-0.65	_1 71 0 41	_1 17	-2.23 -0.10	_0.98	-2.02.0.06	
Baroreflex sensitivity					0.05	-0.18.0.27	0.03	-0.18.0.27	-0.03	-0.25, 0.18	-0.00	-0.25,0.18	
HDI					0.06	-0.25, 0.37	-0.04	-0.36, 0.28	_0.00	-0.35, 0.27	_0.04	-0.42 0.21	
					0.00	_0.22, 0.39	_0.04	-0.36, 0.28	0.11	_0.22_0.43	0.10	-0.27, 0.37	
Trialvoerides					0.00	-0.29, 0.35	-0.04 -0.18	-0.46 0.11	0.11	-0.22, 0.46	-0.01	-0.29,0.29	
Inglycenaes					0.00	0.20, 0.00	0.10	0.40, 0.11	0.12	0.22, 0.40	0.01	0.20, 0.20	
				Ver	sus Entere	d Neighborhood	Poverty						
Systolic BP									-1.23	-2.64, -0.36	-1.15	-2.48, 0.18	
Diastolic BP									-0.36	-1.39, 0.66	-0.33	-1.33, 0.67	
Baroreflex sensitivity									-0.08	-0.31, 0.15	-0.08	-0.31, 0.15	
HDL									-0.10	-0.41, 0.20	-0.06	-0.36, 0.24	
LDL									0.02	-0.29, 0.33	0.09	-0.22, 0.40	
Triglycerides									0.09	-0.20, 0.38	0.18	-0.10, 0.46	

 Table 2.
 Results of Ordinary Least Squares Regression for Blood Pressure, Baroreflex Sensitivity, and Blood Lipids, National Longitudinal Study of Adolescent to Adult Health (n = 11,767), 1995–2008

Abbreviations: BP, blood pressure; CI, confidence interval; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

^a Unadjusted model.

^b Adjusted for sex, race/ethnicity, and wave-1 age, parental educational level, and duration of residence at current address.

^c Ordinary least squares coefficients.

					Ne	eighborhood P	overty E	xposure					
Outcome Measure		Consistently Lived in Poor Neighborhoods			Entered Neighborhood Poverty				Exited Neighborhood Poverty				
	Ν	/lodel 1ª	N	Model 2 ^b		Model 1 ^a		Model 2 ^b		Model 1 ^a		Model 2 ^b	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	
Versus Never Lived in Poor Neighborhoods													
Obese	1.82	1.54, 2.15	1.43	1.18, 1.74	1.13	0.96, 1.32	1.07	0.90, 1.25	1.63	1.39, 1.92	1.39	1.17, 1.65	
High waist circumference	1.45	1.22, 1.71	1.23	0.99, 1.49	1.06	0.91, 1.24	1.05	0.89, 1.24	1.32	1.13, 1.55	1.14	0.95, 1.36	
Diabetic	2.35	1.78, 3.09	1.28	0.93, 1.76	1.74	1.29, 2.34	1.40	1.03, 1.90	1.73	1.29, 2.33	1.12	0.82, 1.53	
			V	ersus Consis	tently L	ived in Poor I	Veighbo	orhoods					
Obese					0.62	0.50, 0.76	0.74	0.60, 0.93	0.90	0.73, 1.11	0.97	0.77, 1.21	
High waist circumference					0.74	0.60, 0.91	0.87	0.68, 1.10	0.91	0.74, 1.13	0.94	0.74, 1.18	
Diabetic					0.74	0.52, 1.05	1.09	0.76, 1.57	0.74	0.52, 1.04	0.88	0.62, 1.25	
				Versus E	ntered l	Neighborhoo	d Pove	rty					
Obese									1.45	1.18, 1.78	1.30	1.05, 1.61	
High waist circumference									1.24	1.02, 1.51	1.08	0.87, 1.34	
Diabetic									1.00	0.69, 1.44	0.80	0.56, 1.16	

Table 3. Results of Logistic Regression Analyses for Obesity, Waist Circumference, and Diabetic Condition, National Longitudinal Study of Adolescent to Adult Health (n = 11,767), 1995–2008

Abbreviations: CI, confidence interval; OR, odds ratio.

^a Unadjusted model.

^b Adjusted for sex, race/ethnicity, and wave-1 age, parental educational level, and duration of residence at current address.

poverty was modeled as an individual-level exposure. For all models, adjustments were made to correct for the design effects of Add Health and standard errors for within-tract homogeneity by clustering on primary (wave-1 tract) and secondary (wave-4 tract) sampling units.

RESULTS

Table 2 presents ordinary least squares regression results for linear outcomes. Results from adjusted ordinary least squares models indicated that blood pressure was higher among those who consistently lived in poor neighborhoods (for systolic blood pressure, b = 1.77, P < 0.01; for diastolic blood pressure, b = 1.73, P < 0.001) or entered neighborhood poverty (for systolic blood pressure, b = 1.08, P < 0.05; for diastolic blood pressure, b = 1.09, P < 0.01) relative to those who never lived in poor neighborhoods,. Those who exited poor neighborhoods also had significantly lower average systolic blood pressure net of controls than did those who consistently lived in poor neighborhoods (b = -1.84, P < 0.01). Models adjusted for baroreflex sensitivity and lipid levels did not yield significant differences across the neighborhood poverty typology.

Table 3 is similarly divided into 3 panels. Results adjusted for obesity showed that the odds of being obese were higher for those who consistently lived in poor neighborhoods (odds ratio (OR) = 1.43, 95% confidence intervals (CI): 1.18, 1.74) and those who exited neighborhood poverty (OR = 1.39, 95% CI: 1.17, 1.65) than for those who never lived in poor neighborhoods. Obesity was less common among those who entered poor neighborhoods than among those who consistently lived in poor neighborhoods (OR = 0.74, 95% CI: 0.60, 0.93). Finally, those who exited versus entered neighborhood poverty had higher odds of being obese (OR = 1.30, 95% CI: 1.05, 1.61). Differences in waist circumference were null net of controls. Adjusted models showed that those who entered versus never lived in poor neighborhoods were at higher risk of diabetes (OR = 1.40, 95% CI: 1.03, 1.90).

Table 4 presents results from models based on the matched sample for select outcomes (i.e., those significantly associated with neighborhood poverty in Tables 2 and 3). Following prior research, we included interaction terms between sex and the neighborhood poverty typology in supplementary analyses (see Appendix Table 2). Two sets of estimates are presented: those adjusted for baseline controls only (model 1) and those adjusted for selection with coarsened matched weights (model 2). Results from supplementary models adjusted with coarsening weights, as well as all coarsened matching variables and controls, were similar to those described here. Results for obesity indicated that those who consistently lived in poor neighborhoods were more likely to be obese than were those who never lived in poor neighborhoods (OR = 1.46, 95% CI: 1.10,1.93). This translated into 46% higher odds of being obese for those who consistently versus never lived in low-income neighborhoods ($[1.46 - 1.00] \times 100 \approx 46\%$).

Net of adjustments for selection, those who consistently lived in poor neighborhoods also had higher blood pressure **Table 4.** Results for Select Outcomes With and Without Coarsened Exact Weights^a, National Longitudinal Study of Adolescent to Adult Health (n = 9,727), 1995–2008

	Consistently Lived in Poor Neighborhoods								
Outcome Measure		Model 1 ^b		Model 2 ^c					
	OR	b ^d	95% CI	OR	b ^d	95% CI			
Versus never lived in poor neighborhoods									
Obese	1.43		1.16, 1.77	1.46		1.10, 1.93			
Diabetic	1.20		0.85, 1.70	0.99		0.63, 1.57			
Systolic BP		1.65	0.32, 2.97		2.04	0.36, 3.71			
Diastolic BP		1.72	0.76, 2.68		1.96	0.79, 3.12			
			Entered Neighbo	orhood Pover	ty				
		Model 1 ^t)		Model 2 ⁶	;			
	OR	b ^d	95% CI	OR	b ^d	95% CI			
Versus never lived in poor neighborhoods									
Obese	0.99		0.82, 1.20	1.26		0.88, 1.81			
Diabetic	1.19		0.85, 1.66	0.80		0.44, 1.45			
Systolic BP		0.73	-0.45, 1.90		2.28	-0.13, 4.68			
Diastolic BP		1.04	0.16, 1.92		1.85	-0.30, 4.01			
Versus consistently lived in poor neighborhoods									
Obese	0.69		0.54, 0.89	0.86		0.60, 1.24			
Diabetic	0.99		0.67, 1.46	0.80		0.47, 1.37			
Systolic BP		-0.92	-2.50, 0.66		0.24	-2.14, 2.63			
Diastolic BP		-0.68	-1.85, 0.49		-0.11	-2.19, 1.98			
	Exited Neighborhood Poverty								
		Model 1 ^t)	Model 2 ^c					
	OR	b ^d	95% CI	OR	b ^d	95% CI			
Versus never lived in poor neighborhoods									
Obese	1.40		1.14, 1.72	1.07		0.73, 1.58			
Diabetic	1.07		0.76, 1.53	0.90		0.50, 1.63			
Systolic BP		0.31	-0.94, 1.55		0.91	-1.21, 3.03			
Diastolic BP		1.24	0.31, 2.17		2.03	0.59, 3.48			
Versus consistently lived in poor neighborhoods									
Obese	0.98		0.76, 1.25	0.73		0.50, 1.07			
Diabetic	0.89		0.61, 1.31	0.90		0.53, 1.53			
Systolic BP		-1.34	-2.85, 0.17		-1.12	-3.04, 0.79			
Diastolic BP		-0.48	-1.60, 0.64		0.07	-1.28, 1.43			
Versus entered neighborhood poverty									
Obese	1.42		1.10, 1.81	0.85		0.54, 1.33			
Diabetic	0.90		0.60, 1.36	1.12		0.58, 2.16			
Systolic BP		-0.42	-1.97, 1.14		-1.36	-4.15, 1.42			
Diastolic BP		0.20	-0.96, 1.36		0.18	-2.10, 2.46			

Abbreviations: BP, blood pressure; CI, confidence interval; OR, odds ratio.

^a All models were restricted to the matched subsample within the region of common support.

^b Model 1 included normal sampling weights with adjustments for controls (sex, race/ethnicity, and wave-1 age, parental educational level, and duration of residence at current address).

^c Model 2 included coarsened exact and normal sampling weights with adjustments for confounders.

^d Ordinary least squares coefficients.

than did those who never lived in poor neighborhoods (for systolic blood pressure, b = 2.04, P < 0.05; for diastolic blood pressure, b = 1.96, P < 0.001). Diastolic blood pressure was similarly higher among those who exited versus never lived in poor neighborhoods (b = 2.03, P < 0.01). In supplementary models, we estimated a logistic model for a dichotomous indicator of hypertension that was equal to 1 for combined blood pressure of 140/90 mm Hg or higher (or 0 otherwise). Selection-adjusted results indicated higher odds of hypertension among those who consistently versus never lived in poor neighborhoods (OR = 1.52, 95% CI: 1.11, 2.08). This translated into 52% greater odds of being hypertensive for those who consistently lived in poor neighborhoods.

To illustrate these findings more plainly, we converted the selection-adjusted logistic results for obesity and hypertension into marginal mean predicted probabilities for all neighborhood poverty exposures (holding controls at mean values). The mean probabilities of being obese were 38.2% for those who never lived in poor neighborhoods, 45.5% for those who entered poor neighborhoods, 40.9% for those who exited poor neighborhoods, and 48.1% for those who consistently lived in poor neighborhoods. For hypertension, mean probabilities were 17.4% for those who never lived in poor neighborhoods, 20.6% for both those who entered and those who exited neighborhood poverty, and 23.4% for those who consistently lived in poor neighborhoods.

Conclusion

Few studies have explored how entering, exiting, or remaining in low-income communities shapes the risk of poor cardiometabolic health. We examined this by using biologically derived health measures and covariate imbalance-reducing methods. Adjusting for confounders and residential selection bias using CEM, we found that those who consistently lived in poor neighborhoods had roughly 46% higher odds of being obese and 52% greater odds of being hypertensive in young adulthood than did those with no exposure to neighborhood poverty. Further, those who exited versus never lived in neighborhood poverty had significantly higher diastolic blood pressure, which is a stronger predictor of early mortality for young adults than is systolic blood pressure (67). Minimally adjusted models revealed more significant patterns, but these were not robust to adjustment for residential selection bias.

It is useful to contrast our estimates to those from MTO participants who were children at baseline. As part of a long-term evaluation of program effects, child participants of MTO were re-evaluated in 2007, and their health status were assessed using self-reports and objective (measured) indicators (68). In most respects, our findings align with the modest effects of upward residential mobility among MTO youth. For instance, 20.7% of MTO youth randomized to nonpoor neighborhoods were obese between the ages of 10-19 years when reinterviewed in 2007 (vs. 22.9% of controls). These differences were not statistically significant, similar to our selectionadjusted findings regarding obesity differences between those exiting versus consistently living in poor neighborhoods. Selfreported health measures among MTO youth reveal similar null differences between treatment and control groups. Although few health gains were identified among treated

MTO youth, results indicated that there were some health benefits for treated MTO adults, which suggests that the health benefits of upward residential mobility may be realized deeper into the life course.

Studies have also used nonexperimental data to examine associations between point-in-time measures of early-life neighborhood disadvantage exposure and later-life cardiometabolic outcomes. Using Add Health data, Nicholson and Browning (12) found higher risk of obesity among women aged 18-26 years who lived in disadvantaged neighborhoods as adolescents (OR = 1.22, 95% CI: 1.11, 1.35). Also using Add Health, Wickrama et al. (69) find a positive association between wave-1 school-district disadvantage and higher blood pressure (for systolic blood pressure, b = 3.2, P < 0.05; for diastolic blood pressure, b = 3.63, P < 0.001). Estimates from the Panel Study of Income Dynamics indicated that adolescents who lived in neighborhoods with poverty rates between 10%-20% had significantly higher risks for self-reported diabetes (hazard ratio = 1.70, 95% CI: 1.07, 2.70) by age 50 years than did those from nonpoor neighborhoods (70).

Considerable differences between the designs of prior investigations and our own may lead to dissimilar findings; yet, we contend that some differences constitute advantages of the current study. First, we relied on objective indicators of pathology, including biometric, anthropometric, and biologically derived measures. Second, we utilized a typology of neighborhood poverty exposures during adolescence and young adulthood. Third, we applied matching methods to make our sample more homogenous across levels of neighborhood poverty exposure.

The present study is not without limitations. First, like other matching methods, CEM is sensitive to the omission of variables associated with the locational attainment process. Second, our study concerned a limited stage in the life course. Without additional observations, it is unclear whether our findings persist into formal adulthood. Third, the mechanisms that link high-risk neighborhood exposures to cardiometabolic health are unclear. Fourth, some of the estimates we presented were bound by wide confidence intervals and should be interpreted cautiously. Finally, without baseline outcome measures, reverse causation remains an alternative interpretation of our findings.

Notwithstanding limitations, our study lends support to the cumulative disadvantage model. We found a general gradient of risk, with obesity and hypertension most common among those locked in poor neighborhoods, least common among those who never lived in poor neighborhoods, and somewhere in between among those who entered or exited neighborhood poverty. The intermediate obesity and hypertension risks associated with neighborhood poverty entry and exit did not differ significantly relative to other exposures. Thus, research using repeated neighborhood measures over longer periods is needed to further test the cumulative disadvantage model.

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(Appendix follows)

APPENDIX

Appendix Table 1. Covariate Imbalance Between Treated and Untreated Case Patients^a Before and After Matching, National Longitudinal Study of Adolescent to Adult Health, 1995–2008

Coorcoring Veriable	Befor	e Matching	After Matching			
Coarsening variable	L ₁ Statistic ^b	Mean Difference ^c	L ₁ Statistic ^b	Mean Difference ^c		
Wave-1 variables						
Income-to-poverty ratio ^d	0.33	-1.54	0.06	-0.20		
Raised in a single-parent family	0.17	0.17	0.00	0.00		
Wave-3 variables						
Educational attainment ^e	0.19	-0.39	0.00	0.00		
Lives with a child dependent	0.13	0.13	0.00	0.00		
Lives with a spouse ^f	0.01	-0.01				
Lives in a poor neighborhood	0.49	0.49	0.01	0.00		
Wave-4 variables						
Income-to-poverty ratio ^d	0.31	-1.62	0.06	-0.18		
Has completed a college degree	0.20	-0.02	0.00	0.00		
Lives with a child dependent ^f	0.11	0.11				
Lives with a spouse ^f	0.11	-0.11				
Euclidean distance (in km) between wave-1 and wave-4 addresses	0.24	-224.99	0.03	-31.92		

^a Treated case patients (1,581 matched, 94 unmatched) were those who consistently lived in poor neighborhoods, whereas untreated case patients (8,146 matched, 2,657 unmatched) comprised those in all other neighborhood poverty exposure categories.

^b Pre- and postmatching covariate imbalance.

^c Mean difference in covariate value between the treated and controls groups.

^d Measured as the ratio of household income to the official poverty line for household size.

^e Included categories for less than high school (0), high school diploma or equivalent (1), currently attending a 4-year university (2), and 4-year college degree or more (3).

^f Variables showed low imbalance before matching and were not used for matching.

Appendix Table 2. Selection-Adjusted Interactions of Sex and Neighborhood Poverty Exposure, National Longitudinal Study of Adolescent to Adult Health (n = 9,727), 1995–2008^a

Exposure		Obese		Diabetic		/stolic BP	Diastolic BP	
		95% CI	OR	95% CI	bb	95% CI	b ^b	95% CI
Consistently lived in poor neighborhoods	1.51	1.06, 2.15	1.14	0.61, 2.14	1.71	-0.84, 4.27	1.98	0.23, 3.73
Entered neighborhood poverty	1.03	0.57, 1.86	0.76	0.34, 1.67	0.79	-3.14, 4.71	1.41	-2.16, 4.98
Exited neighborhood poverty	1.13	0.59, 2.16	1.28	0.51, 3.19	0.39	-2.45, 3.22	1.67	-0.18, 3.51
Female	1.59	1.12, 2.26	1.57	0.84, 2.93	-10.39	-12.48, -8.31	-5.07	-6.59, -3.56
$\label{eq:Female} Female \times consistently lived in poor neighborhoods$	0.94	0.60, 1.48	0.79	0.35, 1.77	0.71	-2.18, 3.59	-0.03	-2.24, 2.19
Female \times entered neighborhood poverty	1.43	0.68, 2.99	1.05	0.35, 3.14	2.74	-1.93, 7.41	0.82	-3.23, 4.87
Female \times exited neighborhood poverty	0.92	0.41, 2.06	0.53	0.17, 1.65	1.11	-2.92, 5.15	0.73	-2.06, 3.52

Abbreviations: BP, blood pressure; CI, confidence interval; OR, odds ratio.

^a In all models, "never lived in poor neighborhoods" served as the reference neighborhood poverty exposure, and coarsened exact and normal sampling weights were applied with adjustments for sex, race/ethnicity, and wave-1 age, parental educational level, and duration of residence at current address.

^b Ordinary least squares coefficients.