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Childhood Adversity, Adult Neighborhood Context, and Cumulative Biological Risk for Chronic Diseases in Adulthood

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Abstract

Objective—We examined the association between childhood adversity and cumulative biological risk for a variety of chronic diseases in adulthood, and whether this association varied by neighborhood affluence.

Methods—Data were drawn from the Chicago Community Adult Health Study (2001-2003), a cross-sectional probability sample which included interviews and blood collection (*n*=550 adults). A childhood adversity score was calculated from eight items. Neighborhood affluence was defined using Census data. An index to reflect cumulative biological risk was constructed as a count of eight biomarkers above clinically-established thresholds, including systolic and diastolic blood pressure, resting heart rate, C-reactive protein, waist circumference, hemoglobin A1c, and total and high density lipoprotein cholesterol. Generalized linear models with a Poisson link function were used to estimate incident rate ratios (IRRs).

Results—A one standard-deviation increase in the childhood adversity score was associated with a 9% increase in cumulative biological risk, after adjustment for demographic and behavioral characteristics (IRR=1.09, 95% confidence interval (CI)=1.02, 1.17). This association was modified by neighborhood affluence (IRR=0.92, 95% CI=0.86, 0.99). Stratified models indicated that childhood adversity was associated with elevated cumulative biological risk only among individuals who resided in low affluence (bottom tertile) neighborhoods (IRR=1.16, 95% CI=1.05, 1.28); there was no association in high affluence (top tertile) neighborhoods (IRR=0.97, 95% CI=0.83, 1.14).

Conclusions—Childhood adversity is associated with elevated cumulative biological risk in adulthood, and neighborhood affluence may buffer this association. Results demonstrate the importance of neighborhood characteristics for associations between childhood adversity and disease risk, even after accounting for adult socioeconomic status.

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Keywords

childhood adversity; cumulative biological risk; allostatic load; neighborhoods; social environment

Adverse experiences in childhood, such as poverty or abuse, can influence physical and mental health across the life course (1, 2), including cardiovascular (3, 4), metabolic (5, 6), and immune (7, 8) function. Researchers have now advanced beyond describing the main effects of childhood adversity on health outcomes to exploring underlying social and physiological pathways (9, 10), and contextual-level influences, such as the effects of early neighborhood context on later health (11-13). This work has also been extended to examine combinations of stressors at multiple points in the life course. Research in this area has been guided by "cumulative risk" models (14-16), which suggest that experiencing multiple stressors over the life course increases likelihood of disorder, "stress-sensitization" models (17-21), which theorize that childhood adversity may sensitize individuals to have enhanced or attenuated responses to subsequent stressors, and "buffering" models (22-26), which suggest that contextual attributes can protect individuals from the typical consequences of stressful experiences. To date, few studies have examined interactions between childhood adversity and later experiences in relation to physiological outcomes (27-29). The notion that social experiences in adulthood can moderate, or "buffer", the effects of childhood adversity on chronic disease risk has not been widely examined. In the current study, we evaluated whether the association between childhood adversity and cumulative biological risk for chronic diseases in adulthood is buffered by advantageous neighborhood conditions in adulthood.

The terms "allostatic load" or "cumulative biological risk" refer to summary measures that characterize functioning across cardiovascular, metabolic, immune, nervous, and hormonal systems (30, 31). McEwen and colleagues introduced this concept to describe the biological consequences of the body's attempts to adapt to external demands (e.g., chronic stressors), and how physiological dysfunction can spread across multiple systems and combine to elevate disease risk (32-35). Increasing research shows that having adverse risk factors across multiple biological systems strongly predicts morbidity and mortality (36). Additionally, some studies have shown that the cumulative total of physiological dysregulation across indicators can predict morbidity and mortality risks better than individual components (37, 38). Assessment of the biological effects of childhood adversity across multiple regulatory systems is valuable for research on the long-term health consequences of childhood adversity, given that childhood adversity has been linked to wide array of diseases which have multifactorial etiologies that involve dysregulation of numerous biological systems, including cardiovascular disease and diabetes. Several studies have examined childhood adversity in relation to cumulative biological risk in children and adolescents (39-42); however, very few studies have examined childhood adversity in relation to cumulative biological risk in adults (43, 44). Therefore, we have a limited understanding of social experiences that could modify the influence of childhood adversity on cumulative biological risk.

Neighborhood context in adulthood may be one factor that could influence the effect of childhood adversity on risk for chronic diseases. Considerable empirical research shows that neighborhood context has implications for health outcomes beyond individual- and family-level risk factors (12, 45-48). Recent studies suggest that *positive* neighborhood attributes may be particularly relevant for health (31, 48-53), and that the mere absence of neighborhood poverty or relative disadvantage does not guarantee that a neighborhood has health-protective attributes that are associated with affluence, such as health services or recreational spaces. For example, in the Chicago Community Adult Health Study (CCHAS), King and colleagues found that neighborhood affluence predicted lower cumulative biological risk, whereas neighborhood disadvantage was not associated with cumulative biological risk (31). Some research has shown that positive neighborhood attributes can buffer the negative physical (23, 24) or mental (24-26) health consequences of individual-level stressful experiences. Consistent with this research, it is plausible that residing in a relatively advantaged neighborhood environment in adulthood may protect individuals from the deleterious health consequences of childhood adversity. An affluent neighborhood context may attenuate the negative impact of childhood adversity on health through a number of health-promoting pathways, including direct and indirect access to important resources for maintaining health (i.e., groceries, parks, safe and friendly streets, community health clinics), strong social networks, and social capital.

The present study used data from the CCAHS to evaluate the association between childhood adversity and cumulative biological risk in adulthood, and to examine whether this relationship varied by adult neighborhood affluence. We hypothesized that childhood adversity would be associated with elevated cumulative biological risk in adulthood, and that the association between childhood adversity and cumulative biological risk would be less pronounced among individuals who lived in higher-affluence neighborhoods relative to individuals who lived in lower-affluence neighborhoods.

Sample

The CCAHS is a cross-sectional household probability sample of 3105 adults aged 18 years and over residing in Chicago, Illinois (March 2001 to March 2003). In-person interviews were completed with one individual per household. Participants were recruited from 343 neighborhood clusters that were initially defined by the Project on Human Development in Chicago Neighborhoods (PHDCN; overall response rate =71.8%) (54). The 343 clusters typically consisted of two census tracts (approximately 8000 people) and had physical borders that reflected socially meaningful divisions. On average, there were 9 respondents per neighborhood cluster (range: 1-21 respondents). Participants were oversampled from 80 neighborhood clusters, referred to as “focal neighborhoods”. The focal neighborhoods were a stratified random sample of the 343 neighborhood clusters (based on cross-classifications of race/ethnicity and socioeconomic status (SES)), designed to capture a socioeconomically- and racially/ethnically-heterogeneous representation of Chicago's neighborhoods (55). Within each focal neighborhood, dwelling units were enumerated and selected at random, followed by random selection of one household member (over the age of 18) per dwelling unit. Individuals who resided in 80 focal areas defined by the PHDCN were sampled at

twice the rate of participants elsewhere in the city, and were invited to provide blood and saliva samples.

A total of 1145 respondents lived in the 80 focus neighborhood clusters, and these individuals were asked to separately consent to a second visit by a trained phlebotomist. A total of 629 respondents provided blood samples (response rate = 55%). Older respondents were more likely than younger respondents to provide blood samples; however, after adjustment for age, there were no significant differences between individuals participating in the biomarker component of the study and the overall sample with regard to race/ethnicity, education, marital status, or functional limitations (56). Of the 629 respondents who provided blood samples, 550 yielded valid data for all eight biomarkers required for the cumulative biological risk score. In this subsample of respondents, there was a mean of 6.9 respondents per neighborhood cluster (range: 2 to 12). Weights were created to account for non-response and the unique sociodemographic composition of the 80 focal neighborhoods. Accordingly, the weighted sample matches the city of Chicago 2000 Census population estimates for age, sex, and race/ethnicity distributions. As described elsewhere (31), the subsample with valid biomarkers has similar sociodemographic characteristics in comparison to the entire subsample invited to provide blood samples (n=1145) and full study sample (n=3105). Original data collection was approved by the University of Michigan Behavioral Sciences and Health Sciences Institutional Review Boards; all subjects provided informed consent.

Methods

Measures

Childhood Adversity—Childhood adversity was assessed using an eight-item measure which asked respondents to reflect on their experiences before age 12 years (α=0.78). Each item was rated on a five-point scale. Participants were asked: how often their parents (1) made them feel loved; (2) physically held and comforted them; (3) physically threatened or abused them; (4) verbally threatened or abused them; (5) participated in activities in their school; (6) read to them; (7) how often they went to bed at night feeling hungry (*very often* to *never*); and, (8) how well off their family was when they were growing up (*quite well off* to *poor*). Factor analysis confirmed the presence of a single factor. We z-scored this measure to normalize the distribution and improve interpretation of the results.

Cumulative Biological Risk—We constructed an index of cumulative biological risk following prior research using this sample (31). For each subject, the index provided a count of the number of biomarkers above the clinically-defined criteria for “high risk”. The index included eight biomarkers: systolic blood pressure (≥140 mm Hg or higher) (57); diastolic blood pressure (≥90 mm Hg) (57); resting heart rate (≥90 beats per minute) (58); glycosylated/glycated hemoglobin (≥0.064) (59); C-reactive protein (CRP; ≥3 mg/dl) (60); total cholesterol (≥240 mg/dL) (61); high density lipoprotein (HDL; ≤40 mg/dL for men, ≤50 mg/dL for women)(62); and waist circumference (≥102 cm for men, and ≥88 cm for women) (63). We created an unweighted index by summing dichotomous variables for each of the 8 biomarkers, which parallels the approach used in many other studies of cumulative

biological risk (36). The components in this inventory included biological indicators of the cardiovascular (systolic and diastolic blood pressure and resting heart rate), metabolic (glycosylated/glycated hemoglobin, total cholesterol, HDL, and waist circumference) and immune (CRP) systems, and this assessment has substantial overlap with biomarkers used to construct indices of cumulative biological risk in other studies with different samples (36, 47, 64). Each of the biological indicators has been shown to be associated with chronic disease (65-71). Although there is substantial variation across studies with regard to how allostatic load is defined (36), we use the term "cumulative biological risk" (31) because the components of our measure are not identical to the most traditional assessments of allostatic load and reflect secondary outcomes rather than primary stress mediators (such as cortisol or catecholamines (32)).

Individual-level Demographic Characteristics—Respondents reported age, sex, education (less than high school, high school, some college, college degree or more), household income (less than $10,000, $10,000-29,999, $30,000-49,999, $50,000 or more, missing), race/ethnicity (Black, White, or Hispanic), and nativity status (i.e., U.S.-born and foreign-born). For our race/ethnicity variable, we stratified the Hispanic category by self-reported nativity based on evidence that this distinction has relevance for health outcomes in this sample (72). Of the 550 respondents, 13 individuals (2.4 percent) reported "other" race/ethnicity. The individuals who identified as "other" race were similar to Whites on a number of demographic characteristics; therefore, we combined the "other" category with Whites to maximize available data (72). An indicator variable for self-reported medication use was constructed to reflect current use of hypertension, arthritis, diabetes, or cholesterol medication.

Health Behaviors and Depressive Symptoms—Smoking was measured as current, previous, never, and alcohol consumption as none, 1-31 drinks per month, 32+ drinks per month. Physical activity was assessed using six questions about frequency, intensity, and duration of activities, derived from the National Health Interview Survey, and was coded as none (i.e., in bed or a chair most of the day, or no light-moderate or vigorous activities), light to moderate (i.e., light-moderate activity 1-3 times a week (any duration), or 2-4 times per week for less than 20 minutes, or vigorous activity once per week (any duration)), and moderate to heavy (i.e., light- moderate activity 4+ times per week for 20 minutes or more, or vigorous activity 2+ times per week (any duration)). Past week depressive symptoms were measured using an 11-item version of the Center for Epidemiologic Studies–Depression scale (CES-D) (α=0.85) (73).

Neighborhood Affluence—A neighborhood affluence scale was constructed using data from the 2000 US Census by calculating the average value of standardized variables for: 1) the proportion of employed civilians aged 16 years and older in professional/managerial occupations, 2) the proportion of individuals aged 25 years and older who have completed 16 or more years of education, and 3) median home values (α=0.94), following prior studies (31, 49). We also created a variable to reflect tertiles of neighborhood affluence.

Analyses

First, we provided a description of the sociodemographic characteristics of the sample. Second, we calculated the frequency of each component in the cumulative biological risk score for the full sample, and presented the mean values of each risk biomarker by quartile of childhood adversity. We evaluated significant differences across quartiles using models that accounted for clustering at the neighborhood level. Third, we fit a null model to examine the proportion of the variance in cumulative biological risk that can be attributed to differences between neighborhoods, to verify the suitability of using a neighborhood-level predictor. We calculated the intraclass correlation coefficient (ICC) using the formula $V_{neighborhood}/(V_{neighborhood} + V_{individual})$ where $V_{\text{neighborhood}}$ is the variance between neighborhoods and $V_{\text{individual}}$ is the variance within neighborhoods or between individuals. Fourth, we estimated associations between the continuous childhood adversity score and cumulative biological risk using a series of regression models. We used generalized linear mixed models with a Poisson link, allowed for neighborhood random effects, and adjusted standard errors for clustering at the neighborhood-level. We transformed the estimated coefficients to incidence rate ratios (IRR) to improve interpretability. All models controlled for sex, age, race/ethnicity, and medication use, and we sequentially introduced variables for 1) education and income, 2) depression, 3) health behaviors (smoking, alcohol consumption, physical activity), and 4) neighborhood affluence. Finally, we examined the interaction between childhood adversity and neighborhood affluence using a multiplicative interaction term; this was the only interaction examined in the present study. This interaction was further examined using models stratified by tertile of neighborhood affluence. All models were performed in using PROC GLIMMIX in SAS v.9.2, and statistical significance was established at $p<.05$ using two-sided tests.

Results

Sample characteristics are presented in Table 1. African Americans comprised approximately 35 percent of the sample, while US-born and foreign-born Hispanics each comprised roughly 10 percent of the sample. Over half of the sample was female (54.1 percent), and there was considerable heterogeneity by education and household income, and across health behaviors.

Among the eight tested biomarkers, high waist circumference was the most common risk factor (44 percent), followed by high CRP (38 percent), low HDL cholesterol (36 percent), and high SBP (19 percent) (see Table 2). High resting heart rate was the least common risk factor (8 percent). The median number of risk factors was 2 (mean=1.87, standard error (SE) = 0.12). The right-hand columns of Table 2 displays the mean values of each risk biomarker, stratified by quartile of childhood adversity. Bivariate analyses indicated significant associations between quartile of childhood adversity and HbA1c, SBP, and HDL cholesterol, whereby greater childhood adversity was associated with higher prevalence of the risk factor (*p*-values<.05). The cumulative biological risk score also increased with each quartile of childhood adversity: the mean cumulative biological risk score was 1.51 (SE = 0.23) in Quartile 1 and 2.31 (SE = 0.26) in Quartile 4 (*F*-value $p < 0.0001$).

In a null generalized linear mixed model, the variance attributable to the neighborhood was 0.13 (SE=0.04) and the variance attributable to the individual was 0.58 (SE=0.06). The ICC of 17.88% indicates that individuals from the same neighborhoods are likely to have more similar cumulative biological risk scores compared to individuals from other neighborhoods, and suggests that some of the variance in cumulative biological risk scores may be explained by neighborhood-level characteristics.

In the base model, childhood adversity was associated with a higher cumulative biological risk score. A 1 standard deviation increase in the childhood adversity z-score was associated with a 13% increase in cumulative biological risk, controlling for covariates in the model (Table 3, Model 1; IRR=1.13, 95% CI=1.06, 1.20). This association was sustained after additional adjustment for income and education (Model 2; IRR=1.11, 95% CI=1.04, 1.18)), depressive symptoms (Model 3; IRR=1.11, 95% CI=1.03, 1.18), health behaviors of smoking, alcohol consumption, and physical activity (Model 4; IRR=1.09, 95% CI=1.02, 1.17), and neighborhood affluence (Model 5; IRR=1.09, 95% CI=1.02, 1.17).Of note, higher neighborhood affluence was associated with a lower cumulative biological risk score, independent of childhood adversity and the other covariates in the models (IRR=0.82, 95% CI=0.74, 0.92).

In a test for a cross-level interaction between childhood adversity and neighborhood affluence using a model that included all covariates in Model 5, we observed a significant interaction between childhood adversity and neighborhood affluence, indicating that the association between childhood adversity and cumulative biological risk was stronger among individuals in low-affluence neighborhoods (IRR for interaction=0.92, 95% CI=0.86, 0.99; p=0.02). This interaction is displayed in Figure 1, which presents the fitted values for prototypical values of high-(80^{th} percentile), medium-(50^{th} percentile), and low-(20^{th} percentile) affluence neighborhoods. We further explored this interaction by computing models stratified by tertile of neighborhood affluence. In low affluence neighborhoods, a single standard deviation (SD) increase in childhood adversity was associated with a 16% increase in cumulative biological risk (IRR=1.16, 95% CI=1.05, 1.28); in contrast, in high-(IRR=0.97, 95% CI=0.83, 1.14) and middle-affluence (IRR=1.15, 95% CI=0.99, 1.33) neighborhoods, childhood adversity and cumulative biological risk were not associated at $p<.05$ (Table 4).

Discussion

In this probability sample of adults in Chicago, IL, childhood adversity was associated with elevated cumulative biological risk in adulthood, and this association was modified by neighborhood context. Specifically, the association between childhood adversity and cumulative biological risk was attenuated among individuals who resided in higher affluence neighborhoods, and amplified among individuals who resided in lower affluence neighborhoods. Models stratified by tertile of neighborhood affluence indicated that childhood adversity was associated with cumulative biological risk, but only among those individuals who lived in neighborhoods characterized by low affluence. These findings are consistent with the cumulative risk model (14-16), which theorizes that exposure to multiple stressors increases likelihood of disorder, and the stress-sensitization model (17-20), which

suggests that adversities in childhood can increase vulnerability to later stressors, thereby exacerbating the health consequences of stressors encountered later in life. It is also consistent with a buffering model (22-26), which suggests that neighborhood context may protect individuals from the consequences of stressful experiences.

These results add to a growing literature on biological and social mechanisms explaining the relationship between childhood adversity and elevated risk for chronic diseases (9, 10, 74), and extend existing evidence that childhood adversity is associated with dysregulation across multiple physiological systems among adults (43, 44). Although several previous studies have examined interactions between childhood adversity and stressors in adulthood on physiological outcomes (27-29), to our knowledge, this is the first study to evaluate whether positive neighborhood context in adulthood modifies the association between childhood adversity and biomarkers of risk in adulthood.

Our results are consistent with prior research which has shown that positive neighborhood attributes, such as social cohesion (26), green space (24, 25), or stability (23), can buffer against the negative consequences of individual vulnerability factors, such as stressors (23-25) or hostile maternal parenting (26). In addition, our finding that the negative health effects of childhood adversity were exacerbated among individuals who reside in low affluence neighborhoods supports previous research which shows that individuals with histories of childhood maltreatment have stronger inflammatory responses to indicators of social adversity, including caregiving stressors (27), daily stressors (28), and stress in a laboratory setting (29). Existing research suggests that the magnitude of the observed associations between childhood adversity and cumulative biological risk among individuals in low affluence neighborhoods is relevant to future morbidity and mortality risk. For example, in a study of high-functioning 70-79 year old adults, Karlamangla and colleagues (75) found that a one unit increase in allostatic load score (comprised of 10 biomarkers, 5 overlapping with our score) over 2.5 years was associated with an all-cause mortality odds ratio of 3.33 (95% CI: 1.14-9.74) over the subsequent 4.5 years.

Previous studies that examined interactions between neighborhood context and individual-level stressors have used self- or parent-reported health outcomes (18, 23); we strengthen this evidence base by documenting this interaction using measured biomarkers that reflect disease processes across multiple physiological systems. In future research, it will be valuable to determine whether this interaction extends to incident chronic diseases and cumulative biological risk calculated using other indicators and aggregation procedures. There is also a need to identify specific mechanisms that confer protection to individuals exposed to childhood adversity who reside in affluent neighborhoods in adulthood. Neighborhood affluence may reflect variation across neighborhoods in characteristics that serve to encourage better health, including: 1) structural resources that facilitate physical activity (i.e., parks, low crime rates), healthy eating (i.e., groceries), social connections (i.e., community centers, religious institutions), and preventive health care; and, 2) social norms that encourage healthy behaviors and discourage unhealthy behaviors (31). It will also be valuable for future research to look at specific components of the childhood adversity score individually, which could be informative for identifying the most efficacious targets for intervention.

The findings of the present study should be considered in the context of several limitations. First, this study used cross-sectional data; therefore, we cannot infer causation for the associations we examined. Related, we do not have information on neighborhood context in childhood, and we cannot disentangle the temporal relationship between income or educational attainment and neighborhood in adulthood. Second, childhood adversities were reported retrospectively, which has been shown to result in false negatives (i.e., under-reporting) and measurement error (76) which may have biased our results. Third, it is likely that there are unmeasured factors that influenced where people live as well as their health. Although we adjusted for demographic and behavioral factors, there is likely to be unmeasured confounding. Fourth, data from this study were drawn from Chicago, IL; further research is needed to establish whether these findings generalize to cities other than Chicago, and to non-urban areas. Fifth, although the neighborhood clusters were defined based on socially meaningful boundaries, in large cities such as Chicago there is likely to be variability within individual neighborhoods (46).

Finally, it is possible that our findings are affected by neighborhood selection, whereby individuals who experienced the greatest childhood adversity are clustered within low affluence neighborhoods (i.e., selection into neighborhoods is a non-random process) (77, 78). We therefore examined the distribution of childhood adversity by neighborhood affluence. We found representation of high childhood adversity (i.e., scores in the top quartile) at all quartiles of neighborhood affluence (see Appendix 1), which provides some evidence that our interaction results were not entirely driven by neighborhood selection (i.e., individuals the most adverse childhood experiences selected into the least affluent neighborhoods). In addition, we attempted to account for selection factors by adjusting for individual characteristics that could be associated with neighborhood selection including race/ethnicity, education, and income. In order to test our research question in the absence of neighborhood selection bias, future studies would require a study design where individuals have been randomly assigned to neighborhoods.

In conclusion, this study offers initial support for the hypothesis that residing in advantaged neighborhoods in adulthood may buffer against the harmful effects of childhood adversity on cumulative biological risk. These associations appear to be independent of adult SES (income or education), suggesting that positive neighborhood context may provide protective benefits beyond individual material or educational advantage. From a policy perspective, our findings suggest that developing and enhancing protective resources at the neighborhood-level may be valuable intervention strategies to protect health over the life course. Future research is needed to examine the sensitizing effect of childhood adversity in relation to other adversities encountered later in life, and to explicitly examine contextual-level processes in high affluence neighborhoods that support health among individuals exposed to childhood adversity. Through this research, it may be possible to identify modifiable individual- and neighborhood-level characteristics that can be targeted within interventions to promote wellbeing over time.

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Appendix 1

Distribution of respondents across quartiles of childhood adversity and neighborhood affluence (n=550); cells show weighted column % (and unweighted n).

	Childhood Adversity Score			
Neighborhood Affluence	**Q1**	**Q2**	**Q3**	**Q4**
Q1	11.19 (22)	16.59 (36)	25.30 (51)	14.60 (31)
Q2	29.92 (32)	30.10 (38)	23.94 (24)	23.01 (41)
Q3	20.35 (33)	23.93 (34)	24.36 (31)	33.11 (37)
Q4	38.54 (47)	29.38 (41)	26.41 (25)	29.28 (27)

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Acronyms

CCAHS	Chicago Community Adult Health Study
SBP	systolic blood pressure
DBP	diastolic blood pressure
HbA1c	hemoglobin A_{1c}
CRP	C-reactive protein
HDL	high density lipoprotein cholesterol
IRR	incident rate ratio
CI	confidence interval

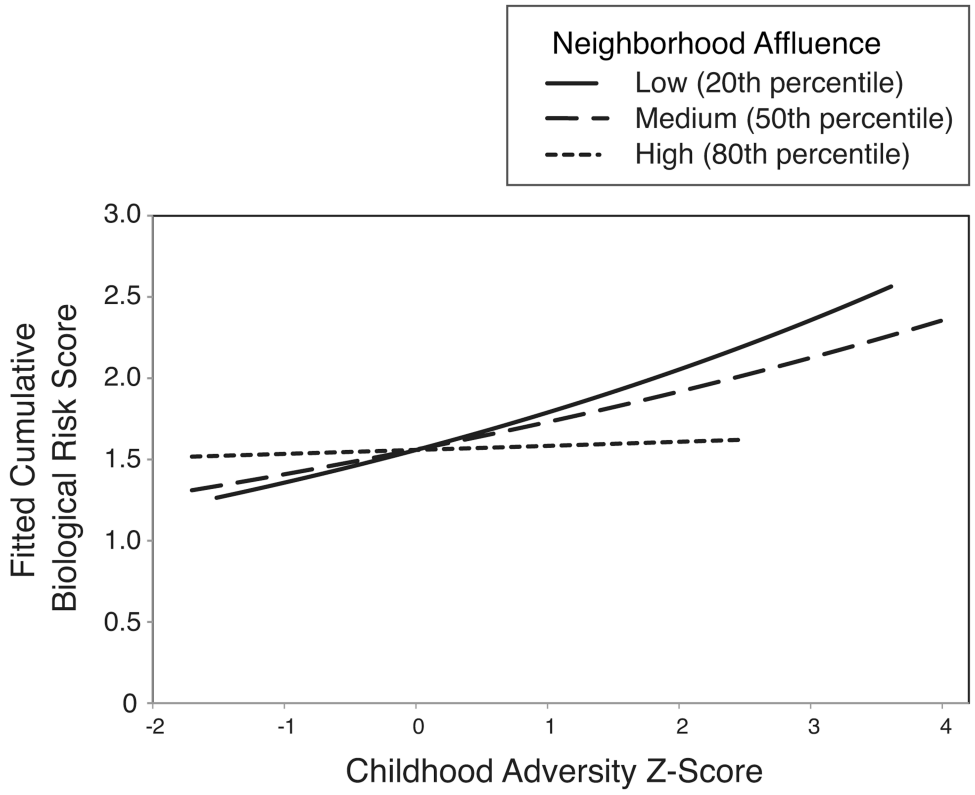

Figure 1.

Fitted values for the relationship between childhood adversity and cumulative biological risk for prototypical high, low, and medium affluence neighborhoods, controlling for sex, race, age, medication use, income, education, depressive symptoms, smoking status, physical activity, and alcohol consumption (n=550).

Table 1

Sociodemographic characteristics of sample (*n*=550)

	Unweighted, N	Weighted, % or mean (SD)
Sex		
Male	231	45.68
Female	319	54.32
Age	550	44.33 (17.08)
Race		
Black	200	35.28
Native Hispanic	72	9.93
Foreign Hispanic	76	10.26
Non-Hispanic White	202	44.53
Education		
<HS	135	21.32
HS	124	22.97
Some College	150	25.86
Bachelors+	141	29.86
Annual Income		
Missing	76	13.21
<10,000	67	10.43
10,000-29,999	150	23.95
30,000-49,999	111	18.60
50,000+	146	33.81
CES-D	550	1.86 (0.54)
Medication use[1]	165	31.90
Smoking		
Current	153	26.65
Previous	100	17.99
Never	297	55.36
Alcohol Consumption		
None	273	55.48

	Unweighted, N	Weighted, % or mean (SD)
0-31 drinks/month	65	9.96
32+ drinks/month	212	34.55
Physical Activity		
No physical activity	89	15.03
Light to moderate	209	37.63
Moderate to heavy	252	47.33

CES-D=Center for Epidemiologic Studies–Depression scale

[1]Self-report of medication to treat hypertension, diabetes, cholesterol, or arthritis.

Table 2

Frequency of individual biological risk factors and mean values (and standard errors) by childhood adversity quartiles (*n*=550)

	Full Sample		Mean Values (and SEs) by Childhood Adversity Quartiles				
Biological Risk Factor	**N[1]**	**Weighted % > threshold[2]**	**Q1**	**Q2**	**Q3**	**Q4**	**Pr >F-value[3]**
Waist circumference (cm)	251	44.03	90.69 (2.49)	94.67 (1.65)	93.38 (1.77)	96.65 (1.79)	0.22
Systolic blood pressure (mm Hg)	109	19.40	117.85 (2.60)	120.61 (2.25)	125.48 (2.53)	126.28 (2.79)	0.04
Diastolic blood pressure (mm Hg)	93	14.86	75.61 (1.67)	77.29 (1.18)	74.66 (1.10)	79.45 (1.38)	0.06
Resting heart rate (beats per minute)	46	8.29	71.78 (1.58)	71.92 (1.42)	69.70 (2.06)	73.91 (1.52)	0.31
HbA1c[4] (%)	66	12.88	5.15 (0.06)	5.57 (0.12)	5.56 (0.11)	5.91 (0.20)	<.0001
C-reactive protein[3] (mg/dL)	227	38.05	2.30 (0.32)	2.45 (0.29)	2.94 (0.49)	3.02 (0.35)	0.49
Total cholesterol (mg/dL)	77	13.32	196.68 (4.19)	190.65 (6.45)	194.45 (3.68)	202.65 (4.49)	0.41
HDL Cholesterol (mg/dL)	203	35.91	57.34 (1.76)	48.79 (1.54)	51.86 (2.36)	53.03 (2.34)	0.001
		Mean (SE)					
Cumulative Biological Risk, Mean (SD)	550	1.87 (0.12)	1.51 (0.23)	1.78 (0.14)	1.85 (0.14)	2.31 (0.26)	<0.0001

SE=standard error

[1] N is reflects unweighted value.

[2] Clinically-defined criteria for "high risk": systolic blood pressure ≥ 140 mm Hg or higher; diastolic blood pressure ≥ 90 mm Hg; resting heart rate ≥ 90 beats per minute; glycosylated/glycated hemoglobin ≥ 6.4%; C-reactive protein ≥ 3 mg/dl; total cholesterol ≥ 240 mg/dL; high density lipoprotein ≤ 40 mg/dL for men and ≤ 50 mg/dL for women; waist circumference ≥ 102 cm for men and ≥88 cm for women.

[3] *P*-values for individual risk factors (continuous data) were calculated used PROC SURVEYREG and reflect test of model effects; the *P*-value for the mean cumulative biological risk (count data) was calculated using PROC GLIMMIX and reflects the Type III test of fixed effects

[4] Difference between childhood adversity quartiles was calculated using log-transformed variable

Table 3

Incident rate ratios (and 95% confidence intervals) from weighted Poisson regressions of cumulative biological risk (CCAHS, *n*=550)[1]

	Model 1	Model 2	Model 3	Model 4	Model 5
Childhood Adversity Score	1.13 (1.06, 1.20)***	1.11 (1.04, 1.18)**	1.11 (1.03, 1.18)*	1.09 (1.02, 1.17)*	1.09 (1.02, 1.17)**
Education (ref=Bachelors+)					
<HS		1.38 (1.09, 1.76)**	1.39 (1.09, 1.76)**	1.30 (1.02, 1.66)*	1.16 (0.91, 1.49)
HS			1.54 (1.24, 1.92)***	1.46 (1.17, 1.82)**	1.32 (1.06, 1.65)*
Some college		1.54 (1.24, 1.92)**	1.27 (1.0.4, 1.55)*	1.28 (1.04, 1.57)*	1.17 (0.95, 1.44)
Annual Income ($) (ref=50,000+)					
Missing		0.87 (0.70, 1.08)	0.87 (0.70, 1.08)	0.77 (0.62, 0.96)*	0.75 (0.60, 0.94)*
<10,000		0.80 (0.63, 1.02)	0.80 (0.63, 1.02)	0.75 (0.59, 0.96)*	0.75 (0.58, 0.95)*
10,000-29,999		0.82 (0.68, 0.98)*	0.82 (0.68, 0.98)*	0.74 (0.61, 0.89)**	0.73 (0.60, 0.88)**
30,000-50,000		0.69 (0.56, 0.85)**	0.69 (0.56, 0.85)**	0.67 (0.54, 0.83)**	0.68 (0.55, 0.83)**
CES-D (continuous score)			1.00 (0.88, 1.13)	0.96 (0.85, 1.09)	0.95 (0.84, 1.08)
Smoking (ref=never smoker)					
Current smoker				1.00 (0.85, 1.17)	0.97 (0.83, 1.14)
Previous smoker				0.97 (0.80, 1.17)	0.96 (0.80, 1.16)
Alcohol Consumption (ref=none)					
1-31 drinks/month				0.77 (0.67, 0.89)**	0.78 (0.68, 0.90)**
32+ drinks/month				0.80 (0.60, 1.05)	0.85 (0.65, 1.12)
Physical Activity (ref=mod/heavy)					
No physical activity				1.36 (1.11, 1.65)**	1.33 (1.10, 1.62)**
Light to moderate activity				1.42 (1.21, 1.66)***	1.38 (1.18, 1.62)***
Neighborhood Affluence					0.82 (0.74, 0.92)**

* p<.05,

** p<.01,

*** p<.001;

ref=reference; CCAHS=Chicago Community Adult Health Study; CES-D= Center for Epidemiologic Studies–Depression scale.

[1] All models are adjusted for age, sex (male; female), race/ethnicity (White; Black; foreign-born Hispanic; US-born Hispanic), and self-report of medication to treat hypertension, diabetes, cholesterol, or arthritis.

Table 4

Associations between childhood adversity (z-score) and cumulative biological risk, stratified by tertile of neighborhood affluence (CCAHS, *n*=550)

	Neighborhood Affluence		
	Low	**Medium**	**High**
Childhood Adversity	1.16 (1.05, 1.28)*	1.15 (0.99, 1.33)	0.97 (0.83, 1.14)

Note: table presents incident rate ratios (and 95% confidence intervals) from weighted Poisson regressions adjusted for age, sex, race/ethnicity, medication use, education, income, CES-D symptoms, smoking status, physical activity, and alcohol consumption.

* p<.05;

CCAHS=Chicago Community Adult Health Study