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Cardiometabolic Dysfunction Among U.S. Adolescents and Area-Level Poverty: Race/Ethnicity-Specific Associations

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Abstract

Purpose: To examine race/ethnicity-specific associations between area-level poverty and cardiometabolic dysfunction among U.S. adolescents.

Methods: Data were from 10,415 adolescents aged 12—19 in the National Health and Nutrition Examination Survey (1999—2012), linked with census tract data on area-level poverty (the percent population living in poverty, grouped into race/ethnicity-specific quartiles). Cardiometabolic dysfunction was parameterized by summing z-scores of six cardiometabolic biomarkers, grouped into quintiles. Hierarchical ordinal models estimated overall and race/ethnicity specific associations. Posthoc analysis explored associations between area-level poverty and family poverty-to-income ratio.

Results: Overall, compared to adolescents residing in areas with the lowest area-level poverty (i.e., first quartile), residents in third (OR 1.32, 95% CI 1.13, 1.53) and fourth (OR 1.27, 95% CI 1.08, 1.50) quartiles of area-level poverty experienced elevated odds of cardiometabolic dysfunction. Area-level poverty predicted cardiometabolic dysfunction between non-Hispanic white and Mexican American adolescents, but not between non-Hispanic black adolescents.

Conclusions: We found race/ethnicity-specific associations between area-level poverty and cardiometabolic dysfunction among U.S. adolescents, highlighting the moderating effect of race-ethnicity. Among non-Hispanic black adolescents, neither higher area-level nor family-level socioeconomic status is associated with cardiometabolic health, in contrast to non-Hispanic white

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Supplementary data

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adolescents. Similar associations among non-Hispanic white and Mexican American groups aligns with evidence of the Hispanic Paradox. Future studies of effect of area-level determinants of cardiometabolic dysfunction may consider race/ethnicity-specific associations.

Keywords

Racial disparities; Poverty; Cardiometabolic; Adolescents; NHANES

Twenty-six percent of deaths in the United State are attributed to cardiometabolic disease, and minority populations bear a disproportionately high burden of mortality due to these diseases [1]. Reducing the burden of cardiometabolic diseases and identifying their precursors are public health priorities for the United States [2] Evidence is accumulating that cardiometabolic dysfunction during adolescence is a precursor of later cardiometabolic disease [3–6], as it tracks over time and can predict onset of cardiometabolic disease 25 years later [6]. Thus, although it is rare for adolescents to meet diagnostic criteria for cardiometabolic diseases, adolescents with cardiometabolic functioning deviating from population-level norms can be considered at risk of developing cardiometabolic disease during adulthood [3–6]. There are persistent racial and ethnic disparities in the occurrence and severity of cardiometabolic diseases. For example, in 2013, the cardiovascular disease mortality rate was 30 percent higher among non-Hispanic black than among non-Hispanic white Americans [7]. As noted by the American heart association, persistent disparities necessitate examination of potential modifying effect of race/ethnicity on determinants of cardiometabolic dysfunction [2]. Below, we examine race/ethnicity-specific associations between exposure to area-level poverty and cardiometabolic dysfunction among U.S. adolescents.

Research on contextual determinants of cardiometabolic dysfunction among adolescents has focused primarily on the association between area-level socioeconomic status (SES) and adiposity or blood pressure. This evidence indicates that residence in low-SES areas predicts an elevated risk of adiposity and explains a significant amount of racial/ethnic disparities in adiposity among U.S. adolescents [8,9]. In contrast, of four studies of area-level SES and blood pressure among adolescents, all conducted among small samples (range: 24–325) [10–13], only one found an association [10]. Two of the studies with null findings included only individuals with a family history of cardiometabolic disease, potentially limiting variability in cardiometabolic function [11,13]. None of the studies examined race/ethnicity-specific associations between area-level SES and blood pressure [10–13].

Prior studies of adolescents have also observed a link between area-level SES and either an index of cardiometabolic dysfunction or allostatic load, a construct related to cardiometabolic dysfunction [14–16]. Among U.S. adolescents (n = 11,030), area-level SES at ages 12–19 predicted cardiometabolic dysfunction at ages 25–32, this association was weakened but remained significant after inclusion of individual-level covariates in regression models [16]. Among U.S. adolescents aged 12–20 (n = 11,886), area-level SES was associated with allostatic load [15], this association was strongest among individuals with low family poverty-to-income ratio (PIR) [15]. Among a sample of non-Hispanic black adolescents (n = 420) increasing area-level poverty between the ages 11 and 19 predicted

allostatic load at age 19 [14]. Of the two studies that examined the interaction between race and area-level SES, one did not find any differences by race [15], and the other observed a stronger association among non-Hispanic white individuals than among racial/ethnic minorities [16]. Neither of these studies [15,16] conducted stratified analysis to examine race/ethnicity-specific associations between area-level SES and cardiometabolic dysfunction among adolescents.

In the United States, non-Hispanic black and Hispanic individuals are more likely to reside in lower SES areas compared with non-Hispanic white individuals [17]. And, evidence is emerging that population-level indices of economic stratification, such as various measures of area-level SES or income inequality, when applied to census tracts or ZIP codes mask racial-ethnic distinctions within these areas. Thus, even within an area with apparently homogenous SES, economic segregation by race/ethnicity persists such that white and racial/ethnic subpopulations have distinct area-level economic experiences [18]. Consequently, a better understanding of the race/ethnicity-specific associations between area-level SES and health outcomes is needed [17]. To our knowledge, this is the first study to examine race/ethnicity-specific associations between area-level SES and cardiometabolic dysfunction in a nationally representative sample of U.S. adolescents. We hypothesized that residence in areas with higher prevalence of poverty is associated with worse cardiometabolic dysfunction and that the association between area-level poverty and cardiometabolic dysfunction will be stronger among white adolescents than other racial/ethnic groups [16,18,19].

Methods

Data were drawn from 1999 to 2012 National Health and Nutrition Examination Survey (NHANES), a cross-sectional survey of the noninstitutionalized U.S. population conducted continuously since 1999 in 2-year cycles [20]. The analytic sample was selected from 13,343 adolescents, aged 12–19 years. We excluded respondents who reported current pregnancy (n = 181), diagnosis of hypertension or diabetes or using medication for hypertension or diabetes (n = 83). Respondents with missing values on any of the cardiometabolic variables were excluded (n = 2,664), leaving an analytic sample of 10,415 adolescents residing within 3,140 census tracts (average 3.34 adolescents per tract).

Area-level data were drawn from 2000 United States decennial census and 5–year estimates (2005–2009; 2009–2013) from the American Community Survey. Individual and area-level data were linked with contemporary census tract identifiers (Online Figure 1).

Cardiometabolic dysfunction score:

We created an index of cardiometabolic dysfunction based on six metabolic and cardiovascular biomarkers. Our decision to create an index of cardiometabolic function was informed, in part, by evidence that a composite score that reflects level of risk across multiple factors better than dichotomous indicator constructed from multiple indicators of risk [3]. We incorporated the following six biomarkers into our index of cardiometabolic dysfunction: glycosylated hemoglobin (%) (three month average blood glucose level) [20]; mean systolic and diastolic blood pressure (mmHg) were obtained when two or more

measurements were available (n = 10,266; 98%); high density lipoprotien cholesterol (mg/dL) (high density lipoprotien was multiplied by –1 to ensure all biomarkers had the same direction) [20]; total cholesterol (mg/dL) [20]; and waist circumference (cm) [20]. Our choice of these biomarkers from biomarkers available in NHANES was informed by how well biomarkers track over time in relation to cardiometabolic health and by considering missingness. Total cholesterol was included as a measure of lipid metabolism as it better tracks over time [21] and also allows for sufficient sample size compared to other measures of lipid metabolism which (i.e., triglycerides) would have reduced the sample size by 52% as these measures are only available for a fasting subsample in NHANES (data not shown). HbA1c was included as a measure of glucose metabolism as it tracks well over time, and is less influenced by recent diet or illness than fasting glucose [22]. We summed z-scores for each biomarker to create an overall cardiometabolic dysfunction index. The z-scores were normed by age and gender, except for blood pressure which was normed by age, gender, and height, consistent with guidelines on hypertension in adolescents [23]. This method of calculating the cardiometabolic dysfunction score can result in negative numbers, as the two blood pressure variables are standardized on an external reference category [23]. Higher cardiometabolic dysfunction scores indicate greater dysfunction. We created quintiles of cardiometabolic dysfunction based on the sample distribution and used the lowest quintile as the reference group.

We used area-level poverty, the proportion of residents in the census tract living below federal poverty threshold, as our measure of area-level SES. Multifactorial indices of area-level SES, such as an index of area-level deprivation, are robust measures of overall area-level SES [8,24]. Yet due to their complexity, it is often unclear which of the component factors are driving an association. Prevalence of poverty is easily interpretable and correlates with other measures of area-level SES such as housing quality and crowded living spaces, and has been shown to produce similar results as multifactorial indices [25,26].

We parameterized cardiometabolic dysfunction into quintiles. We made this choice in order to improve interpretation of our results. Progression from one unit to another on a continuous scale reflects a very small change in the risk of cardiometabolic dysfunction; in contrast, progression from one category to the next category arguably corresponds to a more biologically meaningful change in risk as each of the ordinal categories includes multiple units in a continuous scale. Finally, to account for differences in the distribution of poverty by race/ethnicity (Online Tables 1 and 2), in race/ethnicity-specific analysis, we estimated area-level poverty quartiles on race/ethnicity-specific distribution of poverty. We used census tracts as our unit of analysis because census tracts are more economically homogenous than other geographies such as ZIP codes, commonly used in studies of area-level SES and health outcomes, and smaller geographies such as block groups would not include a sufficient number of NHANES participants to run hierarchical models [27,28].

The following covariates were included in the regression models: 2 year survey cycle, race/ethnicity (non-Hispanic white, non-Hispanic black, Mexican American, and Other), family PIR (an indicator of family SES; continuous), parental education (indicator variables for less than high school, high school, greater than high school), serum cotinine level (continuous), and physical activity (coded 1: yes to “Do you do any vigorous-intensity sports, fitness, or

recreational activities that cause large increases in breathing or heart rate like running or basketball for at least 10 minutes continuously?"), and prevalence of non-Hispanic black population at the census tract level (continuous). These covariates have been identified in previous studies as correlates of cardiometabolic dysfunction [9,10,15].

Statistical analysis:

First, we conducted descriptive analyses, using survey commands to account for the complex survey design. Second, we estimated the association between area-level poverty and cardiometabolic dysfunction using a series of hierarchical ordinal regression models with random intercepts, adjusted for clustering at the census tract level. Each level of cardiometabolic dysfunction serves as a threshold sequentially, yielding a series of binary estimates where odds of being in categories at or above that threshold is compared to odds of being in categories below that threshold (Table 1, Eqs. (1)–(3)). These individual models yield a cumulative odds ratio that reflects the combined odds ratio across all possible thresholds of cardiometabolic dysfunction (Eq. (4)). Thus, the cumulative odd is interpreted as the odds of being in any category of worse cardiometabolic dysfunction (Eq. (4)). Hierarchical ordinal regression models, adjusted for clustering at the census tract level, were fit using PROC GLIMMIX in SAS 9.3, using the lowest cardiometabolic dysfunction category as the reference category.

First, we fit a model that included area-level poverty and indicator variables for survey cycle. Second, race/ethnicity, family PIR, head of household education level, serum cotinine, and physical activity were included in the model. Third, prevalence of non-Hispanic black population was added to the model. Age and gender were not included in the models as cardiometabolic dysfunction is standardized by these two variables. Next, race*poverty interaction terms were fit. If the log likelihood ratio test indicated that inclusion of interaction terms significantly improved model fit, separate models were fit by race/ethnicity. Because racial/ethnic groups are unevenly distributed across area-level poverty quartiles (Online Table 2), we fit stratified models with two methods of determining area-level poverty: (1) Quartiles based on overall sample distribution of area-level poverty and (2) quartiles of area-level poverty based on race/ethnicity-specific poverty distribution. The race/ethnicity group designated as 'other' by NHANES is small and heterogeneous, thus, they were included in the analytic sample but results for this group are not presented. In posthoc analysis, we explored race/ethnicity specific associations between area-level poverty and family PIR. This may provide evidence of race/ethnicity-specific economic experiences in areas of comparable SES.

Responses of "Don't Know," "Refused," and "Missing" were treated as missing values. Missing values for family PIR, serum cotinine, parental education, and physical activity were imputed 10 times using SAS PROC MI, and results were obtained using PROC MIANALYZE in SAS 9.3. As NHANES uses a complex, stratified, and multistage sample design, all analyses used Mobile examination center person-level survey weights, scaled to the census tract level, to account for oversampling, noncoverage and nonresponse [29]. Analysis of restricted data was conducted in the research data center at the National Center

for Health Statistics in Hyattsville, MD, and was considered exempt by the University of Maryland Institutional Review Board.

Results

Table 2 includes mean cardiometabolic dysfunction score and mean cardiometabolic biomarker levels by area and individual-level covariates. Residents of the highest poverty areas (i.e., fourth quartile), compared to lowest poverty areas (i.e., first quartile), experienced an average of 20% increase in cardiometabolic dysfunction score (fourth quartile mean score = −.695 (95% CI –.775, –.614) versus first quartile mean score = –.866 (95% CI –.940, –.792). The negative cardiometabolic dysfunction scores resulted from standardization of the two blood pressure variables by an external reference category [23].

Differences in mean cardiometabolic dysfunction were observed by race/ethnicity. Non-Hispanic black adolescents had the highest mean cardiometabolic dysfunction score (mean –.612, 95% CI –.671, –.552), followed by non-Hispanic white adolescents (mean: −.818, 95% CI –.877, –.759), and Mexican American adolescents (mean –.857, 95% CI –.923, –.791).

Table 3 presents results of hierarchical models estimating the association between area-level SES and the cardiometabolic dysfunction. In model 1, area-level poverty is positively associated with cardiometabolic dysfunction (column 1); this association is independent of individual-level covariates (model 2) and prevalence of non-Hispanic black residents (model 3). Relative to residents of areas with the lowest (i.e., first quartile) area-level poverty, residents of third, and fourth quartiles of area-level poverty experienced 32% (95% CI 1.13, 1.53), and 27% (95% CI 1.08, 1.50) elevated odds of higher cardiometabolic dysfunction score, respectively (model 3).

Inclusion of individual-level covariates (model 2) and prevalence of non-Hispanic black residents (model 3) resulted in similar coefficients for area-level poverty compared to model 1. This suggests that the association between area-level poverty and cardiometabolic dysfunction is not explained by the prevalence of non-Hispanic black residents.

The log likelihood ratio test comparing model 3 with a model that included an interaction term for area-level poverty and race/ethnicity indicates that the interaction significantly improved model fit (χ^2: 18.02, df = 9, p =.034).

In fully adjusted race-ethnicity specific models using race-specific poverty distributions (Table 3, Model 3), among non-Hispanic white adolescents, residents of the fourth quartile of area-level poverty experience 39% (95% CI 1.04, 1.86) elevated odds of greater cardiometabolic dysfunction compared with adolescents living in tracts with the lowest poverty levels. Among Mexican American adolescents, residents in the third and fourth quartiles of area-level poverty experienced 36% (95% CI 1.03, 1.80) and 38% (95% CI 1.04, 1.82) elevated odds of greater cardiometabolic dysfunction, respectively. Among non-Hispanic black adolescents, all ORs were close to 1 (OR range: 0.88–1.07), and none were statistically significant. Similar to overall analyses, inclusion of individual and area-level covariates did not result in a meaningful change in the coefficients for area-level poverty

(Table 3). The patterns of the race/ethnicity-specific associations between area-level poverty and cardiometabolic dysfunction were generally similar when using overall poverty distribution quartiles (Online Table 3).

In posthoc analysis, we explored associations between area-level poverty and family PIR, and whether these associations differ by race/ethnicity (Table 4). Overall, mean family PIR declined from 3.34 in quartile 1 to 1.42 in quartile 4 ($p < .001$). Within each racial/ethnic group, mean family PIR declined across area-level poverty quartiles. However, the magnitude of this difference was greatest among non-Hispanic white adolescents. Between the first and fourth quartiles, non-Hispanic white adolescents had a 1.93 unit decline ($p < .001$), non-Hispanic black adolescents had a 1.27 unit decline ($p < .001$), and Mexican American adolescents had a 1.22 unit decline ($p < .001$).

Within each quartile of area-level poverty, racial/ethnic differences in mean family PIR are attenuated as area-level poverty increases. For example, in quartile 1, mean family PIR for non-Hispanic white adolescents is .92 units higher than mean family PIR for non-Hispanic black adolescents, and this difference is only .26 in quartile 4 (Table 4).

Discussion

In this first examination of race/ethnicity-specific associations between area-level poverty and cardiometabolic dysfunction among U.S. adolescents, we observed a positive association between prevalence of area-level poverty and cardiometabolic dysfunction among non-Hispanic white and Mexican American adolescents but not among non-Hispanic black adolescents. This observation is in line with accumulating evidence of race/ethnicity-specific associations between contextual poverty and various health outcomes. For example, among U.S. adolescents, area-level SES at ages 12–19 predicted cardiometabolic dysfunction at ages 25–32, and this association was stronger among non-Hispanic white adolescents than among other racial/ethnic groups [16]. Similarly, among diverse U.S. samples, area-level SES predicted risk of smoking [30] among non-Hispanic white adolescents but not among other racial/ethnic groups. Race/ethnicity-specific associations have also been observed between income inequality and health among children and adolescents. In U.S. metropolitan areas, income inequality predicts exposure to second hand smoke [18] among non-Hispanic white children, but not among non-Hispanic black children. In U.S. metropolitan areas, increasing income inequality is associated with increasing risk of mortality among non-Hispanic black children, but lower risk of morality among non-Hispanic white children [19].

The lack of an association between area-level poverty and cardiometabolic dysfunction among non-Hispanic black adolescents, implicates the primacy of structural racism that specifically influences the lived experiences of non-Hispanic blacks and limits political power, social status, and access to resources [31]. Among the key consequences of structural racism in US, is diminished returns on achievements such as a hindrance to upward residential mobility that segregates non-Hispanic blacks in relatively high poverty localities regardless of their family income [32]. Our posthoc analysis shows that in the U.S., high-income non-Hispanic white families reside in lower poverty areas than non-Hispanic black

and Mexican American families with similar family income (Table 4). Likewise, Logan et al report that non-Hispanic black households earning over $75,000 in annual income live in areas with about 15% prevalence of poverty, whereas, non-Hispanic white households with less than $40,000 annual income reside in areas with about 13% prevalence of poverty [31]. Consequently, within geographies that may appear to be economically homogenous (e.g., census tracts), non-Hispanic black adolescents are likely to reside in relatively segregated localities with limited structural resources (e.g., safe public spaces and quality educational opportunities) and high prevalence of poverty [33]. In sum, despite the importance of a neighborhood's socioeconomic conditions for its residents health [9], non-Hispanic black adolescent's consistent exposure to relatively high levels of area-level poverty [31] can render a statistically null effect when considering the relative effect of varying degrees of area-level poverty among census-tracts, as is the case in the current study.

Additionally, our observation of similar associations among Mexican American and non-Hispanic white adolescents is in accord with the literature on Hispanic paradox [34]. While Mexican American individuals also experience structural racism [31], the extensive literature that has documented paradoxically positive health outcomes among Hispanic populations in U.S. despite their relatively lower SES supports our findings.

Of note is that non-Hispanic black adolescent's lower cardiometabolic dysfunction scores relative to non-Hispanic white adolescents belie the higher burden of cardiovascular disease among black adults. Although physiologic, genomic, and environmental factors have been proposed as explanations for this phenomenon, our understanding of this phenomenon remains rudimentary [35]. Thus, it remains that cardiometabolic dysfunction may not predict future disease among non-Hispanic blacks as it does among non-Hispanic whites [35,36]. However, this lack of predictability is not plausibly associated with neighborhood poverty and does not explain the null association between poverty and cardiometabolic dysfunction among African- Americans in our sample.

Our analysis has several strengths. First, our ordinal measure of cardiometabolic dysfunction is a crude representation of a continuous cardiometabolic dysfunction score, and may better reflect population-level variation compared to a dichotomous measure and may be a better predictor of adult health than a dichotomous variable [3]. Additionally, the ordinal cardiometabolic dysfunction variable may reduce potential misclassification compared to a dichotomous measure. Secondly, our use of contemporary area-level data minimizes potential misclassification of an individual's area-level poverty due to temporality. For example, for an individual in NHANES 2011–2012, the poverty rate of their census tract of residence will be more accurately reflected in ACS 2009–2013 data than in census 2000 data. Results from the current study are also in line with previous work that area-level prevalence of non-Hispanic black population is often used as an indirect measure of racial makeup of a residential area, a key area-level exposure that was included in the models to allow estimation of the effect of area-level poverty independent of racial-makeup of the area [37].

Third, we used biomarkers of lipid and glucose metabolism (i.e., total cholesterol and HbA1c) that track better over time than biomarkers, such as fasting glucose and

triglycerides, used in other studies [21,22]. Nonetheless, the observed racial/ethnic disparities in cardiometabolic dysfunction among adolescents are consistent with evidence that non-Hispanic black populations have the highest prevalence of cardiometabolic disease in adulthood [1]. Lastly, in line with previous works we included in the regression models the prevalence of non-Hispanic black population as an indirect measure of racial composition of residential areas to allow estimation of the effect of area-level poverty independent of racial composition of the area [37].

These findings should be considered in the context of this study's limitations. Our index of cardiometabolic dysfunction is not a validated predictor of future disease and the quintile cut points used do not represent a clinically meaningful increase in risk. However, our index of cardiometabolic dysfunction reflects levels of six biomarkers informed by measurements of metabolic syndrome [3], and preclinical elevations in these biomarkers track well over time and are predictive of future disease. As with other studies that have relied on administratively defined geographic units, findings may differ based on how geographic units are defined [38]. However, census tracts are typically considered to allow for the most accurate and reliable data on poverty, while also containing a sufficient number of NHANES participants to implement hierarchical models. Furthermore, individuals residing within arbitrarily defined contexts may not identify with those boundaries, thus, introducing a degree of measurement error. Next, cross-sectional epidemiologic associations do not allow causal inference. Given the cross-sectional design of our study, we cannot directly test the hypothesis that adolescents with greater cardiometabolic dysfunction drifted to reside in poorer areas. However, given that adolescents usually do not choose their place of residence, we reason that our findings are in line with the extensive literature on the social causation hypothesis and place of residence as a determinant of poor health [9,39]. Also, we used different biomarkers for lipid metabolism (total cholesterol) and glucose metabolism (HbA1c) than in prior studies of U.S. adolescents, which limits comparability across studies. Additionally, health behavior measures in NHANES, including physical activity, are self-reported and therefore lack precision. Consequently, a degree of residual confounding likely remains. Lastly, individuals with undiagnosed diabetes may have been included in the analyses, likely introducing bias. However, most cases of diabetes among adolescents are type 1 diabetes, which is more likely to be diagnosed, minimizing this concern.

Further research may help in better understanding race/ethnicity-specific associations between contextual factors and cardiometabolic health among adolescents. As contextual factors, such as racial segregation and poverty, change over time, studies can better account for social and economic changes by making use of historic census data. Additionally, longitudinal studies that track individual's residence over time would allow for a better understanding of cumulative exposure to contextual factors, including area-level poverty, and how these cumulative exposures relate to the development of disease and related health disparities across the life course. Investigating interactions between area-level SES and individual-level factors that differ by race/ethnicity, may provide a better understanding of the determinants of racial and ethnic disparities in cardiometabolic health.

In conclusion, we found race/ethnicity-specific associations between area-level poverty and cardiometabolic dysfunction among U.S. adolescents, independent of individual-level and

area-level covariates. The association between area-level poverty and cardiometabolic dysfunction was present for non-Hispanic white and Mexican American adolescents but not for non-Hispanic black adolescents. These findings suggest there may be racial/ethnic differences in the associations between area-level SES and health outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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IMPLICATIONS AND CONTRIBUTION

The present study contributes evidence suggesting race/ethnicity-specific individual-level experiences modify the association between area-level exposures and health among adolescents. Studies of contextual determinants of disparities in cardiometabolic health across the life course would benefit by considering the modifying influence of race and ethnicity.

Table 1

Ordinal logistic regression model specification

Eqs. (1)-(3):
$$L_1 = \log\left(\frac{\pi_1}{\pi_2 +}\pi_3 + \cdots + \pi_r\right)$$
$$L_2 = \log(\pi_1 + \frac{\pi_2}{\pi_3 +}\pi_4 + \cdots + \pi_r)$$
$$L_n = \log(\pi_n + \frac{\pi_{n+1}}{\pi_{n+2} +}\pi_{n+3} + \cdots + \pi_r)$$
Eq. (4):
$$P(Y \leq j) = \pi_1 + \ldots + \pi_J$$
$$\log(P(Y \overset{\leq j)}{P}(Y > j)) = \log\left(\frac{\pi_1 + \cdots + \pi_j}{\pi_{j+1} + \cdots +}\pi_j\right)$$
Components:

π_1: Probability of being in category 1 of Cardiometabolic Dysfunction	π_4: Probability of being in category 4 of Cardiometabolic Dysfunction
π_2: Probability of being in category 2 of Cardiometabolic Dysfunction	π_r: Probability of being in higher ordered category of Cardiometabolic Dysfunction
π_3: Probability of being in category 3 of Cardiometabolic Dysfunction	j: Threshold of interest (Cardiometabolic Dysfunction category 1)

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Table 2

Weighted mean cardiometabolic dysfunction score and biomarkers by demographic variables and area-level variables (NHANES1999–2012)

Variable *(Unweighted N; %)*	Cardiometabolic dysfunction mean (95% CI)	SBP mean (95% CI)	DBP mean (95% CI)	HbA1c mean (95% CI)
Total*(10415; 100)*	−.794 (−.834, −.754)	109.62 (109.34, 109.90)	60.72 (60.41, 61.04)	5.15 (5.15, 5.16)
Area−level poverty				
Quartile 1(2599; 24.95)	−.866 (−.940, −.792)	109.06 (108.55, 109.58)	60.68 (60.10, 61.26)	5.13 (5.11, 5.14)
Quartile 2(2615; 25.10)	−.825 (−.897, −.754)	109.57 (109.04, 110.10)	60.70 (60.11, 61.29)	5.14 (5.13,5.16)
Quartile 3(2584; 24.81)	−.692 (−.782, −.604)	109.98 (109.39,110.58)	60.86 (60.16, 61.55)	5.18 (5.16,5.19)
Quartile 4(2595; 24.91)	−.695 (−.775, −.614)	110.56 (110.00,111.12)	60.69 (60.04, 61.33)	5.20 (5.19,5.22)
Prevalence of NH black population				
Quartile 1(2599; 24.95)	−.796 (−.871, −.722)	109.14 (108.59,109.68)	61.30 (60.71, 61.88)	5.14 (5.12,5.15)
Quartile 2(2604; 25.00)	−.786 (−.867, −.705)	109.59 (109.04,110.15)	60.88 (60.23, 61.53)	5.14 (5.12,5.15)
Quartile 3(2605; 25.01)	−.838 (−.913, −.764)	109.65 (109.13,110.16)	60.06 (59.50, 60.62)	5.16 (5.14,5.17)
Quartile 4(2599; 24.95)	−.727 (−.806, −.645)	110.73 (110.22,111.24)	60.22 (59.93, 60.91)	5.21 (5.20,5.23)
Family PIR				
< 1 (*3074; 29.52*)	−.727 (−.799, −.656)	109.99 (109.49,110.49)	60.72 (60.12, 61.32)	5.18 (5.16,5.19)
1–2.9 (*4005; 38.45*)	−.769 (−.834, −.704)	109.71 (109.27,110.15)	60.53 (60.02, 61.05)	5.15 (5.14,5.17)
3–4.9 (*1606; 15.42*)	−.820 (−.908, −.731)	109.61 (108.95,110.26)	60.85 (60.15, 61.56)	5.15 (5.13,5.17)
> 5 (*967; 9.28*)	−.903 (−.102, −.782)	108.92 (108.07,109.78)	61.12 (60.21,62.03)	5.12 (5.10,5.14)
Missing (*763; 7.33*)	−.799 (−.974, −.624)	109.65 (108.52,110.77)	60.39 (58.90, 61.88)	5.18 (5.15,5.21)
Age				
12–14 (*3981; 38.22*)	−.770 (−.835, −.705)	106.86 (106.45,107.28)	51.98 (57.44, 58.52)	5.18 (5.17,5.19)
15–17 (*3964; 38.06*)	−.757 (−.817, −.696)	110.49 (110.06,110.93)	61.65 (61.17, 62.12)	5.15 (5.13,5.16)
18–19 (*2470;23.72*)	−.893 (−.982, −.803)	112.60 (111.99,113.21)	63.60 (62.97, 64.22)	5.13 (5.11,5.14)
Gender				
Female (*5015; 48.15*)	−.634 (−.691, −.577)	106.83 (106.48,107.18)	61.74 (61.32, 62.16)	5.14 (5.13,5.15)
Male (*5400; 51.85*)	−.942 (−.998, −.887)	112.23 (111.82,112.64)	59.78 (59.31,60.24)	5.17 (5.16,5.18)
Race/Ethnicity				
NH White (*2756; 26.46*)	−.818 (−.877, −.759)	109.42 (109.01,109.83)	61.27 (60.80, 61.74)	5.12 (5.10,5.13)
NH Black (*3052; 29.30*)	−.612 (−.671, −.552)	111.83 (111.40,112.25)	60.71 (60.21,61.21)	5.26 (5.25,5.28)
Mex. Am. (*3342; 32.09*)	−.857 (−.923, −.791)	109.34 (108.90,109.79)	58.33 (57.79,58.87)	5.17 (5.16,5.19)

Variable*(Unweighted N; %)*	WC mean (95% CI)	TC mean (95% CI)>	HDL-C mean (95% CI)
Total (*10415; 100*)	81.42 (81.04,81.81)	159.83 (159.00,160.65)	50.69 (50.37, 51.01)
Area-level poverty			
Quartile 1(2599; 24.95)	79.97 (79.31,80.62)	160.14 (158.61,161.66)	50.81 (50.21,51.41)
Quartile 2(2615; 25.10)	81.57 (80.85, 82.29)	159.76 (158.19,161.33)	50.38 (49.78,50.98)
Quartile 3(2584; 24.81)	82.72 (81.82, 83.61)	159.63 (157.85,161.40)	50.62 (49.94,51.29)
Quartile 4(2595; 24.91)	82.83 (82.00, 83.66)	159.40 (157.87,160.94)	51.05 (50.38,51.72)
Prevalence of NH black population			
Quartile 1(2599; 24.95)	81.54 (80.86, 82.23)	159.91 (158.38,161.44)	50.08 (49.49,50.66)
Quartile 2(2604; 25.00)	81.54 (80.74, 82.34)	159.34 (157.65,161.03)	50.48 (49.84,51.12)
Quartile 3(2605; 25.01)	81.44 (80.71,82.17)	159.92 (158.36,161.48)	50.93 (50.28,51.59)
Quartile 4(2599; 24.95)	80.89 (80.11,81.67)	160.36 (158.80,161.93)	52.02 (51.38,52.66)
Family PIR			
< 1 (*3074; 29.52*)	82.84 (82.08, 83.16)	159.58 (158.03,161.12)	50.37 (49.75,50.99)
1–2.9 (*4005; 38.45*)	81.86 (81.22, 82.05)	160.26 (158.92,161.60)	50.19 (49.68,50.70)
3–4.9 (*1606; 15.42*)	80.54 (79.66, 81.41)	158.71 (156.77,160.65)	50.54 (49.77,51.30)
> 5 (*967; 9.28*)	79.62 (78.62, 80.63)	160.49 (158.08,162.90)	52.12 (51.20,53.04)
Missing (*763; 7.33*)	81.65 (80.18, 83.12)	160.41 (157.59,163.24)	51.62 (50.42,52.82)
Age			
12–14 (*3981; 38.22*)	77.65 (77.07, 78.24)	158.59 (157.33,159.85)	51.70 (51.18,52.22)
15–17 (*3964; 38.06*)	82.35 (81.75, 82.95)	158.03 (156.68,159.38)	49.99 (49.48,50.51)
18–19 (*2470; 23.72*)	85.94 (85.13, 86.75)	164.82 (163.03,166.61)	50.21 (49.54,50.89)
Gender			
Female (*5015; 48.15*)	80.81 (80.28, 81.35)	162.54 (161.36,163.72)	53.14 (52.67,53.62)
Male (*5400; 51.85*)	81.99 (81.45, 82.54)	157.29 (156.15,158.43)	48.39 (47.97,48.82)
Race/Ethnicity			
NH White (*2756; 26.46*)	81.54 (80.97, 82.12)	160.17 (158.93,161.42)	49.82 (49.34,50.30)
NH Black (*3052; 29.30*)	80.66 (80.03, 81.29)	160.26 (159.09,161.42)	54.25 (53.75,54.75)
Mex. Am. (*3342; 32.09*)	83.44 (82.83, 84.06)	158.78 (157.60,159.96)	50.36 (49.83,50.89)

DBP = Diastolic Blood Pressure (mmHg); HbA1c = Glycosylated Hemoglobin (% blood glucose); HDL-C = High Density Lipoprotein Cholesterol (mg/dL); SBP = Systolic BloodPressure (mmHg); TC = total cholesterol (mg/dL); WC = waist circumference (cardiometabolic).

Cardiometabolic dysfunction is a sum of z-scores for glycosylated hemoglobin levels, waist circumference, HDL cholesterol and total cholesterol, and for systolic and diastolicblood pressure. Due to small cell size, missing values not reported for Area Deprivation (n = 22) and Prevalence of non-Hispanic black population (n = 8). Other race/ethnicitynot reported here due to heterogeneity of group.

Table 3

HLM models estimating the association between area-level SES and cardiometabolic index

Variable	Overall poverty distribution	Race/ethnicity-specific Poverty distribution		
	Cardiometabolic dysfunction (n = 10415) OR (95% Ci)	NH white (n = 2756) OR (95% CI)	NH black (n = 3052) OR (95% CI)	Mexican American (n = 3342) OR (95% CI)
Model 1[a]				
Area-level poverty				
Quartile 1	Ref.	Ref.	Ref.	Ref.
Quartile 2	1.05 (.92,1.20)	1.06 (.82, 1.37)	0.88 (.68, 1.13)	1.25 (.96,1.64)
Quartile 3	1.27(1.11,1.45)	1.08 (.83,1.40)	1.04 (.81,1.34)	1.38(1.06,1.79)
Quartile 4	1.25(1.10,1.43)	1.37(1.06,1.77)	1.01 (.79,1.29)	1.34 (1.04, 1.72)
Variance in intercept	0.20[e]	0.77[e]	<0.01[e]	<0.01[e]
Model Fit: -2LL	23068.68	9150.08	5326.13	5240.05
Model 2[b]				
Area-level poverty				
Quartile 1	Ref.	Ref.	Ref.	Ref.
Quartile 2	1.06 (.92,1.22)	.99 (.76,1.30)	0.91 (.69, 1.19)	1.27 (.96,1.69)
Quartile 3	1.30(1.12,1.51)	.96 (.72,1.26)	1.09 (.83,1.42)	1.35(1.02,1.79)
Quartile 4	1.24(1.06,1.45)	1.27 (.96,1.68)	1.06 (.81,1.40)	1.37(1.04,1.81)
Variance in intercept	0.22[e]	0.81[e]	<0.01[e]	<0.01[e]
Model Fit: -2LL	21635.73	8618.70	5031.81	4832.66
Model 3[c]				
Area-level poverty				
Quartile 1	Ref.	Ref.	Ref.	Ref.
Quartile 2	1.06 (.92,1.22)	1.00 (.76,1.30)	0.91 (.70, 1.19)	1.27 (.96,1.69)
Quartile 3	1.32(1.13,1.53)	.98 (.74,1.30)	1.07 (.81,1.40)[d]	1.36(1.03,1.80)[d]
Quartile 4	1.27(1.08,1.50)	1.39(1.04,1.86)	1.03 (.77,1.37)[d]	1.38(1.04,1.82)
Prevalence NH black population	.99 (.99,1.00)	.98 (.98, .99)	1.00 (.99,1.00)	.99 (.99,1.00)
Variance in intercept	0.22[e]	0.80[e]	<0.01[e]	<0.01[e]

Variable	Overall poverty distribution	Race/ethnicity-specific Poverty distribution		
	Cardiometabolic dysfunction (n = 10415) OR (95% Ci)	NH white (n = 2756) OR (95% CI)	NH black (n = 3052) OR (95% CI)	Mexican American (n = 3342) OR (95% CI)
Model Fit: -2LL	21634.37	8610.76	5031.06	4832.52

[a] Model 1 includes NHANES Survey Cycle.

[b] Model 2 includes Race/Ethnicity (except race/ethnicity-specific models), Family Income to Poverty ratio, head of household education, cotinine levels, physical activity,nd NHANES survey cycle.

[c] Model 3 includes all variables in model 2 plus area-level prevalence of black population.

[d] Interaction $p < .05$ suggests statistically different from white.

[e] $p < .01$.

Table 4

Mean family income-poverty ratio by area-level SES quartile and race/ethnicity

	Area-level poverty				
	Quartile 1	**Quartile 2**	**Quartile 3**	**Quartile 4**	
	mean (se)	**mean (se)**	**mean (se)**	**mean (se)**	***p* value**
Total population (*n = 10,415*)	3.34 (.03)	2.59 (.02)	1.95 (.02)	1.42 (.02)	<.05
NH white (*n=2756*)	3.53 (.04)[b,c]	2.79 (.04) [b,c]	2.20 (.07) [b,c]	1.60 (.10) [b,c]	<.05
NH black (*n=3052*)	2.61 (.07)[a]	2.29 (.06)[a,c]	1.84 (.04)[a,c]	1.34(.03)[a]	<.05
Mex. Am. (*n=3342*)	2.55 (.07)[a]	1 91 (04)[a,b]	1 54 (03)[a,b]	1 33 (03)[a]	<.05

[a]Significant difference from White ($p < .05$)

[b]Significant difference from Black ($p < .05$)

[c]Significant difference from Mexican American ($p < .05$)