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Original Scholarship

Weathering in Detroit: Place, Race, Ethnicity, and Poverty as Conceptually Fluctuating Social Constructs Shaping Variation in Allostatic Load

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Policy Points:

- Despite 30 years of attention to eliminating population health inequity, it remains entrenched, calling for new approaches.
- Targeted universalism, wellness-based local development, and Jedi Public Health approaches that are community informed, evidence based, and focused on improving everyday settings and diverse lived experiences are important policy directions.
- State and federal revenue transfers are necessary to mitigate the harms of austerity and assure greater equity in fiscal and population health in places like Detroit, Michigan.

Context: US population health inequity remains entrenched, despite mandates to eliminate it. To promote a public health approach of consequence in this domain, stakeholders call for moving from risk-factor epidemiology toward consideration of dynamic local variations in the physiological impacts of structured lived experience.

Methods: Using a community-based, participatory research approach, we collected and analyzed a unique data set of 239 black, white, and Mexican adults

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from a stratified, multistage probability sample of three Detroit, Michigan, neighborhoods. We drew venous blood, collected saliva, took anthropometric measurements, and assayed specimens to measure allostatic load (AL), an indicator of stress-mediated biological dysregulation, linking participants' AL scores and survey responses. In a series of nested Poisson models, we regressed AL on socioeconomic, psychosocial, neighborhood, and behavioral stressors to test the hypothesis that race/ethnicity and poverty-to-income ratio (PIR) are conceptually fluctuating variables whose impacts on AL are sensitive to structured lived experience.

Findings: White and Mexican Detroit participants with PIR < 1 have higher AL than counterparts nationally; black participants in Detroit and nationwide had comparable AL. Within Detroit, disparities by PIR were higher in whites than blacks, with no significant difference by PIR in Mexicans. The size of estimated effects of having PIR < 1 for whites is 58 percentage points greater than that of Mexicans and twice that of blacks.

Conclusions: Structurally rooted unobserved heterogeneity bias threatens the validity of independent main effects interpretations of associations between race/ethnicity, socioeconomic characteristics, or place and health. One-size-fits-all analytic or policy models developed from the perspective of the dominant social group insufficiently address the experiences of diverse populations in specific settings and historical moments; nor do they recognize culturally mediated protective resources residents may have developed against material and psychosocial hardship.

Keywords: allostatic load, racial/ethnic population health equity, poverty, urban, weathering.

Increasingly, investigators of the fundamental causes of population health inequity are looking to variation across diverse population groups in the environmental, material, and psychosocial stressors and hardships that differentially structure their lived experience, and to the collective coping strategies groups employ in their attempts to mitigate them.[1-6] There are calls for research and policy to go beyond the conventional social determinants risk factor epidemiology and, instead, take a more fluid, historicized, and interactive approach for capturing the unobserved heterogeneity in socially patterned life chances and lived experience that may be key to engaging in a "public health of consequence."[4,7-10]

From a "lived experience" perspective, some common practices in social epidemiology are problematic. These include (1) overreliance on conventional and current microlevel socioeconomic indicators as proxies for the full panoply of socially patterned differences in lived experience across groups; (2) failure to construe race/ethnicity as a contextually fluctuating conceptual variable; and (3) preference for nationally representative samples, which are limited for considering heterogeneity in local lived experience. Further, studies that do include measures of place tend to rely on administrative units such as census tracts or Zip codes, without consideration of residents' own assessments of what constitutes their community, or of the possibility that residents with different racial/ethnic identities have different lived experiences in the same geographic places. These differences are likely to include differences in biopsychosocial exposures and impacts. As such, the idea that fundamental social causes must work through biological mechanisms to exert their impacts on population health puts a premium on understanding how variations in lived experience activate health-harmful or health-promoting physiological processes that may explain inequitable distributions of disease or mortality.

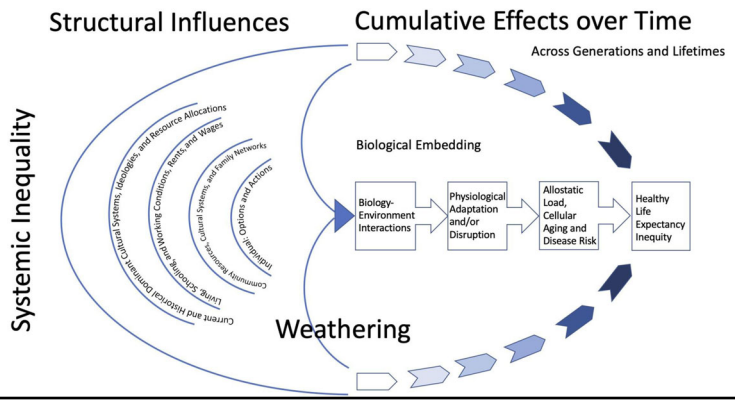
Taking these observations into account, and theoretically informed by the concept of weathering,[11,12] in this study, we sought to examine whether diverse residents of the same neighborhoods exhibited different levels of allostatic load (AL) across race/ethnicity and poverty level. We studied population variation in AL by focusing on a specific setting and time: Detroit, Michigan, between 2008 and 2011. Collaborating with the Healthy Environments Partnership (HEP), we employed a community-based participatory research (CBPR) approach, allowing us to interpret our findings through the understandings of Detroit residents. Primary data collection among residents of three neighborhoods allowed us to explore distinct aspects and experiences of "disadvantage" among white, black, and Mexican/Mexican American residents with low to moderate household incomes. We consider interaction effects of race/ethnicity and poverty status on allostatic load scores—a measure of stress-mediated wear and tear across body systems—to proxy the possibility that poverty in different groups is associated with distinct lived experiences that vary across important biopsychosocial dimensions and crystallize differently to pattern AL scores within the same city.

Weathering Theory

Weathering theory emphasizes health as an emergent capacity of human beings that dynamically develops over intersecting life courses in response to multiple structurally rooted, nested, and contextually fluctuating social, economic, and physical environmental circumstances that impact physiological stress responses, options for individual or collective coping, and biological processes at the molecular and body systems levels.[8,11,12] At the population level, the distribution of morbidity and mortality overall and by age is affected by resources that are allocated inequitably across racialized groups by historic and systemic racism or privilege. These include access to material resources, healthy food, health care, salutary features of the built environment, and toxic environmental exposures. Importantly, weathering theory posits that population health is also the product of social-psychological processes affecting members' sense of belonging or social marginalization. These emanate from and enact racialized ideologies and other core American myths.[2] Their adverse impacts can be buffered by cultural affirmation and social identity safety, which can exist to a degree even in the absence of support or validation from the broader society, if local population groups develop autonomous protections such as shared counternarratives, multigenerational economic risk-pooling strategies and reciprocal caretaking obligations, and access to alternative cultural frameworks to the dominant one that marginalizes them, whether religious or secular.[1,2,13]

As illustrated in Figure 1, weathering recognizes health as dynamic across the full life course as biopsychosocial mechanisms link fundamental social causes[14] and collectively patterned coping strategies and resources to population distributions of health, biological wear and tear, disease, and longevity. Biological age is conceptualized to reflect the cumulative biological impact of lived experiences from conception to current chronological age. In fetal life through adolescence, physical, environmental, and social exposures across windows of human development may have cumulative effects on health trajectories, resulting in increasing health vulnerability for members of marginalized groups.[15] In adults, weathering is intensified by exposure to persistent and multiple stressors—and the high-effort coping they entail. For example, in high-poverty populations, the young adult through middle ages may be a particularly challenging period as family leadership roles are assumed,

Figure 1. Weathering Conceptual Model [Color figure can be viewed at wileyonlinelibrary.com]



responsibilities expand, and individuals contend with competing obligations between work and family and to dependents across generations, all under conditions of material hardship and social exclusion.

Mechanistically, prolonged psychosocial or physical challenges to metabolic homeostasis induced by unremitting exposure to stressors, and the persistent high-effort coping they entail, can accelerate cellular aging; dysregulate or exhaust neuroendocrine, cardiovascular, metabolic, and immune systems; increase general health vulnerability; and promote susceptibility to infectious disease, the early onset of chronic conditions, health-induced disability, and excess death.[12] Based on these health indicators, evidence of weathering has been well documented in the United States for blacks,[8,12,16,17] and also suggested for Latinx immigrants with longer US residence[3] and the poorest urban and rural whites.[8,18]

Allostatic Load

The concept of allostatic load[19,20]—the idea that overexposure to stress hormones can cause wear and tear on important body systems—suggests one plausible biological mechanism for weathering.[12] Empirical evidence from more than two decades of biomedical and social science

research supports the notion that chronic exposure to physical environmental, economic, and psychosocial stressors contributes to the cumulative dysregulation of biological systems, shaping disparities in health and aging.[21,22] Studies of racial/ethnic, socioeconomic, and residential differences in AL within the United States are still relatively few and yield somewhat conflicting results. Still, multiethnic studies of AL indicate that stress-induced biological risk acts as a pathway through which socially constructed hierarchies in the United States, like race and socioeconomic position (SEP), influence persistent differentials in death and disease between majority and minority racialized groups.[23-25]

The National Health and Nutrition Examination Survey (NHANES) offers by far the largest and most nationally representative sample for studies focused on AL disparities.[21] Using NHANES data, Seeman and colleagues found that education and income were each negatively associated with AL independent of age, sex, ethnicity, and lifestyle factors (smoking, physical activity) and that these associations did not vary in ethnic-stratified analyses among oversampled black and Mexican Americans.[26] Geronimus and colleagues observed that independent of poverty, black-white differences in AL are present in all age groups, but were particularly pronounced during middle age.[12] Poor white respondents were less likely to experience high AL than nonpoor black respondents, while black women, whether poor or nonpoor, were found to accrue higher AL scores at younger ages and experience disproportionately higher AL relative to black men and white women.

Also using NHANES data, two studies found that individuals living in socioeconomically disadvantaged census tracts had higher AL burdens than others, independent of measured individual characteristics; although this relationship was consistent across white, black, and Mexican American subgroups,[27] one study found that the relationship between neighborhood SEP and AL was stronger for persons with higher levels of education.[28] Using longitudinal data from the Study of Women Across the Nation (SWAN), a community-based, multiethnic sample of midlife women randomly selected from a diverse, place-based sampling frame, researchers investigated the linkages between a range of psychosocial factors and AL.[29] The authors found that black and low-SEP women had higher AL scores and that measures of discrimination, perceived stress, and hostility partially mediated this relationship.

While AL studies of specific local geographic areas are few, they offer insight on how structurally rooted aspects of residential place may

affect AL, with emerging evidence for interactive influences between residential setting and local population dynamics across racial/ethnic or socioeconomic groups.[30-34] In contrast to findings from NHANES,[35] Peek et al. found that in a population-based sample of adults living in Texas City, Texas, foreign-born Mexicans had significantly *lower* AL scores than white residents, an advantage that persisted even after accounting for health behaviors, acculturation, and time in the United States.[32] The authors attribute this to unmeasured factors associated with the immigrant experience in Texas City, as well as the substantively worse health profile of white residents relative to the NHANES sample. In the same sample, Mair and colleagues found that four chronic stressors—long-term residence in Texas City (which the authors classify as an urban environmental "riskscape"), residential proximity to petrochemical plants, perceived poor neighborhood conditions, and daily hassles—were each linked to elevated AL or AL components.[31]

Studies of other stress-mediated health outcomes also suggest the importance of considering variation in lived experience among racialized groups even in the same city. Studying hypertension, an AL component, Hunte and colleagues found variations among Detroit, Chicago, and the nation that were patterned by race/ethnicity within local areas.[38] In a series of studies of excess mortality across select local areas in 1980, 1990, and 2000, Geronimus and colleagues found the contributions of stress-related chronic disease to excess mortality varied within the same cities and across regions of the country within and across racial/ethnic and poverty groups.[18]

The Setting

Detroit offers an important opportunity to examine the relationship between health, urban disinvestment, racialized population dynamics, and related stressors characteristic of high-poverty urban settings.[39-41] The massive flight of residents and capital from Detroit to its surrounding suburbs over the past six decades significantly eroded the city's tax base and reduced Detroit's population from 1.8 million in 1950 to just 706,640 in 2011 when we completed biomeasure collection.[42] Out-migration of middle- and high-socioeconomic-status white Detroiters has been particularly pronounced, reflected in the proportionate growth of black residents from about 30% in 1960 to about 85% in 2010.[43,44]

Once home to the nation's most affluent black population, Detroit—like other segregated cities in the United States—experienced a resurgence in the number of high-poverty neighborhoods during the first decade of the 21st century when the city also lost a full quarter of its population.[45] As of 2014 about 60% of Detroit's children lived below the poverty threshold—more than any other large US city.[46] Furthermore, the Mexican population, who have maintained a significant presence in Southwest Detroit since the 1940s, grew by approximately 45% between 1950 and 2010 with the post-1965 upsurge in immigration, especially during the 1990s.[47]

Detroit's worsening problems of racialized poverty, disinvestment, and tax base erosion perpetuated by earlier macroeconomic and political restructuring intensified in the wake of the 2008 financial crisis. Chronic revenue losses imposed constraints on the city's government, undermined Detroit as a center of black political power, and reduced its ability to deliver basic public services. Between 1990 and 2013, Detroit reduced its municipal workforce by nearly half.[48] In the 1990s and early 2000s racially targeted subprime loans pervaded Detroit's housing market; between 2005 and 2014 28% of Detroit's mortgageable properties underwent foreclosure, causing property values to decline precipitously.[49] "Austerity urbanism,"[50] or extreme local fiscal retrenchment, culminated in 2013 with state receivership and the subsequent filing of the largest municipal bankruptcy in US history. At the time of data collection, the state's decreased commitment to funding public services in Detroit was manifest in Detroit's renegotiated pay and pensions for the public service workforce, its approximately 78,000 abandoned buildings, scarcity of working residential streetlights, and widespread residential water shutoffs.[51]

Hypotheses

We expect that the pattern of AL across racial/ethnic and socioeconomic groups in Detroit will reflect both the environmental conditions faced by all residents and the variation among groups in experiences that either affirm and protect or stigmatize and threaten social identity.[4,6,13,52-55] On a material level, historical race-conscious beliefs, policies, and practices are fundamental causes of poor urban health because they act to segregate black residents into low-income communities and spur

sustained disinvestment in these areas that ultimately affects all residents.[1] Black, white, Mexican/Mexican American, or other residents of Detroit are likely to experience limited educational and socioeconomic opportunities; overburdened or disrupted social networks; physical environments marked by urban decay, toxic environmental exposures, and poor infrastructure; and high levels of psychosocial distress—any of which could precipitate AL. Put another way, poor white people and Mexican/Mexican Americans living in Detroit could be impacted by the structural racism that ghettoized black people into high-poverty urban areas over the 20th century. Thus, we hypothesize:

Hypothesis 1: *Members of all three racial/ethnic groups in Detroit will have high AL scores relative to national averages*.

We hypothesize that chronic disinvestment, high poverty rates, and spatial stigma will have adverse direct and spillover effects on the lived experience of all Detroit residents whatever their racial/ethnic social identity group.

Hypothesis 2: *We hypothesize interaction effects between race/ethnicity and poverty status on AL score in Detroit*.

The weathering framework posits that poverty is a marker not only for household material disadvantage, but also for how Detroit's racialized history differentially structures current systems of risk pooling, opportunities for cultural affirmation, and exposures to "othering" encounters across racial/ethnic groups. For example, while black residents have suffered the longest history of poverty and marginalization of the three racial/ethnic groups examined here, they also may benefit from greater racial/ethnic density. This density reduces othering encounters, enhances identity safety, and enables dense, autonomous risk-pooling protections, deeply rooted co-ethnic social ties, and shared counternarratives. One implication is that income poverty could impact the health of racialized groups differently, depending on the relative strength of their autonomous social networks and the extent to which they practice reciprocal obligations or a shared sense of collective purpose. Being in poverty would have worse health consequences for population groups who have not developed and relied on such protections than for those who have.

Hypothesis 3: *Neighborhood stressors will be associated with AL scores in absolute terms and in accounting for differences in AL across race/ethnicity × poverty groups (poor or non-poor non-Hispanic whites, non-Hispanic blacks, or Mexicans/Mexican Americans).*

Because we theorize that population differences in AL are expressions of a structurally rooted biopsychosocial process that works through physiological stress processes to wear out body systems, we expect that measured neighborhood stressors will impact AL scores and help to explain any race/ethnicity × poverty interactions on AL we find. Consistent with our theoretical approach, we estimate whether the measured stressors, writ large, account for AL differences within or across populations rather than delineate the precise degree that a given measured stressor mediates Detroit population variation in AL.

Data and Methods

Data Collection

In 2008, the Healthy Environments Partnership, a community-based participatory research partnership, began to field the second wave of a community survey designed to examine and address aspects of Detroit's social and physical environment.[56,57] The study used a stratified, two-stage probability sample of residents aged 25 and older living in three areas of Detroit: Eastside, a segregated high-poverty black community; Northwest, a low- to moderate-income, predominantly black but less segregated community; and Southwest, a high-poverty community with a racial/ethnic mix of residents, including the majority of the city's Mexican population, US-born and immigrant. Southwest residents were oversampled to allow comparisons on the basis of ethnicity. The study sample was designed to observe adequate low to moderate income variation within each of the three predominant racial/ethnic groups in Detroit: black, white, and Mexican/Mexican American.

While collecting a rich set of background and experiential data, the wave 2 HEP Community Survey included only a limited set of anthropometric measures. To generate a comprehensive measure of AL that includes primary mediators and secondary effects, we augmented the

second wave of the HEP survey to include venous blood draw and saliva collection from a subset of participants.

HEP survey participants who expressed interest in the AL portion of the study were given information regarding the purpose of the study and participation requirements. After completing the informed consent process and an in-person interview, 239 people enrolled to participate in biomeasure collection. These enrollees represented 92% of the 259 survey participants we asked to participate, who, in turn, constituted 56% of the full 460 wave 2 participants. After the exclusion of 34 enrollees who did not meet inclusion criteria, the final sample for the current analysis included 205 participants (45 white; 114 black; 46 Mexican/Mexican American).

Blood was drawn according to standard procedures for venous blood collection.[58] Salivary cortisol was collected three times daily over the course of three days (first in the morning right after waking and before brushing or eating, second in the middle of the afternoon, and third just before bed at night). All biospecimen collection tubes and accompanying paperwork were labeled with identification numbers only, and all lab personnel were blind to participants' identities and sociodemographic characteristics. Data collection was completed in 2011. Blood samples were analyzed by the Central Ligand Assay Satellite Services (CLASS) laboratory at the University of Michigan's School of Public Health. Saliva samples were logged and stored at −80° C in the CLASS laboratory until being shipped on dry ice to the University of Trier Psychobiology Laboratory for analysis.

Outcome Variable: Allostatic Load

We construct our AL score to provide a cumulative measure of stress-mediated multisystem physiological dysregulation,[21,22,24,59] which we conceptualize as the manifestation of biological weathering.[12] Although no standard approach to AL measurement exists in the literature,[22] we draw from prior research to employ an AL algorithm comprising 14 biomarkers designed to assess functioning across the inflammatory, cardiovascular, metabolic, and hypothalamic-pituitary-adrenal (HPA) axis systems: albumin, body mass index (BMI), diurnal cortisol slope, C-reactive protein (CRP), dehydroepiandrosterone-sulfate (DHEA-S), diastolic and systolic blood pressures (DBP, SBP), glycated hemoglobin (HbA1c), high- and low-density lipid levels (HDL, LDL), homocysteine,

interleukin 6 (IL-6), total cholesterol, and triglycerides. Anthropometric biomeasures (DBP, SBP, BMI) were obtained from physical examination; serum and saliva-based measures were obtained from laboratory results. Our inclusion of both primary mediators (DHEA-S, cortisol, IL-6) and secondary outcomes is intended to more adequately capture the multiple interacting pathways that affect major bodily systems concurrently.[60] To our knowledge this precise configuration of AL indicators has not been used in prior research,[21,22] as our study is one of the first to employ such a comprehensive battery measure within a geographically situated, predominantly nonwhite US sample of broad age range.[31,32]

To assess the sensitivity of our findings to alternative specifications of AL, we construct two count-based summary measures explored in prior literature. Following a standard approach,[20,29,33,34,61,62] we assign a binary (0/1) variable for each biomarker, with a value of one identifying participants in the highest-risk quartile based on the distribution of that biomarker in the sample (<25th percentile for albumin, HDL, and DHEA-S and >75th percentile for all others). We generate a continuous measure of AL by summing across all biomarkers to obtain scores with a potential range of 0 to 14, where higher scores indicate greater biological weathering. Additionally, we construct a binary (0/1) AL outcome, with a value of one indicating a high AL score, defined as a score of four or above. We chose this high-score threshold based on the mean scores in our study population and also conducted sensitivity analyses using other plausible cut points (3 and 5), which yielded qualitatively similar patterns.

In the Detroit analyses (Hypotheses 2 and 3), Detroit sample-based quartiles were used rather than clinical cut points or high-risk quartiles obtained from more representative study samples because the Detroit-based sample demonstrates high-risk compression relative to these thresholds. While the use of sample-based cut points may underestimate absolute risk levels in the sample, the use of sample-based thresholds more aptly captures sample variation and allows us to examine how contextually specific experience shapes the distribution of risk within our population of interest. Appendix Table A1 displays Detroit sample-based high-risk thresholds for each biomarker.

To explore Hypothesis 1, we used data from the 2009-2010 NHANES to compare AL scores in the HEP sample with those observed in a nationally representative sample. NHANES collects 10 of the 14 biomarkers that make up our AL score: albumin, BMI, CRP, DBP and SBP,

HbA1c, HDL and LDL, total cholesterol, and triglycerides. Data collection procedures for these variables are similar to those employed in the Detroit sample, although some variables required unit conversion to ensure comparability. To have the data resemble our sample in terms of race/ethnicity, we limit the NHANES data set to respondents identifying as non-Hispanic white, non-Hispanic black, or Mexican/Mexican American. Further, we limit the sample to match the age ranges of the Detroit sample by race/ethnicity and sex. Only participants with complete data for all ten biomarkers are included in the data set ($n = 1{,}447$).

To provide proper comparisons across data sets, for Hypothesis 1 only, we recalculate a truncated AL score for the Detroit sample using only the ten variables common to both data sets. Instead of using quartiles from the Detroit sample to dichotomize each biomarker, we used quartile values drawn from the NHANES, weighting for the complex sampling design. In addition, the NHANES was weighted to reflect the age and sex distributions of the black HEP sample for the race/ethnicity comparisons.

In all analyses, participants taking medication for diabetes, hypertension, or cholesterol were classified as high-risk for HbA1c, blood pressure, or total cholesterol, respectively. This decision is based on our theoretical model, which provides that those taking medication (and thus diagnosed with a chronic disease) have already experienced systematic deterioration. Further, taking medication to control the condition does not alleviate any aspects of the social environment that may have helped precipitate the condition.

Explanatory Variables

As explicated in the next section, Poisson models were run, yielding predictions of AL scores stratified by race/ethnicity and the groups denoting the interaction between race/ethnicity and poverty-to-income ratio (PIR). All models are conditioned on age and sex. *Age* was measured in years, calculated as the difference between the respondent's date of birth and the date of the study interview. *Sex* was self-reported, with all participants identifying as either male or female; in each model, males provide the reference group. A categorical variable indicates participants' highest level of education: (1) less than a high school degree (referent category), (2) high school degree or general equivalency diploma, (3) some college or an associate degree, and (4) bachelor's degree or higher.

To study variation in AL across intersections of race, place, and socioeconomic position, we construct a categorical variable based on the interaction between race/ethnicity and poverty status. Participants identified their race/ethnicity as either non-Hispanic black, non-Hispanic white, or Mexican/Mexican American (hereafter referred to as white, black, and Mexican). PIR was calculated based on the respondent's household income relative to the federal poverty level designated for the participant's household size; PIR values greater than or equal to 1 indicate incomes greater than the household-size-specified federal poverty level, and values less than 1 indicate incomes below it. The resulting variable yields six categories: (1) nonpoor white, (2) poor white, (3) nonpoor black, (4) poor black, (5) nonpoor Mexican, (6) poor Mexican. Following weathering theory, we interpret any differential in AL according to this interaction of place, race/ethnicity, and poverty-to-income ratio as a proxy for unobserved heterogeneity in lived experience among the various intersectional groups.

Under the weathering theory, some candidate contributors to the overall unobserved heterogeneity are also included in our models. These variables are intended to reflect exposure to, and coping with, structurally rooted biopsychosocial stressors: education, safety stress, everyday unfair treatment, physical environment stress, negative social interactions, overall neighborhood satisfaction, feelings of anger or despair, and smoking—all risk factors for stress-related physiological wear and tear.

Safety stress was assessed based on three questions concerning how often participants worry about safety in their home or neighborhood, with response options ranging from 1 (never) to 5 (always). *Perceived unfair treatment* was measured using five items from the Everyday Unfair Treatment scale (e.g., how often are you treated with less courtesy or respect than others, how often are you threatened or harassed), modified from the 1995 Detroit Area Study.[63,64] Frequency of perceived unfair treatment ranged from 1 (never) to 5 (always).

Participants' perception of their neighborhood physical environment was assessed by a series of seven questions designed to capture both negative and positive physical features of their neighborhood. Higher scores on this scale reflected better-perceived neighborhood physical environment, with more positive and fewer negative features. We adapted the Negative Social Interactions scale from Schuster, Kessler, and Aseltine[65] to quantify the frequency with which friends and family members either

(1) made too many demands on the respondent or (2) criticized the respondent or the respondent's behavior. Responses ranged from 1 (never) to 5 (always).

Neighborhood satisfaction is operationalized by a five-point Likert scale indicating the respondent's level of agreement to a single item: "I would move out of this neighborhood if I could." Consistent with our orientation toward lived experience, the neighborhood-level questions (neighborhood physical environment, neighborhood satisfaction) did not define "neighborhood" explicitly. Participants responded according to their own definition.

Anger out was measured using a four-item scale regarding how often a person argued with others, struck out, said nasty things, or lost their temper while feeling angry or mad.[66] *Hopelessness* was measured using level of agreement to two items from the Beck Hopelessness Scale: "The future seems to be hopeless, and I can't believe things are changing for the better" and "I feel it is impossible for me to reach the goals I strive for."[67] Higher scores for anger or hopelessness reflected more frequent expressions of anger or stronger agreement with hopelessness statements, respectively.

Using survey items derived from previous studies,[68,69] we designated participants who reported never regularly smoking tobacco products as the reference category relative to current regular smokers and to former regular smokers.

Statistical Analysis (Hypotheses 2 and 3)

We regressed AL scores on the aforementioned variables in a series of nested Poisson models. We used the Poisson model because the allostatic load is a count variable with scores running from 0 to 14. The model takes the form

$$\mathrm{Ln}\left(\mathrm{E}\left[\mathrm{AL}|\mathrm{X}\right]\right) = \theta' X,$$

where X is a set of variables that influence AL and Θ is a set of coefficients to be estimated. These coefficients have the same interpretation they would in a log linear regression; in a log linear regression a one-unit change in X is associated with a Θ unit change in Ln(E[A]). For small values of Θ, estimates can be interpreted as percent changes in AL. Thus, for example, a coefficient of 0.023 on age implies that each additional

year of age is associated with a 2.3% increase in the expected allostatic load score.

Since previous findings have found differential effects of income on health outcomes by race/ethnicity, we stratified the race/ethnicity groups by PIR as interaction variables for the analysis. All models controlled for race/ethnicity, the interaction of race/ethnicity and PIR, age, and sex:

$$\begin{aligned}\mathrm{Ln}\left(\mathrm{E}\left[\mathrm{AL}\right]\right) = {} & \beta_0 + \beta_1 \mathrm{Black} + \beta_2 \mathrm{Mexican} + \beta_3 \left(\mathrm{White} \times \mathrm{PIR}\right) \\ & + \beta_4 \left(\mathrm{Black} \times \mathrm{PIR}\right) \\ & + \beta_5 \left(\mathrm{Mexican} \times \mathrm{PIR}\right) + \gamma_1 \mathrm{age} + \gamma_2 \mathrm{female}\end{aligned}$$

In this model, β_1 and β_2 represent the effect of being nonpoor black or Mexican, relative to being a nonpoor white, while β_3, β_4, and β_5 compare poor whites, poor blacks, and poor Mexicans to their nonpoor counterparts. An alternative parameterization would include a poverty dummy and would drop one of the interaction terms. Such a model would alter the interpretation of the coefficients on the interaction terms, but would not change the implications of the regression.

Although the proportion of missing variables underlying the AL score was minimal, we used Markov Chain Monte Carlo multiple imputation procedures in order to conduct a complete case analysis.[70] We imputed variables underlying the AL score (i.e., albumin, BMI, diurnal cortisol slope, CRP, DHEA-S, DBP, SBP, HbA1c, HDL, LDL, homocysteine, IL-6, total cholesterol, and triglycerides) by creating a conditional distribution of these missing variables derived from all of the underlying AL score variables as well as the explanatory variables. We imputed the data five times for this analysis.

Only one explanatory variable (participants' perceptions of their neighborhood physical environment) contained missing values, with 8% of its values missing. To allow analysis of the full sample, we imputed this variable by fitting a multivariate ordinary least squares regression model and replaced the missing values with the model's estimates. The explanatory variables in this imputation model were the other explanatory variables.[71] Given the minimal number of missing values among our explanatory variables, imputing these missing values for this one variable averted the need to exclude observations from our analysis. All imputations and analyses were conducted using STATA IC version 15.1.

Results

Sample Description

As shown in Table 1, the household income distribution is far more comparable across black, white, and Mexican participants in the Detroit sample and substantially lower than in the nation as a whole. For example, the percentage of whites, blacks, or Mexicans reporting annual incomes of <$10,000 was 38%, 37%, and 35%, respectively, in Detroit, compared to 6%, 15%, and 9%, respectively, in the nation. Race/ethnicity-specific poverty rates among Detroit study participants aged 25 to 64 years are more than double the national rate for black or Mexican participants, and seven times the national rate for whites.

The mean AL scores in the Detroit sample, using the 14-item algorithm and Detroit cut points, standardized to the age and sex distributions of the black participants in the sample, were as follows: whites 3.5, blacks 4.1, and Mexicans 3.4. The percentage with high AL scores was 43% for whites, 60% for blacks, and 45% for Mexicans.

Table 2 displays these means further stratified by poor and nonpoor. For black and white participants in the Detroit sample, being poor is associated with higher AL, although the difference among black participants is smaller than among white participants. In contrast, for Mexican participants, mean allostatic load score is 6.5% *lower* among the poor compared to the nonpoor.

As illustrated in Figure 2, looking at the percentage with high scores (defined as AL scores ≥ 4), these trends are more pronounced. White nonpoor are 39 percentage points less likely to have high AL scores than the white poor, and black nonpoor are 20 percentage points less likely to have high AL scores than the black poor, while Mexican nonpoor are 10 percentage points *more* likely to have high AL scores than Mexicans in poverty.

Hypothesis 1: *Within racial/ethnic group, Detroit participants will have higher AL than in a national sample*

To gauge how Detroit sample means compare to national samples, Table 3 compares NHANES to Detroit sample means, based on the 10-item AL algorithm possible in the NHANES sample, using NHANES cut points, and adjusted in all cases for the age and sex distribution of the black Detroit sample. *Hence, the means for the Detroit sample*

Table 1. Socioeconomic Characteristics of White, Black, and Mexican Respondents[a]

	% of Population, by Race					
	National ACS Estimates			Detroit Sample		
	White	Black	Mexican	White	Black	Mexican
Annual household income						
<$10,000	6%	15%	9%	38%	37%	35%
$10,000-$24,999	15%	23%	21%	29%	31%	37%
$25,000-$49,999	24%	27%	30%	22%	22%	26%
$50,000-$99,999	32%	25%	28%	11%	11%	2%
$100,000+	23%	10%	12%	0%	0%	0%
Poverty rates[b]						
Ages 25 to 64 years	8%	20%	19%	57%	49%	52%
Highest education, ages 25+						
Less than high school	10%	19%	38%	33%	21%	50%
College or above	31%	19%	13%	20%	9%	4%

[a] National American Community Survey (ACS) estimates for non-Hispanic white, black, and Hispanic/Latino US populations, 2008-2010, compared to Detroit participants.

[b] As measured by poverty-to-income ratio less than 1.

Table 2. Mean Allostatic Load Score in Detroit Sample Stratified by Race/Ethnicity and Poverty Status, Age, and Sex Standardized to the Black Detroit Population

Race/Ethnicity	Household Poverty	*n*	Mean AL	% With AL Score ≥4
White	PIR > 1 (nonpoor)	24	2.4	16%
	PIR < 1 (poor)	21	3.9	55%
Black	PIR > 1 (nonpoor)	60	3.2	40%
	PIR < 1 (poor)	54	4.0	60%
Mexican	PIR > 1 (nonpoor)	23	3.1	41%
	PIR < 1 (poor)	23	2.9	31%

Abbreviations: AL, allostatic load; PIR, poverty-to-income ratio.

here differ from those reported earlier because limitations of the NHANES data require us to use an abbreviated algorithm and a national set of cut points. In addition to means, we also compare percentages with high AL scores (≥4) across the two samples.

The mean score point estimates for each racial/ethnic group are higher in the Detroit sample than in the NHANES sample. Differences in means among whites ($p < 0.04$) and for Mexicans ($p < 0.04$) are statistically significant. Whites and Mexicans have a higher percentage with a high AL score in the Detroit sample than in the NHANES sample, with a 24 percentage-point difference among whites ($p < 0.04$) and an 18 percentage-point difference among Mexicans ($p < 0.02$). The point estimates for blacks were 5 percentage points lower in Detroit than in the NHANES sample, but this was not a statistically significant difference.

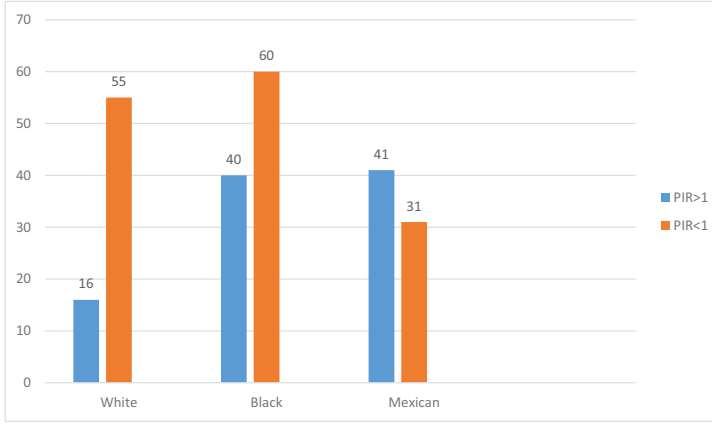
These findings provide general support for Hypothesis 1, that the Detroit sample is more likely to have higher AL than the NHANES sample. The comparability in mean and percentage high AL scores for black participants across the samples is a notable exception.

Table 3. Comparison of Mean Scores and Percent High Scores Between Detroit and NHANES Samples Adjusted for Age and Sex, NHANES Cut Points

	Mean AL Score			% With High AL (Score ≥4)		
	Detroit Sample	NHANES Sample	*p* value	Detroit Sample	NHANES Sample	*p* value
White	3.9	3.2	0.04	71%	47%	0.04
Black	4.2	4.1	ns	62%	67%	ns
Mexican	4.3	3.6	0.04	75%	57%	0.02

Abbreviations: AL, allostatic load; NHANES, National Health and Nutrition Examination Survey; ns, not significant.

Figure 2. Percent High Allostatic Load Score by Race/Ethnicity and PIR, Detroit Sample [Color figure can be viewed at wileyonlinelibrary.com]



Poor defined as poverty-to-income ratio (PIR) < 1. *High allostatic load* defined as allostatic load score ≥ 4 based on 14-item algorithm and Detroit high-risk thresholds.

The differences in means between the Detroit and NHANES samples are likely to be driven at least in part by different income distributions between the two samples, whereby the distribution for the Detroit sample is lower and more compressed than that of the national sample. The differences in means also would reflect other unobserved differences between living in Detroit and being a representative sample member of the United States. To attenuate the degree to which different income distributions, per se, explain the higher scores among the Detroit sample compared to the NHANES sample, Table 4 further stratifies each racial/ethnic group by poverty status. Both the Detroit and NHANES sample members reflected here live in households with poverty-to-income ratios of <1.

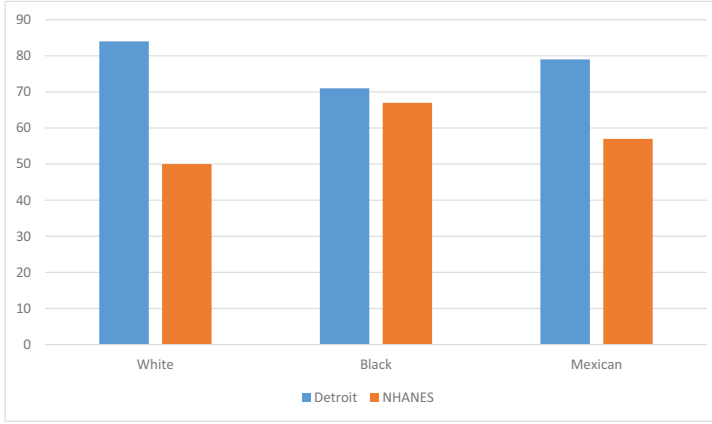
Limiting the sample to those in poverty, all groups show higher mean scores and higher percentages of members with high AL scores if they are in the Detroit sample compared to the NHANES sample, although the

Table 4. Comparison of Mean Scores and Percent High Scores Between Detroit and NHANES Samples Adjusted for Age and Sex, NHANES Cut Points, PIR < 1

	Mean AL Score			% With High AL (Score ≥ 4)		
	Detroit Sample	NHANES Sample	*p* value	Detroit Sample	NHANES Sample	*p* value
White, PIR < 1	4.7	3.7	0.07	84%	50%	0.01
Black, PIR < 1	4.2	4.1	ns	71%	67%	ns
Mexican, PIR < 1	4.6	3.8	0.10	79%	50%	0.02

Abbreviations: AL, allostatic load; HEP, Healthy Environments Partnership; NHANES, National Health and Nutrition Examination Survey; PIR, poverty-to-income ratio; ns, not significant.

Figure 3. Percent of Poor With High Allostatic Load Scores in Detroit and NHANES Samples, by Race/Ethnicity [Color figure can be viewed at wileyonlinelibrary.com]



Abbreviation: NHANES, National Health and Nutrition Examination Survey.
Poor defined as poverty-to-income ratio (PIR) < 1. *High allostatic load* defined as allostatic load score ≥ 4 based on 10-item algorithm and NHANES high-risk thresholds.

differences in means for black participants are statistically insignificant and much smaller than for white ($p < 0.07$) or Mexican ($p < 0.10$) participants. Considering the percentage of each racial/ethnic group with high AL scores, poor black Detroit participants have a 4 percentage-point higher rate of high AL than poor blacks in NHANES, an insignificant difference, while poor whites ($p < 0.01$) and poor Mexicans ($p < 0.02$) have substantially higher percentages in the Detroit sample than in the NHANES sample.

At 84%, poor white participants in Detroit have the highest rate of high AL scores of any group—about 30 percentage points higher than poor whites or poor Mexicans in the nation, 17 percentage points higher than poor black participants in Detroit, and 5 percentage points higher than poor Mexicans in Detroit (Figure 3). Relative to whites and

Mexicans, blacks had the lowest percentage with a high AL in Detroit, yet the highest in NHANES.

The race-specific differences between members of the Detroit and NHANES samples are robust to comparisons of only those with PIR < 1 and, thus, not explained by the substantially higher poverty rates in Detroit compared to the United States. This finding suggests unobserved heterogeneity in aspects of racialized lived experience other than income poverty that affect AL, either by exacerbating it for poor whites compared to blacks or Mexicans, or by being protective for blacks and poor Mexicans relative to poor whites in Detroit.

Regression Analyses: Tests of Hypotheses 2 and 3

Moving to a Poisson regression framework and focusing on variation in the Detroit sample only, using the 14-item AL algorithm, we consider Hypotheses 2 and 3 using estimates reported in Table 5. To consider interactions between race/ethnicity and poverty status (Hypothesis 2), in Model 1 of Table 5, we report estimates of differences in AL scores for nonpoor black and Mexican participants relative to nonpoor white participants in the first two rows of estimates; and in the following three rows of estimates, differences in allostatic load scores for poor versus nonpoor within each race/ethnicity group. Reported estimates are estimates from Poisson regressions using individuals' AL scores as the dependent variable. Reported estimates can be interpreted as the proportion increase in AL score associated with the explanatory variable.

Among the nonpoor, black participants, on average, have a 28% higher AL score than white participants ($p < 0.04$). The estimate for nonpoor Mexican compared to nonpoor white participants is 26% higher ($p < 0.12$), trending toward but not achieving statistical significance at conventional levels.

Among white participants, those who are poor have a 48% higher mean allostatic load score than the nonpoor ($p < 0.01$). The black poor are 23% worse off than the black nonpoor ($p = 0.01$). The point estimate for poor Mexican participants suggests they have a 10% *lower* mean AL score than the nonpoor, but this is not statistically significantly different from the nonpoor.

One might imagine that the differences across the race and ethnicity groups in terms of the estimated effect of poverty on AL could be

Table 5. Effects of Race/Ethnicity × Poverty on Allostatic Load Score, Controlling for Age and Sex and Additional Covariates

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Coef.	*p* value	Coef.	*p* value	Coef.	*p* value	Coef.	*p* value	Coef.	*p* value
Race [ref. = white]										
Black	0.278	0.04	0.272	0.05	0.298	0.03	0.295	0.04	0.296	0.04
Mexican	0.260	0.12	0.229	0.18	0.296	0.09	0.293	0.10	0.305	0.09
Race × Poor										
White × Poor	0.479	<0.01	0.444	0.01	0.433	0.01	0.428	0.01	0.416	0.02
Black × Poor	0.232	0.01	0.203	0.03	0.206	0.03	0.205	0.04	0.195	0.05
Mexican × Poor	−0.096	0.58	−0.127	0.46	−0.128	0.46	−0.133	0.45	−0.141	0.42
Age	0.023	<0.001	0.023	<0.001	0.022	<0.001	0.022	<0.001	0.022	<0.001
Female	−0.071	0.38	−0.071	0.39	−0.057	0.52	−0.058	0.51	−0.046	0.61

Reported estimates are from Poisson regression models and can be interpreted as the proportion increase in the allostatic load (AL) score associated with the explanatory variable.
The AL score is based on 14 biomarkers of inflammatory, cardiovascular, metabolic, and hypothalamic-pituitary-adrenal axis system functioning. All models are conditioned on age and sex. $N = 205$.
Model 2 adds controls for education (less than high school, high school degree, some college; referent = college degree).
Model 3 adds controls for education and psychosocial stress (safety stress, everyday unfair treatment, physical environment, negative social interactions) and neighborhood satisfaction (Likert scale).
Model 4 adds controls for education, psychosocial stress, neighborhood satisfaction, and response type (anger, hopelessness).
Model 5 adds controls for education, psychosocial stress, neighborhood satisfaction, response type, and smoking status (current smoker, former smoker; referent = never smoker).

accounted for by the fact that there are income distribution differences across groups among the poor and, especially, the nonpoor. While such differences exist (the white nonpoor are better off, on average, than the nonwhite nonpoor), when we controlled for income to needs (PIR) as a continuous variable in addition to the dichotomous PIR > 1 variable already in the model, it did not affect the estimated coefficients on the variables already included, suggesting the somewhat different income distributions by race/ethnicity are not a source of bias.

By subtracting the estimated effect of being poor from comparisons of black relative to white participants, and Mexican relative to white participants, we can estimate whether there is racial/ethnic group variation in the impact of being a poor Detroit resident on AL. Doing so, we find the poverty effect for blacks is roughly half as large as the poverty effect for whites (0.23 vs. 0.48; $p < 0.10$). The poverty effect for Mexicans is actually estimated to be negative—that is, Mexicans in poverty have lower AL scores than those not in poverty; and the difference between the poverty effect for white and Mexican participants is significant ($p < 0.01$). In fact, the effect of poverty for white participants is 58 percentage points larger than for Mexicans.

In sum, these estimates do suggest an interaction between race/ethnicity and poverty group within the Detroit sample. The adverse impact of poverty is substantially higher for white than for black or Mexican participants, suggesting the groups face different contingencies of social identity that go beyond income poverty in the context of Detroit residence to affect AL. The largest difference between poor and nonpoor was seen in white participants, with a smaller difference among black participants. Among Mexicans, the poor and nonpoor were statistically no different from each other, with point estimates suggesting the possibility that the poor might have lower mean allostatic load scores than the nonpoor in this setting. Compared to the white poor, the Mexican poor had a 32% *lower* mean AL score ($p < 0.07$); in fact, the Mexican poor had AL scores that were not statistically significantly higher than the white nonpoor. These findings suggest that limiting measurement to independent main effects of race/ethnicity, poverty, or place masks unobserved heterogeneity in lived experience at the intersections of race, place, and poverty.

Of what might such unobserved heterogeneity be composed? We found the stressors we measured to have little effect on AL, as seen in Models 2–5 in Table 5. Only having a less than high school education

and wanting to move out of the neighborhood are marginally statistically significantly associated with AL. Taking account of all measured covariates mildly increases the mean AL score for black and Mexican nonpoor compared to the white nonpoor, mildly decreases the negative association of poverty to AL for whites and blacks, and mildly increases the positive association of poverty for Mexicans. Still, all general race/ethnicity × PIR relationships with AL remain robust to the presence of the measured stressors.

Discussion

The clearest takeaway from this study is that *unobserved heterogeneity bias* is a major threat to the validity of universal interpretations of associations between race/ethnicity, socioeconomic characteristics, or place of residence and allostatic load. Such findings call for greater nuance when interpreting findings from national samples employing independent main effects measures of race/ethnicity, poverty, or place alone, if we are to meaningfully enhance understanding of population health inequity or how to eliminate it. To the extent that unobserved heterogeneity reflects differences in racial/ethnic identity contingencies that vary with lived experience in different times or places, study findings suggest that race/ethnicity and income poverty are best characterized as contextually fluctuating social constructs whose health impacts conventional socioeconomic variables appear insufficient to reflect.[7,72]

For white and Mexican participants, but not black participants, mean AL scores and the probability of having a high AL score were higher in the Detroit sample than in the NHANES sample, as anticipated by Hypothesis 1. Even after restricting these comparisons to only those with PIR < 1 across the two samples, dramatic differences between the local and national samples for white and Mexican participants remained. This was especially the case for whites with poverty-income ratios below 1, suggesting that unobserved differences in lived experience among white people residing in high-poverty urban areas go beyond their greater likelihood of income-poverty compared to nationwide averages.

Like other racial/ethnic groups, whites are a distinct and heterogeneous group facing unique contingencies of racial identity in specific settings. Being white and poor in the face of popular narratives advancing white racial expectations of conventional socioeconomic success

through individual effort, or living in low-income areas of Detroit with its racialized spatial stigma as an "urban black inner city," may be particularly stressful and health compromising for whites when combined with reduced likelihood of health-enhancing collective social resources more reliably associated with black American and Mexican origin populations. Current study findings along with recent findings by Geronimus and colleagues[73] showing growing inequities in life expectancy between less- and more-educated whites over the past three decades reinforce the need to understand variation among whites in lived experience across settings and historical moments, rather than including whites in studies primarily as a referent. Further, the objective evidence of stress-mediated biological deterioration among impoverished Detroit whites we provide based on laboratory assays suggests the need for greater consideration of the contribution of chronic socially structured physiological impacts on health in investigations of growing excess death rates among the least educated whites, in addition to the current popularized focus on existential despair culminating in drug overdose or suicide death.[73]

Nonpoor Mexicans had higher AL relative to nonpoor white or black participants. In contrast to comparisons for poor and nonpoor white and black participants, after adjusting for age and sex, differences in the percentage with high AL scores between nonpoor and poor Mexicans were not statistically significant and the trend favored the poor. Unique stressors among Mexicans in Detroit that cut across the poor and nonpoor may, in part, reflect that data collection coincided with a period of heightened surveillance by immigration officials in Southwest Detroit. As a border community less than 100 miles from the Canadian border, Detroit residents who are Latinx encounter Immigration and Customs Enforcement (ICE) surveillance with fewer restrictions than those living farther from a border. This is an example of a population-specific contingency of social identity that may impact health for specific groups and at specific times or places and not others.[74] While one might imagine such effects would pertain only to undocumented Mexicans in Detroit, studies have shown that fears of immigration enforcement can have adverse health effects on US-born co-ethnics.[74] Our small sample size precluded disaggregating the Mexican population by nativity for statistical analyses, but we note that Mexicans in the nonpoor group were disproportionately US born, while those in the poor group were disproportionately foreign born. If immigrants experience a heavier burden of fear of ICE enforcement, the extent to which differential AL scores between poor

and nonpoor Mexicans favor poor Mexicans might be dampened in our findings.

Despite the added stressor of ICE surveillance, poor Mexicans had lower AL than either poor whites or poor blacks. This finding is consistent with Peek and colleagues' findings on AL in Texas City, Texas,[32] and with Geronimus and colleagues' findings comparing telomere lengths of poor and nonpoor blacks, whites, and Mexicans in Detroit.[8] These findings cast into sharp relief the importance of considering social and psychological health determinants, operating beyond the influence of conventionally measured individual or household income. For example, multiple studies document that recently arrived low-income Mexican immigrants often have significantly better health than other US-born, low-income population groups.[75] This initial Mexican immigrant health advantage is reduced with years of residence in the United States and disappears altogether in subsequent generations.[3,76] The relatively lower AL scores among the poor Mexican group may in part reflect this immigrant health advantage and fewer years of residence in the United States.

Length of residence in the United States may erode this initial immigrant advantage through multiple mechanisms. Qualitative studies conducted in Detroit within the Mexican population by Viruell-Fuentes have reported evidence suggesting that as new immigrants reside in the United States longer, or as their children are raised in the United States, they increasingly engage and negotiate majority-white social institutions in daily interactions.[6,77] The more prevalent negative prejudices and stereotypes encountered through these interactions increase the degree to which immigrants are acutely aware of and attuned to dehumanizing ideologies associated with US racial hierarchies that may activate physiological stress processes.[6,77-79] To the extent that Mexicans in the lower-income group have fewer disconfirming interactions with discriminatory majority institutions, or have encountered those institutions for relatively shorter periods of time, the health-damaging effects of such interactions may not yet be reflected in AL scores. To the extent that those with higher income have more frequent encounters with those same institutions, or have encountered them for longer periods of time, their effects may be reflected in the higher AL scores in the nonpoor Mexican group. The finding that poor Mexicans reported feeling less anger or hopelessness than nonpoor Mexicans is consistent with this perspective.

Encounters with discriminatory institutions may also have differential impacts for Mexicans contingent upon social identities and networks. Eighty percent of poor Mexicans in Detroit reported that Spanish was the most commonly spoken language in their homes, regardless of nativity. Most resided in Southwest Detroit, an area with a substantial Latinx population, restaurants and services catering to Spanish-speaking populations, and many identity-affirming symbols, institutions, and cultural events. As first suggested by James,[80] speaking Spanish in the home may afford some health protection, offering an alternative and affirming sociocultural framework to the dominant marginalizing one.[4]

Finally, potential health-enhancing value of ethnic enclaves for immigrant populations has been hypothesized to operate through market economy dynamics that structure social interactions. Low-wage immigrant enclave economies may promote social well-being and health in two distinct, but integrally related ways. First, such communities provide opportunities for residents to develop and apply skills appropriate for niche labor markets particular to Mexican immigrant populations (food industry, domestic work, construction, yard maintenance, etc.) in ways that are particularly salient and confirming to group members.[81,82] Second, these practices reduce the necessity of engaging labor markets in dominant society, which may be more lucrative, but also more disconfirming.[83,84] Together, fewer encounters with and shorter duration of exposure to discriminatory institutions in the United States; greater access to health-protective, affirming identities; and residing and working within an ethnic enclave are all factors that may contribute to the trend observed here toward lower AL scores among poor Detroit Mexicans relative to poor blacks or poor whites in this sample. Future research exploring these potential protective factors is warranted to better understand these patterns.

Among black participants, we saw less differentiation in AL scores by PIR than in the white and Mexican groups, as well as less within-group variation between the national and local samples. A growing literature finds that being high on the socioeconomic ladder is a porous shield from structurally racist lived experiences that intensify allostatic load among blacks. In addition, much research suggests the separation between poor and nonpoor US blacks in everyday life is less marked than between poor and nonpoor US whites.[2,85,86] US blacks tend to have greater residential proximity across socioeconomic gradients owing to residential segregation. Additionally, poor and nonpoor blacks are more likely to be

members of the same families and social networks, practice reciprocal obligations, or have similar experiences of cycling between low and moderate incomes.[2,86-89] Given deep cross-class affective ties, a strong collective ethos, elastic household boundaries, and shared resources, PIR measured at the individual-household level may not accurately reflect differences in material hardship, life stressors, or pooled cultural strengths among US blacks.

Moreover, structural racism in the United States, predominately anti-black, is historically entrenched. Over the past century, urban ghettoization and disinvestment has provided a critical staging ground for anti-black racism. Our focus on Detroit—one example of that staging ground—may help explain why only small differences were found in AL between blacks in Detroit and in NHANES, while whites and Mexicans showed large differences. Blacks are subjected to racist structural violence wherever they reside in the United States and also disproportionately reside in the most underinvested urban areas. The large nationwide vs. Detroit disparities seen within whites and Mexicans, in contrast, may reflect that only those whites and Mexicans residing in proximity to blacks, particularly in high-poverty urban centers, would experience the residual institutional impacts of structural anti-*black* discrimination. Meanwhile, Detroit blacks may have developed protective systems that better insulate them from harms specific to anti-black racism. This possibility is consistent with the finding of Linnenbringer and colleagues that among California women diagnosed with breast cancer, black women residing in neighborhoods with higher percentages of black residents had significantly lower odds of being diagnosed with triple negative breast cancer, a more aggressive and deadly subtype of the disease.[36] While the etiology of breast cancer subtypes is not well understood, higher levels of prediagnostic AL have recently been associated with poorer prognostic features among black women with breast cancer.[37]

Neither the stressors and response styles we measured nor smoking behavior accounted for the unobserved heterogeneity implied by interactions among race/ethnicity, poverty-to-income ratio, and place. Perhaps this finding reveals misalignment between dominant ideas about what are the major stressors and health risks in low-income racial minority urban areas and those held by the populations confronting the contingencies of this lived experience. Although a number of conventional stressors were measured in our data, additional social risks and

insults specific to Detroit and likely to vary across racial/ethnic groups may be at play. For instance, notions of white racial privilege or entitlement combined with unmet expectations of economic success, not measured in our study, could be particularly health-harmful for poor whites in Detroit.[8,90] In contrast, the alternative sociocultural orientations for Mexicans and blacks referenced previously may, on balance, be health protective for racial-minority populations. And, perhaps, in the Detroit context, specific maladaptive unhealthy individual behavioral responses such as smoking may have marginal health costs beyond other, more fundamental and insidious, structurally rooted, chronic biopsychosocial stressors. Such possibilities require continued empirical study.

On a methodological note, our findings suggest that the common practice of estimating mean AL scores runs the risk of obfuscating important differences that may exist in the population percentage exhibiting high scores. Using mean scores is statistically more efficient, and it evades the question of the most appropriate cut point to determine a high score. Yet, as we found here, diverse population groups with similar mean scores boasted considerably different percentages with multiple morbidities, arguably a conceptually clearer indicator of weathering and a greater cause for concern.[91]

Limitations

We were unable to consider possible variation in exposure to environmental toxins across racialized groups. By focusing on groups living in proximity to each other in a single city, such bias would be smaller than in national samples; however, to the extent that racialization and racialized social bias influence access to labor market segments, it is possible that different racialized groups face different toxic physical and social exposures at work. Prior studies focused on the Detroit metropolitan area provide evidence that structural environmental racism places racialized groups in Detroit and surrounding areas at increased health risk due to toxic environmental exposure. There is also evidence of variation in exposure to air pollutants across neighborhoods within Detroit[92] and that these variations are associated with excess burden of childhood asthma, high blood pressure, and excess cardiopulmonary mortality.[93-97] Importantly, none of these studies tested for variation in race/ethnicity and poverty distributions across neighborhoods, leaving open the question

of whether the pollution-associated health outcomes were mediated or moderated by race/ethnicity and poverty, key foci of our study.

Another limitation is that we were unable to include more affluent households in the Detroit metropolitan area. The compression of our sample's household income distribution at the low end and its inclusion of only Detroit residents made it more statistically efficient and homogeneous in key respects. Further, relative income homogeneity across racial/ethnic groups allowed us to focus on whites who are as poor as the blacks and Mexicans to which they were being compared, a rarity when analyzing metropolitan, state, or national samples. While we consider this a central strength of the study, it nonetheless limited possible comparisons within groups or between the local and national samples.

We would have benefited from a larger sample. Linkage to the HEP survey data placed a ceiling on our *n*, limiting it to those who completed the second wave of the HEP survey and agreed to participate in the biomeasure collection portion of our study. In this primary biomeasure collection effort, our sample size was further restricted by the labor intensity and challenges of obtaining saliva and blood samples from a hard-to-reach, relatively transient, and understandably mistrustful population. (See Caldwell et al.[98] and Pearson and Geronimus[99] for discussions of this issue in Detroit.)

As with many studies of AL, the algorithm we used when making national comparisons (Hypothesis 1) was data driven—that is, limited by the items available in NHANES. In contrast, within-Detroit analyses were based on a primary data collection effort, enabling us to construct a more expansive algorithm drawing on a state-of-the-art understanding of the proper component indicators of AL.

Notwithstanding study limitations, our findings suggest the importance of recognizing the existence and impact of structurally rooted unobserved heterogeneity in life chances and lived experiences of diverse social identity groups on an objective measure of health. This can be true even among those who reside in the same geographic place and have comparable incomes, and even compared to those with the same racial/ethnic identity or poverty level nationwide. Although we were unable to illuminate the specific or measurable aspects that account for racialized health inequity in AL in Detroit, our findings leave little question that such heterogeneity needs to be understood far better than more typical social epidemiological risk-factor approaches allow.

Implications for the Ways Forward

Although methodological efforts we made to focus on a specific setting and employ a community-based participatory approach are suggestive of the limits of more conventional social epidemiological statistical analyses for investigating population health inequity, we do not mean to imply that studies such as ours are likely to quantify precise single-variable pathways to health inequities that could be addressed in a vacuum. Indeed, a core implication of our work highlights that one-size-fits-all conceptual/theoretical models, assessment tools, and interventions developed from the perspective of a single demographic, particularly the dominant social group, are insufficient to reflect the lived experiences of diverse population groups or mitigate the negative health impact of marginalized social status or residence in historically disinvested places.[2,4]

We see the unanswered questions that remain in this study as a critical open door to a new kind of inquiry for population health inequity research that considers variation in lived experience for racialized and impoverished groups across historical moments, and within geographic and institutional settings. Such an approach cultivates empathy across groups and respect for local knowledge, enhancing the chance to illuminate routes to a public health of consequence. Such research would probe the local to identify the magnitude of unobserved heterogeneity across racialized groups and deconstruct what specifically differs in their lived experiences in the same local area that sets their health outcomes apart from each other and from national averages for their sociodemographic group. In addition to differential access to income, health care, or healthy foods and physical environments, we expect some of these differences will be in racially coded cues to biopsychosocial stress arousal that trade on entrenched stereotypes; in the power dynamics among local population groups; and in the more or less adaptive coping strategies culturally available to different groups in the face of hardship.

Our work highlights the importance of considering social difference across multiple dimensions of identity including race/ethnicity, nativity, and socioeconomic gradients within a specific place context. Toward that end, study findings encourage moving beyond community-based samples to active engagement of residents of communities disproportionately affected by structural inequities as they manifest within particular historical, social, and economic contexts.[100,101]

Such work encompasses community-based study designs that are informed by historical and contemporary social and economic contexts, by ethnographic and other qualitative research approaches, and by the active and sustained participation of community groups in the development, implementation, and interpretation of research conducted within local contexts. The establishment of equitable, community-driven, and/or CBPR partnerships among community, academic, and public health practice entities can foster a more nuanced understanding of social determinants of health as they manifest locally. Equally important, CBPR partnerships build knowledge about key strategies for structural and policy change to promote health equity.[102,103]

In the realms of interventions, programs, and policies, our work suggests that social differences across multiple dimensions of identity must be simultaneously addressed if policy makers expect success in alleviating the health effects of urban disinvestment or other political and physical manifestations of structural racism. A well-documented impediment to integrating this range of health equity options is the false dichotomy frequently drawn between policies targeting the specific needs of socially vulnerable populations and universal policies designed to benefit all segments of the broader population. Policies targeting the needs of marginalized groups are often viewed through the egalitarian meritocratic lens of the American Creed and perceived as special treatment for the "undeserving poor." They, thus, frequently elicit majority population resentment and political resistance. Universal policies, by comparison, assume a global norm per the dominant viewpoint that reflects positive bias for that perspective while imposing negative bias against social minorities, thus perpetuating social inequality. Targeted universalism covers the needs of a broad constituency yet stands to leverage equity to address needs of vulnerable populations.[104-106] For example, Canadian Healthy Child programs have successfully implemented a targeted universal approach by contacting all families with children to provide services from pregnancy to school entry. Families found to be facing greater challenges are matched with trained home visitors who provide multifaceted support for up to three years contingent upon need.[105]

Study findings are consistent with the premise that the stress-mediated health impact of being a member of a given racial/ethnic, poverty, or place of residence group varies with the contingencies of racial/ethnic and class identity in one's particular daily round. Consistent with this premise, we also recommend interventions that are

informed by "Jedi Public Health" (JPH) principles to reduce or disrupt repeated physiological stress process activation that fuels population weathering.[13] The objective of JPH is to ensure that stigmatized social identity is not chronically central to who people are in everyday life, and never central in high-stakes performance settings. This goal points to interventions that create identity-safe settings, including by changing adverse situational identity contingencies, the environmental cues that signal them, and the narratives used to interpret them. Some JPH approaches are straightforward, inexpensive, and highly scalable.[13]

Our findings also point to ambitious policies that restructure the ecologies of local areas and highlight equity and investment in doing so.[107] Engaging with community partners who have linkages to ongoing social movements and decision makers creates opportunities to work collaboratively to address fundamental drivers of health inequities. Community-led movements for worker protections, housing rights, and environmental justice address underlying structural determinants as they are manifest on a local level, influencing racial, ethnic, and socioeconomic equity.[1,2,108,109] Examples include Vital Brooklyn,[110] a New York state initiative emphasizing "wellness-based" economic and environmental development. The program works in concert with local anchor institutions, community agencies, and labor unions to improve material living conditions in central Brooklyn and enhance equity across the domains of education, housing, workforce development, public safety, health, and health care. Other promising approaches include community benefits policies, such as those recently implemented in Detroit,[111] which are intended to empower long-term residents to dictate (and enforce) the terms of public and private investments to meet the needs of the local community. Such policies can be used to assure that current residents do not disproportionately experience displacement, pollutants, or adverse health effects associated with development efforts, thus favoring a more equitable paradigm[112] for redevelopment without gentrification and displacement. Although local governments have access to a wide range of existing policy tools (e.g., affordable housing trust funds, inclusionary zoning ordinances, regional tax-base sharing) that can redistribute and prioritize resources for spaces and groups who have borne the brunt of structural racism, state and federal revenue transfers to localities like Detroit are necessary to mitigate the harms of austerity and assure greater equity in fiscal and population health.

References

1. Geronimus AT. To mitigate, resist, or undo: addressing structural influences on the health of urban populations. *Am J Public Health*. 2000;90(6):867-872. https://doi.org/10.2105/ajph.90.6.867.
2. Geronimus AT, Thompson JP. To denigrate, ignore, or disrupt: racial inequality in health and the impact of a policy-induced breakdown of African American communities. *Du Bois Rev*. 2004;1(2):247-279. https://doi.org/10.1017/S1742058X04042031.
3. Kaestner R, Pearson JA, Keene D, Geronimus AT. Stress, allostatic load, and health of Mexican immigrants. *Soc Sci Q*. 2009;90(5):1089-1111. https://doi.org/10.1111/j.1540-6237.2009.00648.x.
4. Pearson JA. Can't buy me whiteness: new lessons from the Titanic on race, ethnicity, and health. *Du Bois Rev*. 2008;5(1):27-47. https://doi.org/10.1017/0S1742058X0808003X.
5. Schulz AJ, Zenk SN, Israel BA, Mentz G, Stokes C, Galea S. Do neighborhood economic characteristics, racial composition, and residential stability predict perceptions of stress associated with the physical and social environment? Findings from a multilevel analysis in Detroit. *J Urban Health*. 2008;85(5):642-661. https://doi.org/10.1007/s11524-008-9288-5.
6. Viruell-Fuentes EA. Beyond acculturation: immigration, discrimination, and health research among Mexicans in the United States. *Soc Sci Med*. 2007;65(7):1524-1535. https://doi.org/10.1016/j.socscimed.2007.05.010.
7. Reed AL, Chowkwanyun M. Race, class, crisis: the discourse of racial disparity and its analytical discontents. *Social Regist*. 2012;48:149-175. https://papers.ssrn.com/sol3/papers.cfm?abstract_id=1970445. Accessed November 22, 2019.
8. Geronimus AT, Pearson JA, Linnenbringer E, et al. Race-ethnicity, poverty, urban stressors, and telomere length in a Detroit community-based sample. *J Health Soc Behav*. 2015;56(2):199-224. https://doi.org/10.1177/0022146515582100.
9. Galea S, Vaughan R. A public health of consequence. *Am J Public Health*. 2016;106(1):10-11. https://doi.org/10.2105/AJPH.2015.302954.
10. McBride CM, Comeau DL, Mastin AE, Cooper HL. A public health of consequence: shifting the cultural narrative from churning grants to a scholarship of consequence. *Am J Public Health*.

2019;109(11):1535-1538. https://doi.org/10.2105/AJPH.2019.305266.

11. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis*. 1992;2(3):207-221.
12. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health*. 2006;96(5):826-833. https://doi.org/10.2105/AJPH.2004.060749.
13. Geronimus A, James SA, Destin M, et al. Jedi public health: co-creating an identity-safe culture to promote health equity. *SSM Popul Health*. 2016;2:105-116.
14. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav*. 1995;1:80-94. https://doi.org/10.2307/2626958.
15. Jones NL, Gilman SE, Cheng TL, Drury SS, Hill CV, Geronimus AT. Life course approaches to the causes of health disparities. *Am J Public Health*. 2019;109(S1):S48-S55. https://doi.org/10.2105/AJPH.2018.304738.
16. Geronimus AT, Bound J, Keene D, Hicken M. Black-white differences in age trajectories of hypertension prevalence among adult women and men, 1999–2002. *Ethn Dis*. 2007;17(1):40-49.
17. Holzman C, Eyster J, Kleyn M, et al. Maternal weathering and risk of preterm delivery. *Am J Public Health*. 2009;99(10):1864-1871. https://doi.org/10.2105/AJPH.2008.151589.
18. Geronimus AT, Bound J, Colen CG. Excess black mortality in the United States and in selected black and white high-poverty areas, 1980–2000. *Am J Public Health*. 2011;101(4):720-729. https://doi.org/10.2105/AJPH.2010.195537.
19. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci*. 1999;896(1):30-47. https://doi.org/10.1111/j.1749-6632.1999.tb08103.x.
20. Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation—allostatic load and its health consequences: MacArthur studies of successful aging. *Arch Intern Med*. 1997;157(19):2259-2268.
21. Beckie TM. A systematic review of allostatic load, health, and health disparities. *Biol Res Nurs*. 2012;14(4):311-346. https://doi.org/10.1177/1099800412455688.
22. Juster RP, McEwen BS, Lupien SJ. Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neurosci*

Biobehav Rev. 2010;35(1):2-16. https://doi.org/10.1016/j.neubiorev.2009.10.002.

23. Crimmins EM, Kim JK, Seeman TE. Poverty and biological risk: the earlier "aging" of the poor. *J Gerontol A Biol Sci Med Sci*. 2009;64(2):286-292. https://doi.org/10.1093/gerona/gln010.
24. Seeman T, Epel E, Gruenewald T, Karlamangla A, McEwen BS. Socio-economic differentials in peripheral biology: cumulative allostatic load. *Ann N Y Acad Sci*. 2010;1186(1):223-239. https://doi.org/10.1111/j.1749-6632.2009.05341.x.
25. Nelson KM, Reiber G, Kohler T, Boyko EJ. Peripheral arterial disease in a multiethnic national sample: the role of conventional risk factors and allostatic load. *Ethn Dis*. 2007;17(4):669-675.
26. Seeman T, Merkin SS, Crimmins E, Koretz B, Charette S, Karlamangla A. Education, income and ethnic differences in cumulative biological risk profiles in a national sample of US adults: NHANES III (1988-1994). *Soc Sci Med*. 2008;66(1):72-87. https://doi.org/10.1016/j.socscimed.2007.08.027.
27. Bird CE, Seeman T, Escarce JJ, et al. Neighbourhood socioeconomic status and biological "wear and tear" in a nationally representative sample of US adults. *J Epidemiol Community Health*. 2010;64(10):860-865. https://doi.org/10.1136/jech.2008.084814.
28. Finch BK, Do DP, Heron M, Bird C, Seeman T, Lurie N. Neighborhood effects on health: concentrated advantage and disadvantage. *Health Place*. 2010;16(5):1058-1060. https://doi.org/10.1016/j.healthplace.2010.05.009.
29. Upchurch DM, Stein J, Greendale GA, et al. A longitudinal investigation of race, socioeconomic status, and psychosocial mediators of allostatic load in midlife women: findings from the Study of Women's Health Across the Nation. *Psychosom Med*. 2015;77(4):402-412. https://doi.org/10.1097/PSY.0000000000000175.
30. King KE, Morenoff JD, House JS. Neighborhood context and social disparities in cumulative biological risk factors. *Psychosom Med*. 2011;73(7):572-579. https://doi.org/10.1097/PSY.0b013e318227b062.
31. Mair CA, Cutchin MP, Peek MK. Allostatic load in an environmental riskscape: the role of stressors and gender. *Health Place*. 2011;17(4):978-987. https://doi.org/10.1016/j.healthplace.2011.03.009.
32. Peek MK, Cutchin MP, Salinas JJ, et al. Allostatic load among non-Hispanic whites, non-Hispanic blacks, and people of Mexican origin: effects of ethnicity, nativity, and acculturation. *Am*

J Public Health. 2010;100(5):940-946. https://doi.org/10.2105/AJPH.2007.129312.

33. Schulz AJ, Mentz G, Lachance L, Johnson J, Gaines C, Israel BA. Associations between socioeconomic status and allostatic load: effects of neighborhood poverty and tests of mediating pathways. *Am J Public Health*. 2012;102(9):1706-1714. https://doi.org/10.2105/AJPH.2011.300412.
34. Schulz AJ, Mentz G, Lachance L, et al. Do observed or perceived characteristics of the neighborhood environment mediate associations between neighborhood poverty and cumulative biological risk? *Health Place*. 2013;24:147-156. https://doi.org/10.1016/j.healthplace.2013.09.005.
35. Crimmins EM, Kim JK, Alley DE, Karlamangla A, Seeman T. Hispanic paradox in biological risk profiles. *Am J Public Health*. 2007;97(7):1305-1310. https://doi.org/10.2105/AJPH.2006.091892.
36. Linnenbringer E, Geronimus AT, Davis KL, Bound J, Ellis L, Gomez SL. Associations between breast cancer subtype and neighborhood socioeconomic and racial composition among black and white women. *Breast Cancer Res Treat*. 2020;180(2):437-447. https://doi.org/10.1007/s10549-020-05545-1.
37. Xing CY, Doose M, Qin B, et al. Prediagnostic allostatic load as a predictor of poorly differentiated and larger sized breast cancers among black women in the Women's Circle of Health Follow-Up Study. *Cancer Epidemiol Biomarkers Prev*. 2020;29(1):216-224. https://doi.org/10.1158/1055-9965.EPI-19-0712.
38. Hunte HE, Mentz G, House JS, et al. Variations in hypertension-related outcomes among blacks, whites and Hispanics in two large urban areas and in the United States. *Ethn Dis*. 2012;22(4):391-397.
39. Schulz A, Williams D, Israel B, et al. Unfair treatment, neighborhood effects, and mental health in the Detroit metropolitan area. *J Health Soc Behav*. 2000;41(3):314-332. https://doi.org/10.2307/2676323.
40. Schulz AJ, Kannan S, Dvonch JT, et al. Social and physical environments and disparities in risk for cardiovascular disease: the Healthy Environments Partnership conceptual model. *Environ Health Perspect*. 2005;113(12):1817-1825. https://doi.org/10.1289/ehp.7913.
41. Sugrue TJ. *The Origins of the Urban Crisis: Race and Inequality in Postwar Detroit*. Princeton, NJ: Princeton University Press; 1996.
42. US Census Bureau. 2011 American Community Survey, Social Explorer Tables, Total population; 2011. Social Ex-

plorer website. https://www.socialexplorer.com/data/ACS2011/metadata/?ds=SE&table=A00001. Accessed July 31, 2019.

43. Gibson C, Jung K. *Historical Census Statistics on Population Totals by Race, 1790 to 1990, and by Hispanic Origin, 1970 to 1990, for Large Cities and Other Urban Places in the United States*. US Census Bureau Working Paper No. 76, Table 23; 2005. https://www.census.gov/content/dam/Census/library/working-papers/2005/demo/POP-twps0076.pdf. Accessed September 23, 2020.
44. US Census Bureau. 2010 Census, Table P5, Hispanic or Latino Origin by Race; 2010. Social Explorer website. https://www.socialexplorer.com/data/C2010/metadata/?ds=SF1&table=P0050. Accessed July 22, 2020.
45. Jargowsky P. *Concentration of Poverty in the New Millennium: Changes in the Prevalence, Composition, and Location of High-Poverty Neighborhoods*. Rutgers, NJ: Century Foundation and Rutgers Center for Urban Research and Education; 2013.
46. Annie E; Casey Foundation. *The 2014 Kids Count Data Book*. Baltimore, MD: Annie E. Casey Foundation; 2014. https://www.aecf.org/resources/the-2014-kids-count-data-book/. Accessed October 5, 2014.
47. Alvarado RV, Alvarado SY. *Mexicans and Mexican Americans in Michigan*. Lansing, MI: Michigan State University Press; 2003.
48. Sugrue TJ. The rise and fall of Detroit's middle class. *New Yorker*. July 22, 2013. https://www.newyorker.com/news/news-desk/the-rise-and-fall-of-detroits-middle-class. Accessed October 5, 2014.
49. Deng L, Seymour E, Dewar M, Manning Thomas J. Saving strong neighborhoods from the destruction of mortgage foreclosures: the impact of community-based efforts in Detroit, Michigan. *Hous Policy Debate*. 2018;28(2):153-179. https://doi.org/10.1080/10511482.2017.1331366.
50. Peck J. Austerity urbanism: American cities under extreme economy. *City*. 2012;16(6):626-655. https://doi.org/10.1080/13604813.2012.734071.
51. UN Office of the High Commissioner for Human Rights. Detroit: disconnecting water from people who cannot pay—an affront to human rights, say UN experts. Press release. June 25, 2014. https://www.ohchr.org/EN/NewsEvents/Pages/DisplayNews.aspx?NewsID=14777&LangID=E. Accessed October 6, 2014.
52. James SA. John Henryism and the health of African-Americans. *Cult Med Psychiatry*. 1994;18(2):163-182. https://doi.org/10.1007/bf01379448.

53. Bécares L, Nazroo J, Stafford M. The buffering effects of ethnic density on experienced racism and health. *Health Place*. 2009;15(3):700-708. https://doi.org/10.1016/j.healthplace.2008.10.008.
54. Halpern D, Nazroo J. The ethnic density effect: results from a national community survey of England and Wales. *Int J Soc Psychiatry*. 1999;46(1):34-46. https://doi.org/10.1177/002076400004600105.
55. Viruell-Fuentes EA, Miranda PY, Abdulrahim S. More than culture: structural racism, intersectionality theory, and immigrant health. *Soc Sci Med*. 2012;75(12):2099-2106. https://doi.org/10.1016/j.socscimed.2011.12.037.
56. LeBrón AM, Schulz AJ, Mentz G, et al. Impact of change over time in self-reported discrimination on blood pressure: implications for inequities in cardiovascular risk for a multi-racial urban community. *Ethn Health*. 2018;21:1-19. https://doi.org/10.1080/13557858.2018.1425378.
57. Schulz AJ, Zenk SN, Kannan S, Israel BA, Koch MA, Stokes C. 2005. Appendix C: selected new and revised items included in the HEP survey after input from the steering committee or survey subcommittee, focus group themes, or pilot testing of existing items. In: Israel BA, Eng E, Schulz AJ, Parker E, eds. *Methods for Conducting Community Based Participatory Research for Health*. San Francisco, CA: Jossey-Bass; 2005:402-406.
58. Centers for Disease Control and Prevention (CDC). *NHANES 2005–2006 Laboratory Procedures Manual*. Atlanta, GA: Centers for Disease Control and Prevention; 2005. https://wwwn.cdc.gov/nchs/data/nhanes/2005-2006/manuals/lab.pdf. Accessed November 22, 2019.
59. Seeman TE, McEwen BS, Rowe JW, Singer BH. Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proc Nat Acad Sci*. 2001;98(8):4770-4775. https://doi.org/10.1073/pnas.081072698.
60. McEwen BS. Biomarkers for assessing population and individual health and disease related to stress and adaptation. *Metabol*. 2015;64(3):S2-S10. https://doi.org/10.1016/j.metabol.2014.10.029.
61. Crimmins EM, Johnston M, Hayward M, Seeman T. Age differences in allostatic load: an index of physiological dysregulation. *Exp Gerontol*. 2003;38(7):731-734. https://doi.org/10.1016/s0531-5565(03)00099-8.
62. Salazar CR, Strizich G, Seeman TE, et al. Nativity differences in allostatic load by age, sex, and Hispanic background from the Hispanic Community Health Study/Study of Latinos. *SSM Popul*

Health. 2016;2:416-424. https://doi.org/10.1016/j.ssmph.2016.05.003.

63. Jackson JS, Knight KM, Rafferty JA. Race and unhealthy behaviors: chronic stress, the HPA axis, and physical and mental health disparities over the life course. *Am J Public Health*. 2010;100(5):933-939. https://doi.org/10.2105/AJPH.2008.143446.
64. Williams DR, Yu Y, Jackson JS, Anderson NB. Racial differences in physical and mental health: socio-economic status, stress and discrimination. *J Health Psychol*. 1997;2(3):335-351. https://doi.org/10.1177/135910539700200305.
65. Schuster TL, Kessler RC, Aseltine RH. Supportive interactions, negative interactions, and depressed mood. *Am J Community Pyschol*. 1990;18(3):423-438. https://doi.org/10.1007/bf00938116.
66. Spielberger D, Johnson E, Russel S, Crane R, Jacobs Q, Warden T. The experience and expression of anger: construction and validation of an anger expression scale. In: Cheaney M, Rosenman R, eds. *Anger and Hostility in Cardiovascular and Behavioral Disorders*. New York, NY: Hemisphere; 1985:5-30.
67. Beck AT, Weissman A, Lester D, Trexler L. The measurement of pessimism: the hopelessness scale. *J Consult Clin Psychol*. 1974;42(6):861-865. https://doi.org/10.1037/h0037562.
68. Frazier EL, Franks AL, Sanderson LM. Behavioral Risk Factor data. In: Centers for Disease Control and Prevention, ed. *Using Chronic Disease Data: A Handbook for Public Health Practitioners*. Atlanta, GA: US Department of Health and Human Services; 1992.
69. Gentry EM, Kalsbeek WD, Hogelin GC, et al. The behavioral risk factor surveys: II. Design, methods, and estimates from combined state data. *Am J Prev Med*. 1985;1(6):9-14.
70. Li KH. Imputation using Markov chains. *J Stat Comput Simul*. 1988;30(1):57-79. https://doi.org/10.1080/00949658808811085.
71. Little RJA. Regression with missing X's: a review. *J Am Stat Assoc*. 1992;87(420):1227-1237. https://doi.org/10.2307/2290664.
72. Brubaker R. *Ethnicity Without Groups*. Cambridge, MA: Harvard University Press; 2004.
73. Geronimus AT, Bound J, Waidmann TA, Rodriguez J, Timpe B. Weathering, drugs and whack-a-mole: fundamental and proximate causes of widening educational inequity in U.S. life expectancy by sex and race, 1990–2015. *J*

Health Soc Behav. 2019;60(2):222-239. https://doi.org/10.1177/0022146519849932.

74. Novak N, Geronimus AT, Martinez-Cardoso A. Change in birth outcomes among infants born to Latina mothers after a major immigration raid. *Int J Epid*. 2017;3:839-849. https://doi.org/10.1093/ije/dyw346.
75. Markides KS, Eschbach K. Aging, migration, and mortality: current status of research on the Hispanic paradox. *J Gerontol B Psychol Sci Soc Sci*. 2005;60(Special Issue 2):S68-S75. https://doi.org/10.1093/geronb/60.special_issue_2.s68.
76. Collins JJ, Papacek E, Schulte NF, Drolet A. Differing postneonatal mortality rates of Mexican-American infants with United-States-born and Mexico-born mothers in Chicago. *Ethn Dis*. 2001;11(4):606-613. https://doi.org/10.1023/A:1015464207740.
77. Viruell-Fuentes EA, Schulz AJ. Toward a dynamic conceptualization of social ties and context: implications for understanding immigrant and Latino health. *Am J Public Health*. 2009;99(12):2167-2175. https://doi.org/10.2105/AJPH.2008.158956.
78. Bostean G, Andrade FC, Viruell-Fuentes EA. Neighborhood stressors and psychological distress among US Latinos: measuring the protective effects of social support from family and friends. *Stress Health*. 2019;35(2):115-126. https://doi.org/10.1002/smi.2843.
79. LeBrón AM, Schulz AJ, Mentz GB, Israel BA, Stokes CA. Social relationships, neighbourhood poverty and cumulative biological risk: findings from a multi-racial US urban community. *J Biosoc Sci*. 2019;51(6):799-816. https://doi.org/10.1017/S002193201900004X.
80. James SA. Racial and ethnic differences in infant mortality and low birth weight: a psychosocial critique. *Ann Epidemiol*. 1993;3(2):130-136. https://doi.org/10.1016/1047-2797(93)90125-n.
81. Jiobu RM. Ethnic hegemony and the Japanese of California. *Am Sociol Rev*. 1988;53(3):353-367. https://doi.org/10.2307/2095644.
82. Waldinger R. The ethnic enclave debate revisited. *Int J Urban Reg Res*. 1993;17(3):444-452. https://doi.org/10.1111/j.1468-2427.1993.tb00232.x.
83. Waldinger R, Bailey T. The continuing significance of race: racial conflict and racial discrimination in construction. *Polit Soc*. 1991;19(3):291-323.

84. Waldinger RL, Lichter MI. *How the Other Half Works: Immigration and the Social Organization of Labor*. Berkeley, CA: University of California Press; 2003.
85. Heflin CM, Pattillo M. Poverty in the family: race, siblings, and socioeconomic heterogeneity. *Soc Sci Res*. 2006;35(4):804-822. https://doi.org/10.1016/j.ssresearch.2004.09.002.
86. Pattillo-McCoy M. *Black Picket Fences: Privilege and Peril Among the Black Middle Class*. Chicago, IL: University of Chicago Press; 1999.
87. Stack CM. *All Our Kin: Strategies for Survival in a Black Community*. New York: Basic Books; 1974.
88. Burton LM, Whitfield KE. Weathering toward poorer health in later life: comorbidity in low-income urban families. *Public Policy Aging Rep*. 2003;13(3):8-13. https://doi.org/10.1093/ppar/13.3.13.
89. Hicks-Bartlett S. Between a rock and a hard place: the labyrinth of working and parenting in a poor community. In: Danziger S, Lin C, eds. *Coping With Poverty*. Ann Arbor, MI: University of Michigan Press; 2000:27-52.
90. Siddiqi A, Sod-Erdene O, Hamilton D, McMillan Cottom T, Darity W Jr. Growing sense of social status threat and concomitant deaths of despair among whites. *SSM Popul Health*. 2019;9:100449. https://doi.org/10.1016/j.ssmph.2019.100449.
91. Sturmberg JP, Bennett JM, Martin CM, Picard M. "Multimorbidity" as the manifestation of network disturbances. *J Eval Clin Pract*. 2016;23(1):199-208. https://doi.org/10.1111/jep.12587.
92. Schulz AJ, Mentz GB, Sampson N, et al. Race and the distribution of social and physical environmental risk: a case example from the Detroit metropolitan area. *Du Bois Rev*. 2016;13(2):285-304. https://doi.org/10.1017/S1742058X16000163.
93. Li S, Batterman S, Wasilevich E, Elasaad H, Wahl R, Mukherjee B. Asthma exacerbation and proximity of residence to major roads: a population-based matched case-control study among the pediatric Medicaid population in Detroit, Michigan. *Environ Health*. 2011;10(1):34. https://doi.org/10.1186/1476-069X-10-34.
94. Li S, Batterman S, Wasilevich E, et al. Association of daily asthma emergency department visits and hospital admissions with ambient air pollutants among the pediatric Medicaid population in Detroit: time-series and time-stratified case-crossover analyses with threshold effects. *Environ Res*. 2011;111(8):1137-1147. https://doi.org/10.1016/j.envres.2011.06.002.

95. Dvonch JT, Kannan S, Schulz AJ, et al. Acute effects of ambient particulate matter on blood pressure: differential effects across urban communities. *Hypertension*. 2009;53(5):853-859. https://doi.org/10.1161/HYPERTENSIONAHA.108.123877.
96. Schulz AJ, Mentz GB, Sampson NR, Dvonch JT, Reyes AG, Izumi B. Effects of particulate matter and antioxidant dietary intake on blood pressure. *Am J Public Health*. 2015;105(6):1254-1261. https://doi.org/10.2105/AJPH.2014.302176.
97. Le HQ, Batterman SA, Wirth JJ, et al. Air pollutant exposure and preterm and term small-for-gestational-age births in Detroit, Michigan: long-term trends and associations. *Environ Int*. 2012;44:7-17. https://doi.org/10.1016/j.envint.2012.01.003.
98. Caldwell WB, Reyes AG, Rowe Z, Weinert J, Israel BA. Community partner perspectives on benefits, challenges, facilitating factors, and lessons learned from community-based participatory research partnerships in Detroit. *Prog Community Health Partnersh*. 2015;9(2):299-311. https://doi.org/10.1353/cpr.2015.0031.
99. Pearson J, Geronimus AT. A practical guide to biological primary data collection in an impoverished urban setting: illuminating structural and social influences on population health inequity. *SAGE Research Methods Cases*. 2018. https://doi.org/10.4135/9781526444554.
100. Cacari-Stone L, Minkler M, Freudenberg N, Themba M. CBPR for health equity policy making. In: Wallerstein N, Duran B, Oetzel J, Minkler M, eds. *Community-Based Participatory Research for Health*. 3rd ed. San Francisco, CA: Jossey-Bass; 2018:277-291.
101. Minkler M, Freudenberg N. From community-based participatory research to policy change. In: Fitzgerald HE, Burack C, Seifer S, eds. *Handbook of Engaged Scholarship: The Contemporary Landscape*. Vol. 2. East Lansing, MI: Michigan State University Press; 2010:275-294.
102. Israel BA, Coombe CM, Cheezum RR, et al. Community-based participatory research: a capacity-building approach for policy advocacy aimed at eliminating health disparities. *Am J Public Health*. 2010;100(11):2094-2102. https://doi.org/10.2105/AJPH.2009.170506.
103. Minkler M. Linking science and policy through community-based participatory research to study and address health disparities. *Am J Public Health*. 2010;100(1-1):S81-S87. https://doi.org/10.2105/AJPH.2009.165720.
104. Powell JA. *Racing to Justice: Transforming Our Conceptions of Self and Other to Build an Inclusive Society*. Bloomington, IN: Indiana University Press; 2012.

105. Healthy Child Manitoba. *2012 Report of Manitoba's Children and Youth*. Winnipeg, MB: Healthy Child Manitoba; 2012. https://www.gov.mb.ca/healthychild/publications/hcm_2012report.pdf. Accessed June 9, 2020.
106. Powell JA, Menendian S, Reece J. *The Importance of Targeted Universalism*. Washington, DC: Poverty & Research Action Council; 2009.
107. Lewis J, Hernandez D, Geronimus AT. Weatherization as energy justice: addressing racial inequities through investments in people and places. *Energy Efficiency*. 2019. https://doi.org/10.1007/s12053-019-09820-z.
108. Freudenberg N, Franzosa E, Chisholm J. New approaches for moving upstream: how state and local health departments can transform practice to reduce health inequalities. *Health Educ Behav*. 2015;42(1):46S-56S. https://doi.org/10.1177/1090198114568304.
109. Schulz AJ, Mehdipanah R, Chatters L, Reyes AG, Neblett E, Israel BA. Moving health education & behavior upstream: lessons from COVID-19 for addressing structural drivers of health inequities. *Health Educ Behav*. 2020. https://doi.org/10.1177/1090198120929985.
110. The Vital Brooklyn Initiative. New York State website. https://www.ny.gov/transforming-central-brooklyn/vital-brooklyn-initiative-0. Accessed August 25, 2019.
111. Community benefits ordinance. City of Detroit website. https://detroitmi.gov/departments/planning-and-development-department/design-and-development-innovation/community-benefits-ordinance. Accessed June 6, 2020.
112. Sands D. Fighting for better outcomes in Delray. *Michigan Night Light*. April 9, 2014. http://www.michigannightlight.com/people/SimoneSagovac.aspx. Accessed June 6, 2020.

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Appendix

Table A1. High-Risk Thresholds for Each Component Biomarker of the AL Score

Biomarker	High-Risk Threshold
Albumin	<4.30 g/dL
Body mass index	>35.48
Cortisol	>−0.03 nmol/L/hr
CRP	>9.08 mg/L
DHEA-S	<65 μg/dL
DBP	>90 mmHg
HDL	<46 mg/dL
HbA1c	>6%
Homocysteine	>17.18 μmol/L
IL-6	>4.09 pg/mL
LDL	>145 mg/dL
SBP	>143 mmHg
Total cholesterol	>229.50 mg/dL
Triglycerides	>159 mg/dL

Abbreviations: CRP, C-reactive protein; DHEA-S, dehydroepiandrosterone-sulfate; DBP, diastolic blood pressure; HDL, high-density lipid level; HbA1c, glycated hemoglobin; IL-6, interleukin 6; LDL, low-density lipid level; SBP, systolic blood pressure; g, gram; dL, deciliter; nmol, nanomole; L, liter; hr, hour; mg, milligram; μg, microgram; mmHg, millimeters of mercury; μmol, micromole; pg, picogram; mL, milliliter.