

Research Article

Cardiovascular Health at the Intersection of Race and Gender: Identifying Life-Course Processes to Reduce Health Disparities

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Abstract

Objectives: Cardiovascular health (CVH) is associated with reductions in age-related disease and later-life mortality. Black adults, particularly Black women, are less likely to achieve ideal CVH. Guided by intersectionality and life-course approaches, we examine to what degree (a) disparities in CVH exist at the intersection of race and gender and (b) CVH disparities would be reduced if marginalized groups had the same levels of resources and adversities as privileged groups.

Methods: We used biomarker subsamples from the Midlife in the United States Core and Refresher studies ($N = 1,948$). Causal decomposition analysis was implemented to test hypothetical interventions to equalize the distribution of early-life adversities (ELAs), perceived discrimination, or midlife socioeconomic status (SES) between marginalized and privileged groups. We conducted sensitivity analyses to determine to what degree unmeasured confounders would invalidate our findings.

Results: White women have the highest CVH score, followed by White men, Black men, and Black women. Intervening on ELAs would reduce the disparities: White men versus Black women (30% reduction) and White women versus Black women (15%). Intervening on perceived discrimination would not substantially change initial disparities. Intervening on midlife SES would yield large disparity reductions: White men versus Black men (64%), White men versus Black women (60%), and White women versus Black women (27%). These reductions are robust to unmeasured confounders.

Discussion: Providing economic security in adulthood for Blacks may help reduce racial disparities in CVH. Preventing exposure to ELAs among Black women may reduce their vulnerability to cardiovascular disease, compared to White adults.

Keywords: Adverse childhood experience, Cardiovascular health, Gender, Race, SES

Cardiovascular disease (CVD) is common, affecting the majority of Americans older than age 60. In an effort to foster CVD-free longevity, the American Heart Association (AHA) introduced a metric known as ideal cardiovascular health (CVH); the metric includes seven components across biological (blood pressure, glucose, cholesterol, body mass index [BMI]) and behavioral (diet, physical activity, and smoking) domains (Lloyd-Jones et al., 2010). Cumulative evidence shows that maintaining ideal CVH is key for

healthy aging, as it is linked to lower CVD morbidity and mortality and reduced physical disability and cognitive impairment in old age (Deckers et al., 2017; Dong et al., 2012; Samieri et al., 2018).

Epidemiological studies show that Black or African American adults (hereafter “Black adults”) are less likely than non-Hispanic White adults (hereafter “White adults”) to achieve ideal scores across nearly every component of CVH, except for lipid profiles (Benjamin et al., 2019). The

racial gaps are larger among women than men (Pool et al., 2017). Racial gaps are also evident among children (Ning et al., 2015), indicating that such disparities may originate in early life. While the roles of early-life adversities (ELAs), discrimination, and socioeconomic status (SES) have been widely implicated as potential mechanisms (Glymour et al., 2014; Redmond et al., 2013; Su et al., 2015), few studies have systematically investigated the contributions of life-course factors to gendered racial disparities in CVH in a causal framework.

Using biological data from the Midlife in the United States (MIDUS) study, we investigate life-course pathways that explain patterns in CVH at the intersection of race and gender. We apply decomposition analysis in a causal framework to test hypothetical interventions, that is, the degree to which CVH disparities would be reduced if Black adults had the same levels of ELAs, perceived discrimination, or adult SES as White adults.

Background

Intersectionality and CVH Disparities

Racial and sex disparities in CVD are well documented. However, considering the interaction between race and sex reveals a unique pattern of CVH inequalities. Studies based on the National Health and Nutrition Examination Survey and the Coronary Artery Risk Development in Young Adults study show that White women have better CVH than White men, while Black women have similar CVH compared to Black men (Bey et al., 2019; Pool et al., 2017). The higher CVH for women than men among White adults is partially attributed to different levels of endogenous estrogens, which exert protective effects in the cardiovascular system, particularly before menopause (Groban et al., 2016). The question remains—why does the same pattern not hold for Black adults?

The pattern described above fits the tenets of an intersectionality framework, which posits that social stratification, including race/ethnicity, gender (a socially constructed category related to sex), and age, among others, forms an interlocking system of oppression, limiting opportunities for those with marginalized social statuses (Choo & Ferree, 2010; Collins, 2002). Thus, having multiple nondominant positions may be associated with adverse health outcomes above and beyond any individual nondominant position. Black women, who are positioned at the bottom of racial and gender social hierarchies, may face more obstacles to achieving ideal CVH than other intersectional groups, possibly because Black women are more likely to experience more severe life adversities and social or material difficulties, while simultaneously having fewer resources to cope with these challenges (Bowleg, 2012; Brown, 2012; Geronimus et al., 2006). Moreover, the intersectionality framework further suggests how one can experience oppression in one social system of inequality

but be privileged in other systems. For instance, Black men might be privileged in the gender hierarchy but subordinate in the racial hierarchy. Relatively little attention has been given to health in mixed-privilege groups, compared to multiply marginalized groups.

Life-Course Mechanisms

Because one cannot modify ascribed characteristics like race and sex, identifying modifiable pathways that link marginalized social statuses with health outcomes is a key feature of intersectionality and health disparities research (Bauer & Scheim, 2019). Kuh et al. have proposed life-course models of how early- and later-life biological, behavioral, and psychosocial exposures affect the development of chronic disease (see Ben-Shlomo et al., 2014 for review). The accumulation of risk model, in particular, suggests that exposures to health risks that gradually accumulate over the life course can ultimately increase the risk of developing chronic disease and, moreover, that differential exposures over the life course are an underlying mechanism of health disparities between socially advantaged and disadvantaged groups. Among various exposures, this study focuses on the cumulative effects of three interrelated life-course factors—ELAs, perceived discrimination, and low SES—each of which is significantly associated with poor CVH.

Stressful or traumatic experiences during early life may have powerful and life-long consequences for CVH because they may alter the structure and function of physiological systems, negatively influencing cognitive and socioemotional development and increasing the likelihood of engaging in unhealthy behaviors and lifestyles (Miller et al., 2011; Shonkoff et al., 2009; Suglia et al., 2018). Such early-life conditions shape later-life health through further hardships throughout the life course. For example, individuals who were maltreated as children are likely to experience disadvantaged SES trajectories, such as lower educational attainment in young adulthood and economic strain in midlife (Jaffee et al., 2018; Lee & Ryff, 2019). Moreover, adolescents who grew up in poverty are likely to experience higher levels of discrimination, which is linked with elevated allostatic load (Fuller-Rowell et al., 2012). Exposure to ELAs is far more common and more likely to co-occur among non-White adults than White adults, and women are more likely than men to experience trauma (e.g., sexual abuse) during childhood (Merrick et al., 2018). Therefore, we examine ELAs along life-course pathways to explain why Black adults (particularly Black women) exhibit poor CVH, possibly through other life-course factors, such as perceived discrimination and low SES in adulthood.

Socioeconomic position is a major determinant of health, including CVD incidence and ensuing mortality (Glymour et al., 2014; Link & Phelan, 1995). Throughout the life course, Black adults are socially disadvantaged relative to White adults in the United States in terms of health care access and insurance coverage (Sohn, 2017) as well

as multiple domains of SES, including education, income, and wealth (Kilewald & Brielle, 2018). Black women are particularly likely to experience insecure economic positions throughout the life course (Brown, 2012). As a result, SES inequality may play a major role in disparities in CVH, possibly through differences in health-related behaviors/lifestyles, access to quality health care systems, living in a safe and recreational neighborhood, having beneficial social connections, and exposure to chronic stressors (Adler & Newman, 2002).

Many studies, however, have found that racial disparities in health remain significant, even after accounting for SES (Hayward et al., 2000). Racism has been considered as an underlying cause of adverse health for racial/ethnic minorities (Williams et al., 1997). Structural racism exists in multiple social systems, including education, labor, housing, and criminal justice systems, reducing opportunities and resources for racial minorities, thus ultimately contributing to racial disparities in health (Williams et al., 2019). Discrimination is the most widely studied form of racism in the health disparities literature and is often captured by individuals' reports of unfair treatment (Williams et al., 2019). A growing body of literature suggests that self-reported discrimination is significantly associated with multiple indices of poor CVH (Fuller-Rowell et al., 2019). Therefore, we expect that unequal exposure to discrimination may be one life-course factor via adult SES that explains why Black adults (particularly Black women who may experience both racism and sexism) have poor CVH.

Thus far, we have argued that unequal exposure to potential mediators contributes to health disparities. A remaining question is whether the effect of mediators differs across intersectional groups. Investigating both mechanisms (differential exposure and vulnerability) is critical for estimating unbiased mediating effects. Vulnerability to life-course factors that affect health has been shown to vary by race and gender. For example, diminishing returns of adult SES on health outcomes for Black adults compared to White adults (Brown et al., 2016), possibly because of chronic stress associated with achieving higher social positions amid institutional and interpersonal racism (Colen, 2011). Similarly, while Black adults report greater exposure to discrimination than White adults, Black adults' CVH is less adversely affected by it than White adults, possibly due to lower tolerance and less acceptance of discrimination among White adults (Bey et al., 2019). These findings indicate the importance of using complex mediation models that address both differential exposure and vulnerability to investigate the contribution of these life-course factors to health disparities across intersectional groups.

Aims of the Current Study

Based on intersectionality and life-course approaches, we advance prior studies in two ways. First, we explicitly quantify patterns of CVH disparities in the race-gender

hierarchy: the most privileged group (White men), the least privileged group (Black women), and mixed-privilege groups (White women and Black men). Second, we systematically investigate the observed CVH disparities in terms of the cumulative effects of three inter-connected life-course factors: ELAs, perceived discrimination, and adult SES. Using causal decomposition analysis, we estimate the degree to which CVH disparities in midlife would be reduced if marginalized groups had the same levels of ELAs, adult SES, or perceived discrimination as more privileged groups. Our rigorous approach to mediation analysis accounts for the effects of both differential exposure and differential vulnerability. We also conduct sensitivity analyses to determine the degree to which unmeasured confounders might bias our estimates. Our study is unique in incorporating causal inference and mediation analyses into the life-course model.

Data and Methods

Sample

Data come from the MIDUS study, a national survey of health and aging that began in 1995–1996 (M1) with a sample of 7,108 noninstitutionalized, English-speaking adults aged 25–74 in the continental United States. A follow-up interview (M2) was completed in 2004–2006 ($n = 4,963$) and, after adjusting for mortality between M1 and M2, the retention rate was 75%. An oversample of Black adults from Milwaukee ($n = 592$) was added at M2 to increase the representation of African Americans and facilitate analyses of racial disparities in health. At M2, a subset of respondents from M2 and the Milwaukee samples ($n = 1,255$, 16% Milwaukee) traveled to one of the three General Clinical Research Centers for an overnight stay where biological data were collected. The response rate among eligible participants was 43%, reflecting the demanding protocol and extensive travel for participants (Love et al., 2010). In 2011–2014, a new MIDUS refresher (MR) cohort ($n = 3,577$) was recruited to match the age and gender distribution of the M1 sample. An additional sample of Black adults from Milwaukee was recruited at MR ($n = 508$). Consistent with M2, biomarker data were collected on a subset of respondents at MR ($n = 863$, 13.6% Milwaukee). The response rate for participating in the MR biological substudy was 42%.

Our sample included respondents ($n = 2,118$) who participated in the M2 or MR biological data collection. The M2 biological subsample was comparable to the M2 survey sample on most demographic and health characteristics, although they were more educated and less likely to smoke than nonparticipants (Love et al., 2010). Similarly, the Refresher biological subsample, although slightly older, of higher SES, and having better health than the main Refresher sample, was comparable on most characteristics (Supplementary Table S1). Among 2,118 participants, we excluded 170 respondents who (a) identified as a race other

than White or Black and/or African American, (b) identified as an ethnicity with Hispanic origins, or (c) did not report gender. Given the small sample of Latinxs from M2 and MR (<4%), any respondents who identified themselves as of Spanish, Hispanic, or Latino descent were excluded from the analytic sample. Thus, the final analytic sample consists of 1,948 men and women who self-identified as either non-Hispanic White or non-Hispanic Black.

Measures

Intersectional status was created by following the inter-categorical complexity approach (Bauer & Scheim, 2019). Racial and gender statuses were created using the nexus of self-identified race/ethnicity and gender. MIDUS respondents were asked about their gender (man vs. woman), their racial origins, their best description of their race if they reported multiple racial origins, and whether they are of Spanish, Hispanic, or Latino descent. We coded four race–gender intersectionality statuses and ranked them based on socioeconomic inequality: White men are the most privileged group, White women and Black men are the mixed-privilege groups, and Black women are the marginalized group.

CVH was created with seven metrics based on the AHA's criteria: smoking, BMI, physical activity, diet, total cholesterol, blood pressure, and fasting glucose. Individuals received two points if their value was in the ideal range of CVH, one point for intermediate, and zero points for poor (Lloyd-Jones et al., 2010). These points were summed to create an overall *index of CVH* (ranging from 0 to 14, mean = 8.09, *SD* = 2.29), with higher values reflecting better CVH. For details on how each component was constructed and coded, see S1 in [Supplementary Material A](#).

ELA is a summary score of 13 binary items that fall into three domains. First, *Socioeconomic disadvantage* includes four items: (a) neither parent had obtained at least a high school degree or GED, (b) the family received welfare or Aid to Dependent Children for at least 6 months, (c) respondents reported that their financial level growing up was worse off than others, and (d) neither parent always/most of the time had a paid job. Second, *Family instability* includes four items: (a) not living with both biological parents until age 16, (b) moved to a new neighborhood 3 or more times, (c) death of a parent prior to age 19, and (d) death of a sibling prior to age 19. Third, *Childhood maltreatment* includes five indicators: (a) emotional abuse, (b) physical abuse, (c) sexual abuse, (d) emotional neglect, and (e) physical neglect, all of which are drawn from the Childhood Trauma Questionnaire (Bernstein & Fink, 1998). Each of the five types included five items with response options on a 5-point scale from 1 (*never true*) to 5 (*very often true*). For each type, item responses were averaged and then created as dichotomous variables, coded as 1 for the highest quartile and 0 for the lowest three quartiles. Our operationalization of this summary score of ELAs is consistent with prior work (Fuller-Rowell et al., 2019).

Perceived discrimination was measured using lifetime discrimination and everyday discrimination scales (Williams et al., 1997). On 11 questions categorized into seven domains (education, work, housing, policing, banking, medical care, and service), respondents were asked to report the number of times in their life they faced discrimination because of race, gender, or other characteristics. Each item was recoded 1 if respondents reported 1 or more times, otherwise 0. An inventory of lifetime discrimination was constructed by summing the items with possible scores ranging from 0 to 11. Everyday discrimination was assessed through nine forms of discrimination (e.g., treated with less respect or courtesy), each measured with a 4-point scale (1 = never through 4 = often). An index of perceived discrimination was created by standardizing (mean 0; *SD* 1) and computing the average across these scales (Cronbach's $\alpha = 0.67$).

Adult SES included eight SES indicators in midlife: (a) highest level of education completed (1 = no school/some grade school to 12 = PhD, MD, or other professional degree), (b) household income (\$0–\$300,000 or more), (c) wage/salary income (\$0–\$100,000 or more), (d) current financial situation (0 = worst possible through 10 = best possible), (e) control over financial situation (0 = worst possible through 10 = best possible), (f) availability of money to meet basic needs (1 = more than enough through 3 = not enough, reverse coded), (g) level of difficulty paying bills (1 = very difficult through 4 = not at all difficult), and (h) money remaining after liquidating all assets and allocating everything toward any debts (1 = would still owe money, 2 = debts would just about equal assets, 3 = would have money left over). We standardized each indicator and coded them so that higher values represented more advantages. We then created an index by computing the average across indicators (Cronbach's $\alpha = 0.81$).

Covariates included *age* (mean = 48.7, *SD* = 12.53) as a continuous variable. *Refresher* (vs. M2 sample) was included to address the possibility that differences in data collection across the samples and cohorts might influence the findings. Because *history of cardiovascular illness for biological parents* (heart problems, stroke, and diabetes) may reflect genetic susceptibility and shared lifestyle/environments that may increase respondent's CVD risk and may also negatively affect ELAs (e.g., SES), we included this covariate as a potential mediator–outcome confounder.

Analytic Strategies

Descriptive statistics for all variables used in the analyses by race–gender intersectionality groups are presented in [Table 1](#). The magnitude of disparities across intersectional groups may vary by an individual component of CVH and mediators. In [Table 2](#), we created six reference and comparison subsets among race–gender groups. The reference group for each comparison was specified as the more advantaged group. We used analysis of variance and

Table 1. Descriptive Statistics of Variables by Race–Gender Intersectionality Groups, Mean (SD) or %

Variables (range)	White men (n = 751)	White women (n = 799)	Black men (n = 126)	Black women (n = 272)
Early life adversities (z score)	–0.19 (0.85)	–0.06 (0.98)	0.34 (1.07)	0.54 (1.15)
Perceived discrimination (z score)	–0.25 (0.77)	–0.10 (0.79)	0.78 (1.42)	0.63 (1.36)
Adult SES (z score)	0.33 (0.87)	0.09 (0.90)	–0.73 (1.00)	–0.84 (0.96)
Total cardiovascular health score (CVH, 0–14)	7.95 (2.11)	8.73 (2.29)	7.29 (2.07)	6.99 (2.09)
Each CVH component (0 = poor – 2 = ideal)				
Blood pressure	0.80 (0.64)	1.04 (0.76)	0.79 (0.69)	0.86 (0.69)
Total cholesterol	1.36 (0.61)	1.35 (0.65)	1.44 (0.65)	1.50 (0.64)
Glucose	1.45 (0.64)	1.66 (0.56)	1.36 (0.66)	1.37 (0.70)
BMI	0.76 (0.73)	0.97 (0.84)	0.81 (0.80)	0.43 (0.67)
Physical activity	1.43 (0.83)	1.41 (0.81)	1.32 (0.89)	0.97 (0.90)
Diet	0.36 (0.51)	0.51 (0.57)	0.31 (0.51)	0.31 (0.51)
Smoking	1.79 (0.60)	1.79 (0.60)	1.25 (0.96)	1.50 (0.85)
Covariates				
Age in years	49.93 (13.28)	47.74 (12.30)	48.67 (10.83)	48.21 (11.60)
Sample (1 = Refresher, %)	41%	35%	41%	43%
Biological parent’s history, %				
Heart problems	48%	51%	29%	38%
Diabetes	23%	27%	30%	45%
Stroke	19%	24%	21%	31%

Note: BMI = body mass index; CVH = cardiovascular health; SES = socioeconomic status.

Table 2. Observed Disparities in CVH and Potential Mediators Across Intersectional Groups

Reference group	White men			White women		Black men
	Black men	Black women	White women	Black men	Black women	Black women
Early-life adversities (z score)	0.53 ^a	0.73 ^a	0.13	0.40 ^a	0.61 ^a	0.21
Perceived discrimination (z score)	1.04 ^a	0.88 ^a	0.15	0.88 ^a	0.73 ^a	–0.15
Adult SES (z score)	–1.06 ^a	–1.17 ^a	–0.23 ^a	–0.82 ^a	–0.93 ^a	–0.11
Overall CVH score	–0.67 ^a	–0.97 ^a	0.77 ^a	–1.45 ^a	–1.74 ^a	–0.29
Seven components of CVH						
Blood pressure criteria	–0.01	0.06	0.24 ^a	–0.25 ^a	–0.18 ^a	0.07
Total cholesterol criteria	0.08	0.14 ^a	–0.01	0.09	0.15 ^a	0.06
Glucose criteria	–0.09	–0.08	0.21 ^a	–0.30 ^a	–0.29 ^a	0.01
BMI criteria	0.05	–0.33 ^a	0.21 ^a	–0.17	–0.54 ^a	–0.38 ^a
Physical activity criteria	–0.11	–0.46 ^a	–0.02	–0.09	–0.44 ^a	–0.35 ^a
Diet criteria	–0.05	–0.06	0.14 ^a	–0.20 ^a	–0.20 ^a	0.00
Smoking criteria	–0.54 ^a	–0.29 ^a	0.00	–0.54 ^a	–0.29 ^a	0.25 ^a

Notes: BMI = body mass index; CVH = cardiovascular health; SES = socioeconomic status. Negative values in CVH indicate that the comparison group has worse health than the reference group. For multiple comparisons, we used Tukey–Kramer pairwise comparisons to contrast each possible pair of intersectionality groups. ^aDifferences in the reference and comparison groups are statistically significant even after adjusting for multiple comparisons.

Tukey–Kramer pairwise comparisons to contrast each possible pair of intersectionality groups, adjusting for multiple comparisons.

Decomposition Analysis

Decomposition analyses followed two steps: (a) estimation of initial disparities in CVH between comparison and reference groups and (b) estimation of how much that disparity

would be reduced if the distributions of mediator(s) were equal between comparison and reference groups. To do this, we considered the following three hypothetical interventions: *Intervention 1* on ELAs, *Intervention 2* on discrimination, and *Intervention 3* on adult SES. Figure 1 depicts the directed acyclic graph. Formally, let Y be CVH, R be the intersectional group indicator (R = 0: White men, R = 1: White women, R = 2: Black men, R = 3: Black women), and C be pre-exposure confounders (age, parental

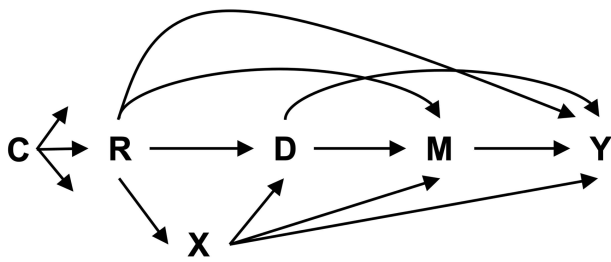


Figure 1. Directed acyclic graph showing relationships between race-gender intersectionality status, cardiovascular health, and three potential mediators. *Note:* Diagram presenting the relationship between race and gender intersectionality status R , cardiovascular health Y , early-life adversity X , discrimination D , and adult socioeconomic status M as well as covariates (age, sample, and parental history of cardiovascular illnesses) C .

history of cardiovascular illness, refresher sample). The intervening variables are denoted as X (ELAs), D (discrimination), and M (adult SES). The causal pathway between mediators is $X \rightarrow D \rightarrow M$. Given the temporal ordering, X confounds the relationship between D and Y , and both X and D confound the relationship between M and Y . Here, X for *Intervention 2* and X and D for *Intervention 3* are defined as post-exposure confounders.

In order to identify disparity reductions, we assume no mediator-outcome confounding given covariates (i.e., conditional ignorability). For example, for *Intervention 1*, we assume that the effect of ELAs on CVH is unconfounded given the race-gender group and covariates. Under the assumption, the estimates of disparity reductions can be given a causal interpretation. The estimation method was built on VanderWeele and Vansteelandt (2014). Suppose that the reference group is White men ($R = 0$) and the comparison group is Black women ($R = 3$). Then, the initial disparity is estimated as

$$\text{Initial disparity} = E[W_{3y} | R = 3] - E[W_{0y} | R = 0] \quad (1)$$

where $W_r = \frac{P(R=r)}{P(R=r|C)}$. This weight (W_r) is computed based on a multinomial logistic model that regresses the race-gender group indicator on the covariates. After the hypothetical interventions of equalizing distributions of mediators, the disparity reduction is estimated as

$$\text{Remaining disparity} = E[W_{0\mu_3} | R = 0] - E[W_{0y} | R = 0] \quad (2)$$

$$\text{Disparity reduction} = E[W_{3y} | R = 3] - E[W_{0\mu_3} | R = 0] \quad (3)$$

where $\mu_3 = E[Y | R = 3, X, c]$ for *Intervention 1*, $\mu_3 = \sum_x E[Y | R = 3, x, D, c] P(x|R = 3, c)$ for *Intervention 2*, and $\mu_3 = \sum_{x,d} E[Y | R = 3, M, c] P(x, d|R = 3, c)$ for *Intervention 3*. For estimating μ_3 , we fitted a normal regression model and obtained a predicted estimate of the outcome if the individual was a Black woman ($R = 3$), but using White men's ($R = 0$) values of the intervening mediator (after controlling for the post-exposure confounders for *Interventions 2* and

3). More technical readers may want to review the detailed formulas given in [Supplementary Material B](#).

Exposure-mediator interactions, if present and neglected, can lead to biased estimates. As we investigated the interaction between intersectional status and each mediator, we found that the effect of discrimination on CVH is in general larger for White adults than Black adults (White men: -0.28 , White women: -0.42 , Black men: -0.12 , and Black women: -0.02). The interaction effect of discrimination by race is significant between White women and Black women (0.39 , $p < .01$), yet there was no significant interaction for either adult SES or ELAs. Thus, we included exposure-mediator interactions for *Intervention 2* (discrimination) only. Standard errors were obtained by bootstrapping. Because the rate of missing data is minimal (all variables have 0%–2% of data missing on average), we handled missing data by imputing modes for categorical variables and medians for continuous variables (Scheffer, 2002). All analyses were carried out in R.

Sensitivity Analysis

Our results might not be valid if there are omitted variables that confound the relationship between mediators and CVH. To assess the robustness of results to potential violations of the conditional ignorability assumption, we examined the extent to which an unmeasured confounder would have to affect both the mediator and the outcome to invalidate our findings (VanderWeele, 2010). Suppose that an omitted variable is binary and denoted as U and that we want to calculate the bias for disparity reductions for Black women ($R = 3$) compared to White men ($R = 0$). Then, the bias would be expressed as the product of two sensitivity parameters: $\text{Bias} = -\gamma \times \beta$.

Specifically, for *Intervention 1*, γ is the difference in the prevalence of omitted variable U between the reference and comparison groups after conditioning on covariates and ELAs; and β is the effect of omitted variable U on CVH after conditioning on race, gender, ELAs, and pretest covariates. The interpretation of γ and β for *Interventions 2* and *3* is the same as for *Intervention 1* after replacing ELAs with the intervening mediator and post-exposure confounders. The key idea is to find values for these two sensitivity parameters that would make the estimated disparity reduction zero.

Results

Descriptive Statistics

Tables 1 and 2 display that Black adults, regardless of gender, reported greater ELAs, higher levels of perceived discrimination, and lower levels of adult SES than White adults. Compared to White men, White women reported lower SES. Compared to Black men, Black women reported

greater exposure to ELAs. We found that Black adults achieved lower scores for most components of CVH, except that they achieved similar or slightly lower total cholesterol scores than White adults. Overall, White women had the highest total scores of CVH followed by White men, Black men, and Black women. The individual CVH components that drive the disparity in total CVH scores across intersectional groups vary, yet the differentials in the overall components are mainly driven by the behavioral component. Specifically, the disparity in total CVH between White men and Black men (0.67 points on the 14-point scale) is driven mainly by smoking disparity (0.54), while the disparity between White men and Black women (0.97) is driven by disparities in physical activity (0.46), BMI (0.34) and smoking (0.29). Compared to White women, Black men show lower total CVH because of lower scores in blood pressure, glucose, diet, and smoking, while Black women exhibit lower scores for nearly every component. Compared to Black men, Black women achieve better scores for smoking but worse scores for BMI and physical activity, which results in no significant gender difference in total CVH among Black adults.

Decomposition Analysis

Table 3 presents the initial disparities, disparity remaining, and disparity reduction in CVH between comparison and reference groups. Compared to White men, initial disparities for Black men and Black women are -0.60 and -0.98 , respectively, conditioning on controls. When compared to White women, initial disparities for Black men and women are even bigger: -1.39 and -1.77 , respectively. The disparity between White men and White women is 0.79 , while the disparity between Black men and Black women is -0.37 .

Equalizing the distributions of ELAs (*Intervention 1*) between reference and comparison groups would reduce disparity by 30% between White men and Black women and by 15% between White women and Black women. As for *Intervention 2* on discrimination, we found little change in the initial disparity except that the disparity between White men and White women would increase by 6%. That is, if White women experienced similar levels of discrimination as White men, White women would be even healthier than White men.

After equalizing the distributions of adult SES (*Intervention 3*) between reference and comparison groups, the disparity would be reduced by 64% for Black men and 60% for Black women, when compared to White men. The disparity remaining would no longer be significant for Black men. When compared to White women, equalizing the distribution of adult SES would reduce a relatively smaller portion of disparity. The initial disparity would be reduced by 20% for Black men and 27% for Black women. We also found that the disparity between White men and

White women would increase by 12% by intervening on adult SES.

Sensitivity Analyses

Figure 2 shows the combinations of sensitivity parameters (γ and β) that would explain away the estimates of disparity reduction (solid line); and the combinations of sensitivity parameters that would change the significance of the estimates at the 95% confidence level (dashed arrow). For an intuitive understanding of sensitivity parameters, suppose that neighborhood deprivation is an omitted confounding variable. Neighborhood deprivation is known to negatively affect ideal CVH (Mujahid et al., 2017), and it is more prevalent among Black men than White men (Logan, 2013). If living in poor neighborhoods decreases CVH by, say, $\beta = 0.6$ (i.e., as strong as parental diabetes which corresponds to 0.2 standard deviations after accounting for other existing covariates and the mediator), then in order to completely explain away the estimated disparity reduction between White men and Black men through intervening on adult SES ($= -0.38$; 95% CI $-0.59, -0.14$), γ would need to be close to 0.6 (because $-0.38 - (-0.60 \times 0.63) \approx 0$); in the example, the difference in the likelihood of living in poor neighborhoods between Black men and White men would need to be 63% even after holding the pretreatment covariate (parental history of CVH), ELAs, and perceived discrimination constant. Even for an upper confidence interval ($= -0.14$) to be zero, γ_m would need to be close to 0.23 (because $-0.14 - (-0.60 \times 0.23) \approx 0$). This large amount of confounding may not be present in practice because prior studies suggest that the effect of neighborhood SES on ideal CVH is significant but not large (Mujahid et al., 2017). With a similar level of household income, Black adults are more likely to live in poor neighborhoods than White adults, yet the racial difference in neighborhood poverty is less than 10% after controlling for individual's SES (Logan, 2013).

Using similar logic, we found that most disparity reductions would be robust, even when unobserved confounding exists that is as strong as parental diabetes: White men versus Black women (Figure 2A) and White women versus Black women (Figure 2B) through *Intervention 1* and White men versus Black men (Figure 2D), White men versus Black women (Figure 2E), and White women versus Black women (Figure 2G) through *Intervention 3*. This is because unobserved mediator–outcome confounders between comparison and reference groups would have to differ by at least 20% to change the significance of the estimates at the 95% confidence level. In contrast, disparity reductions would be sensitive to unobserved confounders for White men versus White women (Figure 2C) through *Intervention 2* as well as White men versus White women (Figure 2F) and White women versus Black men (Figure 2H) through *Intervention 3* if there is unobserved confounding as strong as parental

Table 3. Estimates of Initial Disparity, Disparity Remaining, and Disparity Reductions (95% CI) in Cardiovascular Health Score Under Three Hypothetical Interventions by Equalizing Mediators Between Reference and Comparison Groups

Reference group	White men		White women		Black men		Black women	
	Black men	Black women	White men	White women	Black men	Black women	Black men	Black women
<i>Intervention 1: ELAs</i>								
Initial disparity	-0.60 (-0.99, -0.19)	-0.98 (-1.26, -0.66)	0.79 (0.59, 1.02)	0.82 (0.62, 1.04)	-1.39 (-1.82, -0.98)	-1.77 (-2.08, -1.45)	-0.37 (-0.86, 0.11)	-0.29 (-0.74, 0.18)
Remaining disparity	-0.53 (-0.91, -0.15)	-0.68 (-0.96, -0.38)	0.82 (0.62, 1.04)	0.82 (0.62, 1.04)	-1.36 (-1.78, -0.96)	-1.51 (-1.81, -1.21)	-0.29 (-0.74, 0.18)	-0.09 (-0.23, 0.05)
Disparity reduction	-0.07 (-0.28, 0.14)	-0.30 (-0.45, -0.16)	-0.03 (-0.07, 0.00)	-0.03 (-0.07, 0.00)	-0.03 (-0.24, 0.18)	-0.26 (-0.40, -0.13)	-	-
% reduction	-	30.5% ^a	-	-	-	14.5% ^a	-	-
<i>Intervention 2: Discrimination</i>								
Remaining disparity	-0.58 (-1.04, -0.04)	-0.91 (-1.23, -0.60)	0.84 (0.63, 1.04)	0.84 (0.63, 1.04)	-1.39 (-1.85, -0.89)	-1.71 (-2.01, -1.41)	-0.33 (-0.81, 0.10)	-0.04 (-0.17, 0.08)
Disparity reduction	0.02 (-0.33, 0.30)	-0.06 (-0.24, 0.11)	-0.04 (-0.09, -0.00)	-0.04 (-0.09, -0.00)	-0.00 (-0.30, 0.29)	-0.06 (-0.22, 0.09)	-0.04 (-0.17, 0.08)	-
% reduction	-	-	-5.7%	-5.7%	-	-	-	-
<i>Intervention 3: Adult SES</i>								
Remaining disparity	-0.22 (-0.63, 0.21)	-0.40 (-0.73, -0.09)	0.89 (0.68, 1.10)	0.89 (0.68, 1.10)	-1.11 (-1.50, -0.68)	-1.29 (-1.62, -0.99)	-0.30 (-0.79, 0.15)	-0.08 (-0.24, 0.07)
Disparity reduction	-0.38 (-0.59, -0.14)	-0.58 (-0.77, -0.40)	-0.10 (-0.16, -0.05)	-0.10 (-0.16, -0.05)	-0.28 (-0.49, -0.06)	-0.47 (-0.66, -0.32)	-0.08 (-0.24, 0.07)	-
% reduction	64.0% ^a	59.2% ^a	-12.4%	-12.4%	20.1%	26.8% ^a	-	-

Notes: ELA = early-life adversity; SES = socioeconomic status. The percentage of reduction was calculated when disparity reduction is statistically significant.

^aSensitivity analysis shows that the disparity reductions would remain significant at $p < .05$.

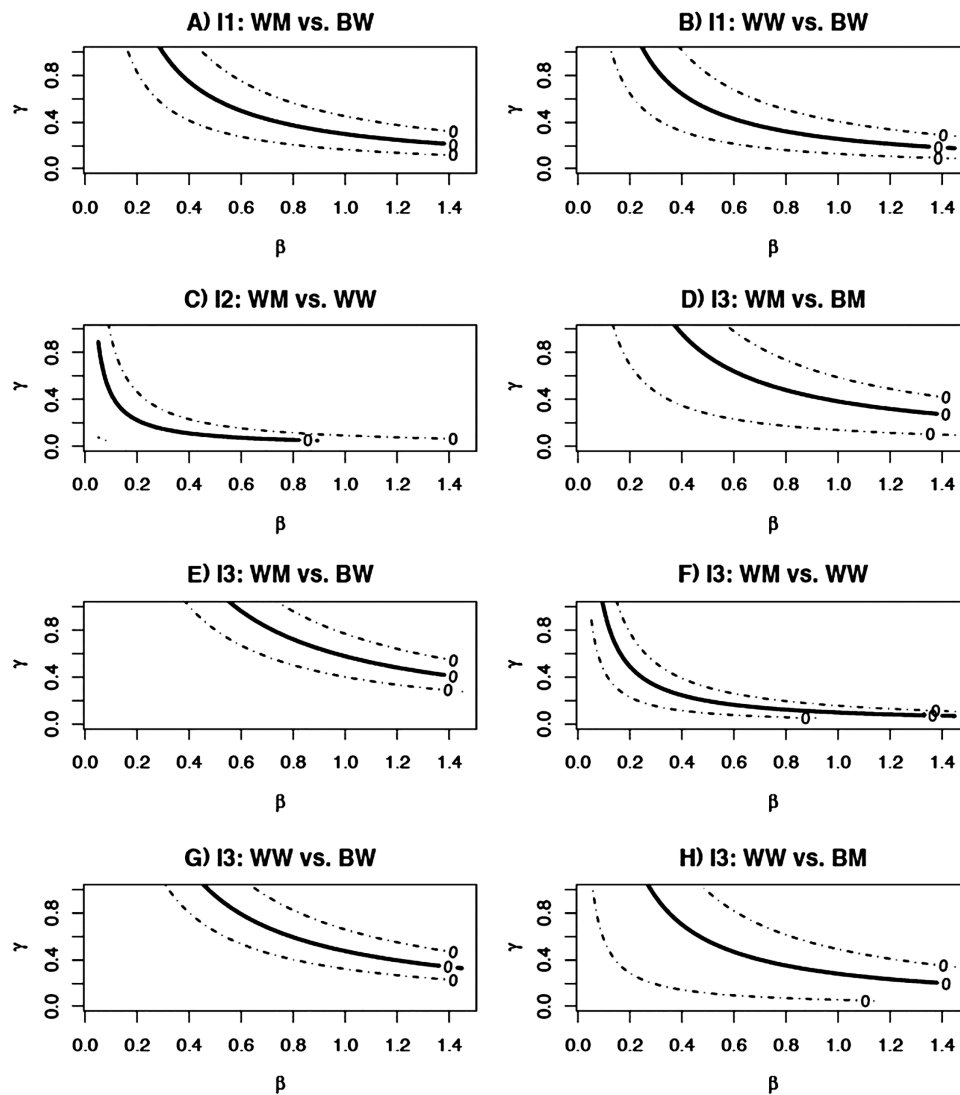


Figure 2. Sensitivity analysis for disparity reductions between the reference and comparison groups. *Note:* I1 = Intervention on early-life adversities; I2 = Intervention on perceived discrimination; I3 = Intervention on adult socioeconomic status; WM = White men; WW = White women; BM = Black men; BW = Black women. Solid lines represent the points at which the estimate of disparity reduction becomes zero due to omitted variable *U*, and dashed lines represent the points at which the 95% intervals of disparity reduction become zero due to omitted variable *U*.

diabetes. This is because unobserved mediator–outcome confounders which differ by around 10% between comparison and reference groups could change the significance of the estimates at the 95% confidence level.

Discussion

Using MIDUS respondents who provided biological data, we observed that CVH was patterned by race and gender. White women had the highest (healthiest) scores and Black women had the lowest scores although gender differences among Black adults were not statistically significant. Given that individuals who participated in biomarker data collections were more likely to be women, better educated, and healthier than those who only participated in

MIDUS baseline studies, the observed patterns might be biased from selective attrition. In a sensitivity analysis, we weighted the biomarker subsample by the inverse of the probability of attrition given sociodemographic and health characteristics (Supplementary Table S1). The observed disparities from sensitivity analysis (with the attrition weight) tend to be slightly larger than the results presented in Table 2, but the substantive findings are the same (Supplementary Table S2). We observed a similar pattern even after controlling for pre-exposure confounders (e.g., parental history of CVH). Our findings are consistent with those from epidemiological studies (Bey et al., 2019; Pool et al., 2017).

Using causal decomposition analysis, we further investigated hypothetical interventions on ELAs, discrimination,

and adult SES to identify the extent to which such health disparities would be reduced if we intervened on these life-course factors across intersectional groups. Results from decomposition analyses yielded three findings. First, those with intersecting disadvantaged statuses (Black women) are most vulnerable to childhood adversities. Our decomposition analysis shows that if Black women had the same levels of ELAs as White men, the health disparity between these two groups would reduce by one third. In the same vein, if Black women had the same levels of ELAs as White women, the health disparity would be reduced by around one sixth. The results of the sensitivity analysis show that such reductions would remain significant even if unobserved confounders (as strong as parental diabetes) exist. In terms of potential mechanisms to reduce racial disparities in CVH, prior studies have given more attention to socioeconomic disadvantage and discrimination than to ELAs. Our findings suggest that preventing exposure to ELAs among Black women would be the first and critical step to reducing their vulnerability to poor CVH.

Second, consistent with prior studies (Bey et al., 2019), we found that Black men have the highest levels of self-reported discrimination, followed by Black women, White women, and White men. However, intervention on perceived discrimination would not significantly reduce the health disparity between any group comparisons except for the disparity between White men and White women, which is vulnerable to unobserved confounders. In terms of differential vulnerability, the effect of perceived discrimination on CVH varies by intersectional group. Predicted scores of CVH are lower when White adults report higher levels of perceived discrimination. Yet, there is no statistically significant association for either Black women or Black men. These findings, though perplexing, follow a similar pattern to those from prior work that found a salient inverse association between interpersonal discrimination and CVH scores for White adults, but not for Black adults (Bey et al., 2019). Prior studies have reported lower resilience to chronic stress for White adults compared to Black adults (Brown et al., 2020). Black men and women who regularly experience unfair treatment may develop adaptive coping strategies and psychological resources, for example, religious coping (Chatters et al., 2008), that help minimize their appraisal of chronic strains and reduce the adverse health consequences of discrimination.

Third, socioeconomic conditions in midlife are patterned by race, which substantially contributes to racial disparities in CVH. Echoing deep economic inequality in later life between Black adults and White adults (Killewald & Brielle, 2018), we found that adult SES for Black men and Black women is more than 1 SD below that of White men. Similarly, the average SES for White women is close to 1 SD above that for Black women and Black men. The decomposition analysis shows that CVH disparities would be substantially reduced for Black men and women by two thirds if they had the same levels of adult SES as White

men. Similarly, if Black men and women had the same level of SES as White women, the health gap between Black adults and White women would be significantly reduced. Our finding suggests that health interventions that increase economic resources for Black adults might alleviate racial disparities in CVH in adulthood. It is important to notice that, in the causal framework described in Figure 1, the disparity reduction through *intervention on adult SES* is an accumulating effect, that is, a sum of all life-course pathways from intersectionality status to CVH via adult SES, including the pathways from ELAs or discrimination. That is, unequal SES in midlife is a consequence of unequal exposure to ELAs and disadvantage, which in turn result in poor CVH in later life (for details on analytic approach and interpretation, see S2 in Supplementary Material A).

Several methodological limitations and future considerations should be acknowledged. First, nearly all Black adults in this study were recruited as part of the Milwaukee sample. Though recruited through probability sampling, because it was drawn from one of the most segregated cities in the United States, it may not allow for generalizable findings for Black adults in the United States nationally. Nonetheless, Milwaukee offers an informative context for understanding racial disparities in health, since many Black adults live in segregated urban contexts. Second, our measure of discrimination mainly captures individuals' awareness of or willingness to report discrimination, which possibly underestimates the actual effect of discrimination on health outcomes (Krieger, 2020). Future researchers should study structural racism and a broader spectrum of discrimination (e.g., micro-aggressions) to better capture the actual effects of discrimination on health. Third, CVH is measured as a composite score of both health behaviors and biomarkers. Despite its comprehensiveness, the main disadvantage of the CVH score is its lack of specification in terms of which indicators and mediators are the main contributors to the observed disparities. In Supplementary Material, we analyzed composite scores for biological and behavioral components separately (Supplementary Tables S3–S6). The substantive findings were driven more by behavioral than biological components, partially because initial disparities in behavioral components were larger. There may be unique mediators and confounders if CVH components are considered individually, which is an important direction for future research. Moreover, researchers focusing on investigating race–gender disparities in physiological dysregulation should use a broader range of biomarkers and different quantifications of biological aging (Gaydos et al., 2020). Fourth, all three mediators were retrospectively reported or measured in midlife, thus limiting our ability to make strong claims on the direction of associations between mediators (i.e., ELAs and discrimination). In a sensitivity analysis, when we applied alternative ordering and intervened on the mediators simultaneously, the substantive findings were consistent with alternative approaches (Supplementary Table S6). Finally, the result

that significant disparities in CVH would remain even after hypothetical interventions warrants further investigation for other life-course mechanisms, for example, early reproduction and parenthood between White women and Black women (Sweeny & Raley, 2014).

Despite such limitations, our study has advanced prior work both conceptually and methodologically. We have integrated life-course perspectives and intersectionality to better understand why Black adults, particularly Black women, have poorer CVH than White adults. We have used causal decomposition methods and sensitivity analysis to rigorously test the relative importance of ELAs, perceived discrimination, and adult SES in shaping racial and gendered disparities in CVH. Our results suggest that reducing SES inequality is fundamental to reducing racial disparities in CVH. Moreover, policy programs initiated in early life that minimize exposure to multiple adversities, particularly for women of color, could be critical to improving CVH in later life.

Supplementary Material

Supplementary data are available at *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences* online.

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Conflict of Interest

None declared.

Author Contributions

C. Lee initiated and designed the study, conducted parts of the analysis, and wrote the entire manuscript. S. Park designed and conducted the central part of the analysis and drafted parts of the method and the result sections. J. M. Boylan helped to formulate the initial research questions, contributed to data organization and preparation, and provided critical feedback on the entire manuscript.

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