

Racial and Educational Disparities in Cumulative Exposure to Hardships of the 2008 Great Recession and Inflammation

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ABSTRACT

Objective: This cross-sectional analysis examined self-reported economic hardships of the 2008 Great Recession, race/ethnicity, educational attainment, and psychological well-being (PWB) as predictors of systemic inflammatory physiology at midlife. We also tested for differential vulnerability in the relationship between recession hardship and inflammatory physiology by race/ethnicity, education, and PWB.

Methods: Adults from the Midlife in the United States Refresher sample completed a survey and biomedical assessments after the recession ($n = 592$ non-Hispanic White respondents, $n = 158$ Black/African American respondents, $n = 108$ respondents with other race/ethnicity). Cumulative recession hardship was the sum of financial, housing, and employment-related events. Outcomes included circulating levels of interleukin 6 and C-reactive protein. General linear regression models tested main effects interactions between primary predictor variables.

Results: Educational attainment was inversely associated with recession hardships ($b = -0.18$, 95% confidence interval = -0.26 to -0.11 , $p < .001$). Black/African American respondents reported more recession hardships than White respondents ($b = 1.17$, 95% confidence interval = 0.67 to 1.68 , $p < .001$). More recession hardships predicted higher levels of interleukin 6 ($b = 0.06$, $p < .001$) and C-reactive protein ($b = 0.04$, $p = .004$). Analyses did not support race/ethnicity, education, and PWB as moderators of the association between recession hardship and inflammatory markers.

Conclusions: Race/ethnicity and education independently predicted disparities in cumulative recession hardship exposure. Recession hardship predicted higher blood levels of inflammatory proteins associated with long-term health. The lack of findings for differential vulnerability in the relationship between recession hardship and inflammatory markers by race/ethnicity, education, or PWB was possibly due to the limited sample size.

Key words: recession, unemployment, health, stress, psychological well-being, interleukin 6, C-reactive protein.

INTRODUCTION

The 2007–2009 economic downturn, known as the Great Recession, exposed many Americans to housing loss, financial strain, and job insecurity (1). Racial/ethnic minorities and individuals with lower educational attainment experienced more recession-related hardships than White or educationally advantaged individuals (2–4). Policy efforts like the Healthy People 2020 initiative aimed to reduce socioeconomic status (SES) and racial disparities in disease and mortality (5), yet a stratification of physical health and mortality has persisted in the United States and even widened in the aftermath of the Great Recession (6). Identifying population groups and individuals who are most vulnerable to the health impacts of the Great Recession may help guide policies and target interventions to reduce health disparities. The primary aims of the present study were to examine the extent of the differential exposure to recession-related hardships by race and educational

attainment and to test whether associations between recession-related hardships and physical health were amplified in minoritized racial groups and in educationally disadvantaged relative to more advantaged groups. An additional aim was to evaluate psychological protective factors that may lessen the physical health tolls of widespread economic hardship. Using cross-sectional data from the Midlife in the United States (MIDUS) project, the current analysis investigated race and educational disparities in the impact of hardships of the Great Recession with respect to two indicators of systemic inflammation, interleukin 6 (IL-6) and C-reactive protein (CRP),

AA = African American, BMI = body mass index, CRP = C-reactive protein, EWB = eudaimonic well-being, HWB = hedonic well-being, IL-6 = interleukin 6, MIDUS = Midlife in the United States, PWB = psychological well-being, SES = socioeconomic status, VIF = variance inflation factor

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and assessed whether dimensions of psychological well-being (PWB) moderated the effect on inflammatory physiology.

The stress/distress pathway is the most cited biobehavioral process linking economic downturns to biological dysregulation and poor health (7). The Great Recession has been associated with declines in self-reported indicators of mental (8–11) and physical health (12,13). The evidence has been more mixed regarding the impact of recession-related hardships on individual-level morbidity and mortality outcomes (14). Inconsistencies may be partially related to the fact that many studies have focused on single exposures (i.e., job loss) in the immediate aftermath of the Great Recession when it officially ended in 2009 (2). It is now known that policy decisions slowed the economic recovery (15), and that the economy and employment rate did not fully recover until 2015 (16). In addition, many chronic diseases take years to develop, and some may not yet be evident in the first few years after the Great Recession. The present study therefore investigated a comprehensive measure of recession-related experiences ranging from job loss to financial strain and to housing hardships assessed 3 to 4 years after the official end of the Great Recession in 2009. Linking multiple hardships of the Great Recession with biological processes that precede disease onset or worsen physical functioning will identify individuals who may be more vulnerable to the long-term health impacts of economic downturns.

Repeated immune system activation in response to uncontrollable and unpredictable stressors may lead to chronic upregulation in the synthesis and release of signaling proteins, resulting in subclinical proinflammatory states (17). This type of sustained increase in systemic inflammatory activity is one biological pathway linking psychosocial stressors to disease outcomes in midlife and older adulthood (18). The inflammatory cytokine IL-6 and the hepatic release of the acute-phase CRP are widely assessed measures of systemic inflammation in clinical and epidemiological research. Elevated levels of inflammatory markers have been associated with increased disease and mortality risk (19–22).

There is only limited research on associations between recession-related events and inflammatory physiology. In one longitudinal study, declines in individual wealth during the Great Recession were associated with increases in CRP from prerecession to postrecession (23). Prior research has shown that stressful life events (e.g., unstable housing, job loss, serious financial problems, relationship difficulties) are associated with higher levels of systemic inflammation. However, the associations generally have small effect sizes and are further reduced when other health behavior factors, body mass index (BMI), and chronic conditions such as diabetes are also considered (24,25). More research is needed to determine whether recession-related events are associated with inflammatory physiology independent of other health behavior and clinical factors.

Before the Great Recession, racial and SES disparities in inflammatory physiology have been widely documented in middle-aged and older adults in the United States. Relative to the more SES-advantaged and White respondents, lower-SES and Black/African American (AA) respondents in the United States had higher circulating levels of IL-6 and CRP (26–30). Recession-related hardships may further contribute to racial and SES disparities in inflammatory markers through two mechanisms. First, through the pathway of differential exposure, racial and ethnic minorities and educationally disadvantaged individuals experienced more economic losses than White and educationally advantaged groups (2,3). Individuals

without a college degree experienced more job losses and gained back fewer jobs during the recovery (31), and had greater declines in wealth (3) relative to individuals with a college degree. According to data from the US Census Bureau, inflation-adjusted median wealth declined by 66% among Latino(a)/Hispanic households and 53% among Black households, compared with only 16% among White households (3). Such racial and ethnic disparities in wealth declines are rooted in racist and discriminatory mortgage lending practices that heightened vulnerability to foreclosures (32). Employment rates also declined, to a greater extent, among Black and Latino(a)/Hispanic groups (33), reflecting the concentration of minoritized racial/ethnic groups in service, manufacturing, and construction sectors that were disproportionately impacted by the Great Recession (32).

The second mechanism through which recession-related hardships may contribute to disparities in inflammation is differential vulnerability. That is, recession-related hardships may have more deleterious effects on physical health among minoritized and low-SES groups compared with more privileged groups because the former may have less access to social capital and information that can be used to help cope with economic stressors (34). Economic stressors may therefore have more prolonged consequences for health among racial/ethnic minorities and the SES disadvantaged. Long-standing racial and socioeconomic inequities in access to mental and physical health services also exacerbate disparities in health conditions because these conditions are more likely to go untreated in less advantaged groups (35,36). After the recession, education-based disparities and Black/AA-White and Hispanic-White disparities in postponed health care increased (37).

Psychosocial factors are theorized to contribute to variability in race/ethnicity and SES disparities in health outcomes (38,39). However, limited evidence is available on psychosocial contributions to the heterogeneity between recession hardship and inflammatory physiology (40). PWB is a multidimensional domain that derives from several different theoretical frameworks and fields (41,42). The hedonic approach centers on global evaluations of positive and negative affect and life quality (43). In contrast, the eudaimonic approach emphasizes self-actualization and considers the human potential that may arise from key challenges in life (44,45). These challenges include feeling good about oneself while having awareness of personal limitations (self-acceptance), maintaining warm and trusting interpersonal relationships (positive relations with others), shaping the environment to meet personal needs and desires (environmental mastery), seeking self-determination and personal authority (autonomy), finding meaning in one's efforts and challenges (purpose in life), and making the most of one's talents and capacities (personal growth).

Hedonic and eudaimonic dimensions are correlated to some degree but are empirically distinct (42,46,47). Notably, hedonic (HWB) and eudaimonic well-being (EWB) explain physical health and mortality risk independently from depression and other indicators of mental illness (48). In some studies, both dimensions of well-being independently related to health outcomes and their association with health was not attenuated (27,49–51). In addition, HWB and EWB are relatively stable and are resilient to longer-term impacts of economic shocks. Before the Great Recession, a majority (80%) of respondents from the MIDUS study showed stable patterns in EWB across two longitudinal assessments that were more than 10 years apart (MIDUS 1 to MIDUS 2) (52). HWB and EWB in the MIDUS 2 and MIDUS 3 waves,

which were approximately 10 years apart and on either side of the Great Recession, were also highly stable (53). Because of their stability over time, hedonic and eudaimonic indicators serve as informative indicators of individual differences in health-protective psychological factors.

To date, limited research has tested the health-protective role of PWB in the aftermath of the Great Recession. According to The Reserve Capacity Model and Lifespan Biopsychosocial Model of Cumulative Vulnerability and Minority Health (38,39), HWB and EWB may act as protective resources that mitigate the health consequences of lower SES and other forms of social disadvantage (54). Although lower SES is associated with lower levels of HWB and EWB (55,56), many individuals have the capacity to maintain high levels of well-being in the face of adversity (29,57). The mitigation hypothesis proposes that high levels of well-being can substitute for other missing health-enhancing resources and can attenuate the negative health consequences of preexisting vulnerability (low SES or minority racial/ethnicity status) and stressor exposures such as recession hardship. For example, prior research conducted before the Great Recession found that both HWB and EWB attenuated associations between lower levels of educational attainment and higher levels of inflammatory proteins—educational gradients in IL-6 and CRP were smaller among respondents with high levels of well-being compared with respondents with lower levels of well-being (27).

An alternative sociological hypothesis proposes that high levels of social and economic adversity undermine or “disable” the health protective benefits of psychological resources (58,59). The concept of *disabling* originated from cohort comparisons of young adults who entered the labor market either during or after Great Depression in the 1930s (58). For cohorts who entered the labor market in optimal economic conditions, higher levels of conscientiousness predicted better life outcomes such as more stable marriages and secure employment. In contrast, conscientiousness did not predict future life outcomes for the cohort that entered the labor market during the 1930s Great Depression. According to the disabling hypothesis, PWB should be less health protective for individuals with preexisting vulnerability and/or more exposure to recession hardships.

Previous cross-sectional work on adults of the Great Recession from MIDUS found that lower educational attainment interacted with recession hardship exposure and psychological resources to predict self-reported health outcomes (self-rated physical health, acute symptoms, functional limitations, chronic conditions). Findings showed the disabling of psychological resources among participants who were educationally disadvantaged and who experienced greater cumulative recession hardship (13). Indicators of purpose in life did not protect against poorer health among those who experienced the combined disadvantage of lower educational status and high levels of recession hardship.

The present study used cross-sectional survey and biomarker data obtained after the Great Recession from MIDUS to examine: a) disparities in recession hardship exposure by race/ethnicity and educational attainment, b) effects of recession hardship on systemic inflammatory markers, and c) differential vulnerability in the effects of recession hardship on inflammatory physiology by race/ethnicity and educational attainment. Although previous studies have focused primarily on more immediate health outcomes of the Great Recession, this analysis examined inflammatory markers

about 5 to 8 years after the start of the Great Recession. The study also assessed PWB as moderators of the association between recession hardship and inflammation and tested for mitigating or disabling effects. Tests of well-being indicators as moderators were exploratory, given limited prior evidence, the relatively small sample size, and the cross-sectional design.

METHODS

Sample

Data came from the MIDUS study (midus.wisc.edu), which began in 1995 with a national sample of more than 7000 adults (age, 25–75 years) from the 48 contiguous states (60). In 2012, a new nationally representative sample of adults was recruited to augment (“refresh”) the original study to investigate psychosocial and health impacts, broadly defined, of historical change, including the economic recession. Paralleling the MIDUS baseline sample, the Refresher sample (MIDUS-R) included adults aged 25 to 74 years (52.8% female) who were selected from the 48 contiguous states using random-digit dialing of landlines and cell phones ($N = 3577$). A supplemental city-specific Black/AA sample (referred to as MKE-R) was recruited from Milwaukee, WI ($n = 508$), to increase representation and to examine psychosocial factors and health in a highly segregated city. Door-to-door canvassing of individuals residing in census blocks in which at least 40% residents were Black was performed based on race, sex, age, and income to match the Refresher survey distribution. Inclusion criteria were as follows: participants self-identified as Black/AA, lived in noninstitutionalized settings, spoke English with sufficient literacy to complete the self-administered questionnaire, and were healthy enough to complete the interview.

The analytic sample included Refresher participants from both the national cohort ($n = 746$) and the Milwaukee sample ($n = 117$) who both completed the main survey project and participated in the biomarker project. Biomarker data were collected from a subset of participants who agreed to travel to one of three Clinical and Translational Research Centers (Madison, WI; Washington, DC; or Los Angeles, CA) for an overnight visit. The response rate was 42% among those eligible (adjusted for those who could not be reached) and was comparable to the biomarker sample from the original MIDUS baseline cohort (61). Despite the low response rate, likely tied to the time and travel demands of participating in the biomarker project, the analytic sample was comparable to the larger Refresher sample with respect to age, sex, and marital status, although had higher levels of educational attainment. The sample ranged in age from 25 to 75 years (mean [standard deviation] = 50.84 [13.41] years). Survey and biological data collections were approved by the institutional review boards at Georgetown University, University of California, Los Angeles, and University of Wisconsin-Madison. All participants provided written informed consent. Table 1 includes the descriptive statistics for the analytic sample.

Measures

Social Vulnerability

Information on education and racial status was collected from the survey assessments. Respondents reported how many years of school or college they had completed. The 12 response categories

TABLE 1. Descriptive Statistics for MIDUS and Milwaukee—Refresher Biomarker Sample ($n = 863$)

| Variable | M (SD) or n (%) | Range | N Missing |
|---|----------------------|---------------|-------------|
| Sociodemographic covariates | | | |
| Age | 52.7 (13.4) | 26 to 78 | |
| Sex (% female) | 450 (52.1) | | |
| Marital status (% married/cohabitating) | 547 (63.4) | | |
| Employment status, 2007 | | | 1 |
| % Employed | 656 (76.1) | | |
| % Retired | 81 (9.4) | | |
| % Not in work force | 125 (14.5) | | |
| Preexisting vulnerability | | | |
| Race | | | 5 |
| % Non-Hispanic White | 592 (69.9) | | |
| % Non-Hispanic Black/African American | 158 (18.4) | | |
| % Asian, NHPI, AI/AN, Hispanic, multiracial | 108 (12.6) | | |
| Education (highest degree completed) | 8.1 (2.4) | 2 to 12 | 1 |
| Cumulative recession hardship | 3.03 (2.79) | 0 to 10 | 0 |
| Hedonic well-being | 0 (1) | -3.8 to 1.7 | |
| Positive affect | 3.51 (0.82) | 1 to 5 | 3 |
| Negative affect | 1.61 (0.62) | 1 to 5 | 4 |
| Life satisfaction | 7.59 (1.73) | 0 to 10 | 7 |
| Eudaimonic well-being | 0 (1) | -2.7 to 1.4 | 2 |
| Autonomy | 37.0 (6.88) | 7 to 49 | 4 |
| Personal growth | 39.8 (6.37) | 7 to 49 | 4 |
| Purpose in life | 39.0 (6.88) | 7 to 49 | 2 |
| Positive relations with others | 39.2 (7.30) | 7 to 49 | |
| Health covariates | | | |
| % Currently smoking | 94 (11.3) | | 2 |
| Body mass index | 30.4 (7.65) | 17 to 78 | |
| Physician-diagnosed conditions | 4.29 (3.40) | 1 to 30 | |
| % Taking medications ^a | 558 (64.7) | | |
| Inflammation | | | |
| ln IL-6, pg/ml ^b | 0.72 (0.80) | -2.15 to 2.85 | 11 |
| ln CRP, mg/l ^b | 0.34 (1.21) | -3.00 to 4.37 | 14 |

MIDUS = Midlife in the United States; M (SD) = mean (standard deviation); NHPI = Native Hawaiian or Pacific Islander; AI/AN = American Indian/Alaskan Native; IL-6 = interleukin 6; CRP = C-reactive protein.

^a Medications included antihypertensive, cholesterol lowering, anti-inflammatory, steroid, and antidepressants.

^b Values were natural-log transformed. Education is coded as (1 = no school/some grade school; 2 = eighth grade/junior high school; 3 = some high school; 4 = GED; 5 = graduated from high school; 6 = 1 to 2 years of college, no degree yet; 7 = 3 or more years of college, no degree yet; 8 = graduated from a 2-year college, vocational school, or associates degree; 9 = graduated from a 4- or 5-year college, or bachelor's degree; 10 = some graduate school; 11 = master's degree; 12 = professional degree).

ranged from no schooling to completion of a professional degree. Education was mean-centered so that regression coefficients in subsequent analyses could be interpreted as the effect corresponding to 1-unit change in education level. Race/ethnicity was based on self-reported main racial origins (parents, grandparents, and other ancestors; $n = 12$ Asian respondents, $n = 161$ Black respondents, $n = 22$ Native American or Alaskan Indigenous respondents, $n = 2$ Native Hawaiian or Pacific Islander respondents, $n = 55$ multiracial or other identity respondents, $n = 606$ White respondents) and Hispanic or Latino(a) ethnic origins ($n = 34$ Hispanic or Latino(a) respondents, $n = 827$ non-Hispanic or Latino (a) respondents). Because of limited representation of other

minoritized groups other than Black/AA, race/ethnicity was coded into a three-level variable (White, non-Hispanic Black/AA, and other racial category). Most Black respondents were from the Milwaukee sample ($n = 117$ of 161).

Recession Hardship Exposure

The total number of recession-related events was assessed with 18 items. These items derived from a national survey of unemployed adults conducted by the Heldrich Center for Workforce Development, Rutgers (62). The following question opened the MIDUS-R phone/MKE-R in-person interview, "Which of the following have you experienced since the beginning of the Recession

in August 2009?" Respondents reported "yes" or "no" to each item. Four items assessed job impact (e.g., lost your job), seven items assessed home impact (e.g., lost your home because of foreclosure), and seven items assessed financial impact (e.g., declared bankruptcy). Table S2, Supplemental Digital Content, <http://links.lww.com/PSYMED/A951>, reports the proportion of respondents reporting "yes" to each indicator. Consistent with how other checklist inventories of stressful life events have been studied (63), the items were summed to create a cumulative index of exposure. The measure was capped at 10 events to avoid producing unstable estimates that would result from extremely small sample cell sizes, especially those produced in interaction models. In analyses, recession hardship was mean-centered.

Well-Being

Well-being indicators were assessed in the self-administered questionnaire. Ryff's theoretical framework of EWB includes six scales: autonomy, environmental mastery, personal growth, positive relations with others, purpose in life, and self-acceptance (44,47). Each scale included seven items, with internal consistency for each scale ranging from 0.71 to 0.86. On a scale of 1 to 7, respondents indicated the extent to which they agreed or disagreed strongly, moderately, or slightly that an item described how they thought and felt.

HWB was measured with a single item indicator of global life satisfaction and scales of positive and negative affect. The life satisfaction measure was adapted from Cantril's self-anchoring scale, and it asked respondents to "rate their life overall these days" on a scale from 0 to 10, where 0 = worst possible life overall and 10 = best possible life overall. Respondents rated their positive affect and negative affect according to how much of the time in the past 30 days they felt six types of positive (e.g., cheerful) and six types of negative (e.g., nervous) affective states on a scale ranging from 1 = none of the time and 5 = all of the time. Scores for negative affect were reverse coded. Internal consistency for positive affect and negative affect was 0.92 and 0.88, respectively.

Composite measures of HWB and EWB were computed and analyzed as observed variables. A composite index of HWB was computed by standardizing life satisfaction, and positive and negative affect (reverse coded) and summing them. Factor analyses had shown that environmental mastery and self-acceptance cross-loaded with both EWB and HWB dimensions (46). These items were therefore excluded from the composite measure of EWB. The remaining four indicators of EWB were standardized and summed. Both sum scores for HWB and EWB were standardized for regression analysis.

Inflammatory Proteins

Fasting blood samples were collected from each participant in the early morning and processed using standardized procedures of centrifugation and freezing in an ultracold freezer $<-70^{\circ}\text{C}$. Serum levels of IL-6 were measured with the Quantikine high-sensitivity immunosorbent assay kit (ELISA; R&D Systems, Minneapolis, Minnesota). The intra-assay coefficient of variance for the duplicate quantification of each sample was 4.7%, and maximum interassay coefficient of variance was 15% across batches. Plasma CRP was measured initially using the BNII nephelometer (Dade Behring, Inc., Deerfield, Illinois) and then later with an electrochemiluminescence platform (Meso Scale Discovery, Rockville, MD), after confirming

that quantification was similar. The intra-assay and interassay coefficients of variance ranged from 1.1% to 4.4%. In all analyses, IL-6 and CRP were natural logged to address upward skew in the distributions and normalized.

Covariates

Models included age, sex, marital status, employment status before recession, BMI (weight in kilograms divided by height in meters squared), chronic health conditions, and smoking status. Age in years was divided by 10 and mean-centered. Sex was self-reported as male or female and was centered (-0.5 = male, 0.5 = female). Marital status was analyzed as two categories and centered (-0.5 = married/cohabitating, 0.5 = not married). BMI, chronic health conditions, prescription medication use, and smoking are known to be associated with inflammatory measures and were thus included as covariates in the analyses (64). Height and weight were measured by clinical staff and used to calculate BMI (mean-centered). Consistent with prior cross-sectional studies conducted with the MIDUS biomarker data (26,27,29), chronic health conditions were based on the sum score of 30 self-reported physician-diagnosed diseases (summed and mean-centered). Conditions included mental and physical health conditions (e.g., depression, alcohol use disorder, diabetes, liver disease, cancer, cholesterol problems, heart disease). Medications known to affect inflammatory physiology were reported at the clinic visit and classified with pharmaceutical class codes related to antihypertensive, cholesterol lowering, anti-inflammatory, steroids, and antidepressants. Medication use was centered as -0.5 = no medications recorded and 0.5 = any medication recorded. Smoking status was centered as -0.5 = not a current smoker and 0.5 = current smoker. Employment status was based on self-reported employment situation in January 2008 and coded into three categories (working, retired, not currently in work force).

Statistical Analyses

Data are publicly available online at the Inter-university Consortium for Political and Social Research, and analysis code is available upon request from the first author. Table 1 lists the total missing cases for each variable included in the analyses. The percent of complete cases across the entire set of variables was 93.6%. Because of the limited amount of missing data, we used listwise deletion and conducted primary analyses with complete cases (65). Separate hierarchical ordinary least squares regression models were estimated for IL-6 and CRP using the *lm* function in R. Differential exposure was tested with main effects of race/ethnicity and educational attainment in relation to recession hardship in both unadjusted and adjusted models including age, sex, marital/cohabitation status, and work status before the recession. Main effects predicting IL-6 and CRP were examined by first testing bivariate associations with recession hardship, education, race, HWB, EWB, and sociodemographic and health covariates. In hierarchical multivariate models, the first step included recession hardship, education, race, HWB, and EWB simultaneously with sociodemographic and health covariates. In the second step, two-way interactions between education and race with recession hardship tested evidence for differentiability vulnerability. In the third step, two-way interactions between recession hardship and HWB and EWB were examined.

Exploratory analyses tested for the disabling hypothesis versus mitigation hypothesis with all possible two-way and three-way interactions involving HWB or EWB with recession hardship, educational attainment, or race/ethnicity. To interpret significant interactions, simple slopes were calculated at 1 standard deviation below and above the mean, and model predictions for health outcomes were graphed by recession hardship, education or race, and well-being indicators. Significant effects involving HWB or EWB were followed up with tests of each individual indicator. These analyses were exploratory to see if results were consistent across each subscale. Two-sided p values were considered significant at the $<.05$ level.

Case analyses tested for statistical outliers and influential data points. Tests of model assumptions indicated that assumptions of normality, homogeneity, and linearity were met. To test whether multicollinearity was an issue, the variance inflation factor (VIF) was calculated. VIFs indicate how much the standard error of the parameter estimate increases because of redundancy among the predictor variables. None of the VIFs calculated exceeded the threshold of 5, indicating that multicollinearity was not problematic for the tested models. Therefore, models included all variables of interest in the same multivariate models. Little's multivariate test of the Missing Completely at Random assumption (66) was not supported. We therefore examined observed predictors of missingness. Education and BMI were statistically associated with missingness. Sensitivity analyses imputed missing data with pooled estimates across 20-multiple imputation data sets using the mice package in R (67). For composite measures, each item was imputed before being summed.

RESULTS

Sample Descriptive Statistics and Bivariate Associations

Table 1 provides descriptive statistics of all study variables. Table S1, Supplemental Digital Content, <http://links.lww.com/PSYMED/A951>, summarizes the bivariate associations for all study variables. All study variables, except for sex, were significantly associated with IL-6, whereas all study variables, except for age and working status before the recession, were significantly associated with CRP. Recession hardship and identifying as Black/AA versus White were positively associated with IL-6 and CRP. Educational attainment and HWB and EWB were inversely associated with IL-6 and CRP.

Associations Between Individual Recession Hardship Indicators and Inflammatory Markers

Preliminary analyses tested separate regression models of the association for each recession hardship indicator with IL-6 and CRP. All tests were adjusted for sociodemographic and health covariates and well-being indicators. Figure 1 shows that most hardships were positively associated with IL-6, but some of the associations were not statistically significantly different from 0 at the $p < .05$ level. Figure 1B shows that most associations for recession hardship items evinced a trend for higher CRP levels, but were not significantly different from 0, except for missing a mortgage payment and missing a credit card payment. Many hardships shared overlapping 95% confidence intervals (CIs), indicating that they were similarly associated with IL-6 and CRP.

Tests of Differential Exposure to Recession Hardship by Race/Ethnicity and Education

In unadjusted models, the inverse association between education and total recession hardships attained statistical significance ($b = -0.27$, 95% CI = -0.34 to -0.19 , $p < .001$) and remained significant in the model adjusted with sociodemographic covariates ($b = -0.18$, 95% CI = -0.26 to -0.11 , $p < .001$). Black/AA respondents reported more recession hardships than White respondents in the unadjusted model ($b = 2.25$, 95% CI = 1.75 to 2.74 , $p < .001$) and in the adjusted model ($b = 1.17$, 95% CI = 0.67 to 1.68 , $p < .001$). In both unadjusted and adjusted models, respondents who identified as other minoritized racial/ethnic identity did not differ significantly from White respondents ($p = .21$ and $p = .78$, respectively). Follow-up analyses also compared recession hardship exposure in Black/AAs from the MIDUS-R sample ($n = 51$) with that in Black/AAs from the MKE-R sample ($n = 102$) and found no significant differences in unadjusted and adjusted models ($p = .08$ and $p = .37$, respectively).

Tests of Recession Hardship, Race/Ethnicity, Education, and PWB in Relation to Inflammation

Regression analyses for IL-6 and CRP are presented in Table 2. Unadjusted estimates and adjusted multivariate estimates are shown. Adjusting for all sociodemographic and health covariates and well-being indicators, an increase in one recession hardship was associated with higher IL-6 ($b = 0.06$, 95% CI = 0.03 to 0.08 , $p < .001$) and CRP levels ($b = 0.04$, 95% CI = 0.01 to 0.06 , $p = .004$). In adjusted models, the association between education and inflammatory markers was attenuated below the $p < .05$ threshold for IL-6 ($b = -0.01$, 95% CI = -0.04 to 0.01 , $p = .259$), but remained statistically significant for CRP ($b = -0.05$, 95% CI = -0.07 to 0.02 , $p < .001$). In adjusted models, both Black/AA ($b = 0.20$, 95% CI = 0.04 to 0.36 , $p = .015$) and other race/ethnicity respondents ($b = 0.19$, 95% CI = 0.02 to 0.35 , $p = .027$) had statistically significantly higher levels of IL-6 than White respondents. For adjusted models predicting CRP, Black/AA respondents ($b = 0.04$, 95% CI = -0.13 to 0.21 , $p = .67$) and other race/ethnicity respondents ($b = 0.06$, 95% CI = -0.11 to 0.24 , $p = .47$) did not differ significantly from White respondents. In adjusted models for IL-6, the ΔR^2 effect size for recession hardship ($\Delta R^2 = 0.017$) was approximately twice that of race/ethnicity ($\Delta R^2 = 0.008$). For CRP, the effect sizes for educational attainment and recession hardship were similar ($\Delta R^2 \sim 0.01$). Although statistically significant in unadjusted models, in fully adjusted models, the main effect for EWB and HWB was not statistically significantly associated with IL-6 (EWB: $p = .63$; HWB: $p = .11$) or CRP (EWB: $p = .93$; HWB: $p = .24$).

Tests of Differential Vulnerability by Race and Education in Relation to Inflammation

Table 2 presents the results of the two-way interactions between race/ethnicity and recession hardship and educational attainment and recession hardship in relation to IL-6 and CRP. In fully adjusted models, none of the two-way interactions with IL-6 and CRP were statistically significant, and all had ΔR^2 effect sizes less than 0.002.

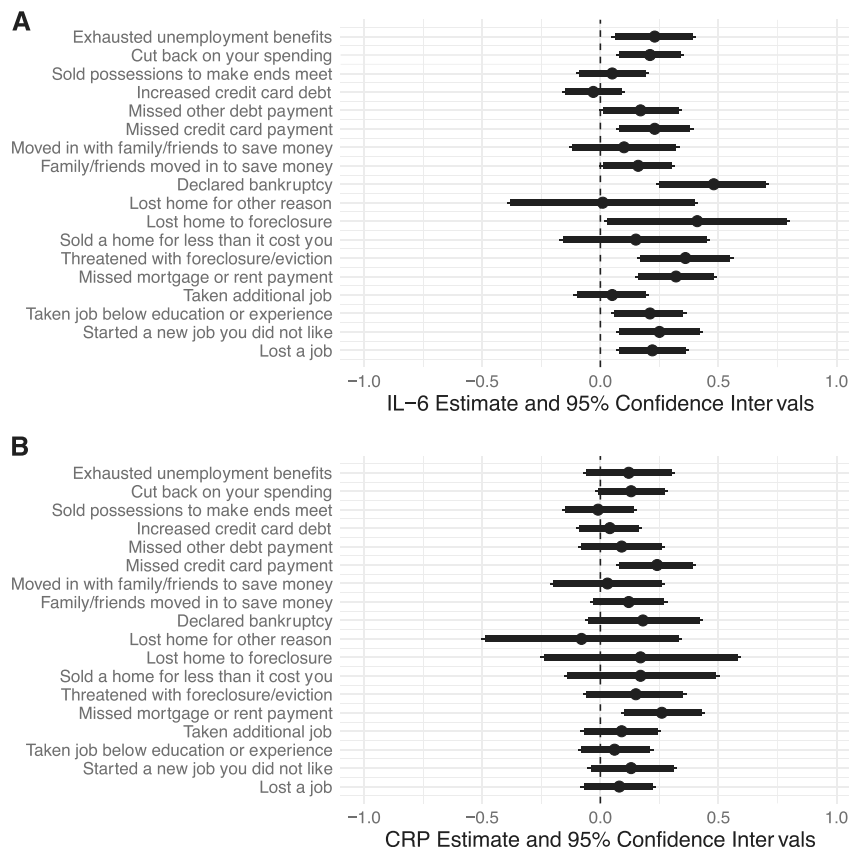


FIGURE 1. A, Ordinary least squares–adjusted associations between individual recession hardship items and IL-6 with 95% confidence intervals. B, Ordinary least squares–adjusted associations between individual recession hardship items and CRP. Models are adjusted for age, sex, partner status, working status, education, race/ethnicity, hedonic well-being, eudaimonic well-being, smoking status, body mass index, total chronic conditions, and medication use ($N = 826$). IL-6 = interleukin 6 (natural log transformed and standardized); CRP = C-reactive protein (natural log transformed and standardized).

Tests of HWB and EWB as Moderators of Recession Hardship and Inflammatory Marker Associations

Although there was no evidence of significant main effects for HWB or EWB, the mitigation of PWB could have resulted in crossover interactions that masked a delineation of main effects. Table 2 shows the tests of two-way interactions between well-being indicators with recession hardship, controlling for all main effects. None of the two-way interactions were statistically significant for IL-6 (HWB by Recession: $p = .85$; EWB by Recession: $p = .57$) or for CRP (HWB by Recession: $p = .94$; EWB by Recession: $p = .21$). Exploratory analyses tested for all possible two-way and three-way interactions between recession hardship, preexisting vulnerability (race/ethnicity or educational attainment), and HWB or EWB. The results were comparable when interactions with EWB and HWB were examined either separately or in the same model. All three-way and two-way lower order interactions were examined in the same model and shown in Table S3, Supplemental Digital Content, <http://links.lww.com/PSYMED/A951>. None of the two-way or three-way interactions for HWB and EWB were statistically significant.

Sensitivity Analyses

Sensitivity tests for main effects predicting IL-6 and CRP were rerun excluding outliers for the model parameters. Outliers were defined

as DFBETA values greater than $2/\sqrt{N}$. Table S4, Supplemental Digital Content, <http://links.lww.com/PSYMED/A951>, shows that the pattern of results remained unchanged after excluding outlier values. Analyses conducted with imputed data also produced similar results (Table S5, Supplemental Digital Content, <http://links.lww.com/PSYMED/A951>).

DISCUSSION

The present study investigated a comprehensive measure of recession-related experiences ranging from job loss to financial strain and to housing hardships assessed during the recovery period after the 2008 Great Recession. The primary aims of the current analyses were to test racial and educational disparities in cumulative exposure to recession hardships and to determine whether these hardships increased synthesis and release of inflammatory proteins approximately 5 to 8 years after the start of the Great Recession. We also explored HWB and EWB as potential moderators of the impact of recession hardships on physical health. These questions were addressed in a diverse sample of middle-aged and older adult Americans for whom postrecession biological indicators of inflammatory activity were available.

The analyses revealed important sources of heterogeneity in exposure to hardships of the Great Recession. Both racial and educational disadvantages emerged as preexisting vulnerability

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TABLE 2. Ordinary Least Squares Regression Model Results of Recession Hardship, Education, Race, HWB, EWB, and Their Interactions Predicting IL-6 and CRP (n = 826)

| Predictor | IL-6 | | | | | | CRP | | | | | |
|---|-------------------------------|----------------|----------------------------|-------------------|-------------------------------|-----------------|-------------------------------|----------------|-----------------|-------------------------------|----------------|-----------------|
| | Unadjusted Models | | | Adjusted Models | | | Unadjusted Models | | | Adjusted Models | | |
| | Estimate (95% CI) | R ² | ΔR ² | Estimate (95% CI) | R ² | ΔR ² | Estimate (95% CI) | R ² | ΔR ² | Estimate (95% CI) | R ² | ΔR ² |
| Step 1: Main effects only | | | | | | | | | | | | |
| Age (by decade) | 0.27 (0.22 to 0.31) | 0.129 | 0.26 (0.21 to 0.32) | 0.071 | 0.04 (-0.01 to 0.09) | 0.003 | 0.06 (0.01 to 0.12) | 0.003 | 0.000 | 0.06 (0.01 to 0.12) | 0.003 | 0.005 |
| Male versus female | 0.03 (-0.10 to 0.17) | <0.001 | 0.08 (-0.04 to 0.19) | 0.001 | -0.33 (-0.47 to -0.20) | 0.028 | -0.25 (-0.37 to -0.14) | 0.028 | 0.000 | -0.25 (-0.37 to -0.14) | 0.028 | 0.014 |
| Married/cohabitating versus not | -0.16 (-0.30 to -0.01) | 0.006 | -0.01 (-0.13 to 0.12) | <0.001 | -0.21 (-0.36 to -0.07) | 0.010 | -0.02 (-0.15 to 0.11) | 0.010 | <0.001 | -0.02 (-0.15 to 0.11) | 0.010 | <0.001 |
| Working status before recession | | 0.048 | | 0.005 | | | | | | | | <0.001 |
| Retired versus working | 0.75 (0.55 to 0.98) | | 0.38 (0.18 to 0.58) | | 0.02 (-0.21 to 0.25) | | -0.02 (-0.23 to 0.20) | | | -0.02 (-0.23 to 0.20) | | 0.005 |
| Not in work force versus working | 0.10 (-0.10 to 0.29) | | 0.08 (-0.08 to 0.24) | | 0.07 (-0.13 to 0.27) | | -0.05 (-0.22 to 0.21) | | | -0.05 (-0.22 to 0.21) | | 0.014 |
| Currently smoke versus not | 0.26 (0.03 to 0.48) | 0.006 | 0.11 (-0.08 to 0.30) | <0.001 | 0.21 (0.01 to 0.43) | 0.004 | 0.05 (-0.15 to 0.25) | 0.004 | 0.000 | 0.05 (-0.15 to 0.25) | 0.004 | <0.001 |
| Body mass index | 0.06 (0.05 to 0.07) | 0.195 | 0.05 (0.04 to 0.06) | 0.122 | 0.07 (0.06 to 0.08) | 0.261 | 0.06 (0.05 to 0.07) | 0.261 | 0.165 | 0.06 (0.05 to 0.07) | 0.261 | 0.165 |
| Chronic conditions (1–32) | 0.09 (0.07 to 0.11) | 0.097 | 0.01 (-0.01 to 0.03) | 0.001 | 0.04 (0.02 to 0.06) | 0.020 | -0.01 (-0.03 to 0.01) | 0.020 | 0.001 | -0.01 (-0.03 to 0.01) | 0.020 | 0.001 |
| Medications versus none | 0.53 (0.39 to 0.67) | 0.065 | 0.08 (-0.05 to 0.21) | 0.001 | 0.43 (0.30 to 0.57) | 0.043 | 0.26 (0.12 to 0.40) | 0.043 | 0.011 | 0.26 (0.12 to 0.40) | 0.043 | 0.011 |
| Recession hardships (0–10) | 0.06 (0.03 to 0.08) | 0.026 | 0.06 (0.03 to 0.08) | 0.017 | 0.08 (0.05 to 0.10) | 0.047 | 0.04 (0.01 to 0.06) | 0.047 | 0.007 | 0.04 (0.01 to 0.06) | 0.047 | 0.007 |
| Education (1–12) | -0.07 (-0.10 to -0.05) | 0.032 | -0.01 (-0.04 to 0.01) | <0.001 | -0.10 (-0.13 to -0.07) | 0.056 | -0.05 (-0.07 to -0.02) | 0.056 | 0.010 | -0.05 (-0.07 to -0.02) | 0.056 | 0.010 |
| Race/ethnicity | | 0.021 | | 0.008 | | | | | | | | <0.001 |
| Black/AA versus White | 0.39 (0.21 to 0.57) | | 0.20 (0.04 to 0.36) | | 0.46 (0.28 to 0.64) | | 0.04 (-0.13 to 0.21) | | | 0.04 (-0.13 to 0.21) | | 0.002 |
| Other versus White | 0.06 (-0.14 to 0.27) | | 0.19 (0.02 to 0.35) | | 0.03 (-0.18 to 0.23) | | 0.06 (-0.11 to 0.24) | | | 0.06 (-0.11 to 0.24) | | 0.002 |
| HWB | -0.14 (-0.21 to -0.07) | 0.022 | -0.06 (-0.13 to 0.01) | 0.002 | -0.17 (-0.24 to -0.10) | 0.028 | -0.05 (-0.12 to 0.03) | 0.028 | 0.001 | -0.05 (-0.12 to 0.03) | 0.028 | 0.001 |
| EWB | -0.09 (-0.15 to -0.02) | 0.007 | -0.02 (-0.08 to 0.05) | <0.001 | -0.08 (-0.15 to -0.02) | 0.007 | -0.003 (-0.07 to 0.07) | 0.007 | <0.001 | -0.003 (-0.07 to 0.07) | 0.007 | <0.001 |
| Step 2: Tests of moderation by preexisting vulnerability | | | | | | | | | | | | |
| Education by Recession | | | -0.003 (-0.01 to 0.01) | <0.001 | | | 0.01 (-0.002 to 0.02) | | | 0.01 (-0.002 to 0.02) | | 0.002 |
| Race/ethnicity by Recession | | | -0.02 (-0.07 to 0.04) | 0.001 | | | -0.04 (-0.09 to 0.01) | | | -0.04 (-0.09 to 0.01) | | 0.002 |
| Black/AA versus White | | | 0.04 (-0.02 to 0.10) | | | | 0.01 (-0.05 to 0.07) | | | 0.01 (-0.05 to 0.07) | | 0.002 |
| Other versus White | | | | | | | | | | | | |
| Step 3: Tests of moderation by HWB and EWB | | | | | | | | | | | | |
| HWB by Recession | | | -0.002 (-0.02 to 0.02) | <0.001 | | | 0.001 (-0.02 to 0.02) | | | 0.001 (-0.02 to 0.02) | | <0.001 |
| EWB by Recession | | | -0.02 (-0.03 to 0.02) | <0.001 | | | 0.02 (-0.009 to 0.02) | | | 0.02 (-0.009 to 0.02) | | <0.001 |

IL-6 = interleukin 6; CRP = C-reactive protein; CI = confidence interval; ΔR² = squared semipartial correlation; AA = African American; HWB = hedonic well-being; EWB = eudaimonic well-being. Bold: p < .05. IL-6 and CRP are natural-log transformed and standardized. Parameter estimates presented are unstandardized.

factors. That is, educationally disadvantaged and Black/AA mid-life adults reported more recession hardships than advantaged adults. Adults who identified as another minoritized group (Asian, Native Hawaiian/Pacific Islander, American Indian/Alaskan Native, and multiracial, Hispanic/Latino(a)) did not report more hardships than White respondents. However, this group is heterogeneous, and further research that is focused on more specific racial/ethnic identities and nuanced experiences of recession hardship is needed.

Consistent with prior research on recession hardship and biological outcomes (23), greater recession hardship exposure was associated with higher circulating levels of IL-6 and CRP. The associations were strong enough to be manifest even after considering other sociodemographic and health factors that may contribute to the relationships between recession hardship and inflammation. In unadjusted models, Black/AA respondents relative to White respondents had higher levels of IL-6, although adjusted associations were attenuated and no longer significant for CRP. In unadjusted models, respondents from other racial/ethnic minority groups did not differ from White respondents in IL-6 and CRP but had higher levels of IL-6 in adjusted models. Lower educational attainment was associated with higher levels of IL-6 and CRP in the unadjusted models, although the association in adjusted associations was attenuated and no longer significant for IL-6. These findings did not support differential vulnerability to recession hardship by race/ethnicity and educational attainment. When included in the model, race/ethnicity and education, as independent factors, did not further moderate the impact of recession hardship on inflammatory physiology.

Longitudinal research is needed to test whether differential exposure to recession hardships is a mediator of educational and racial/ethnic disparities in inflammatory physiology. Cross-sectional findings cannot rule out whether educationally disadvantaged and racially minoritized groups had worse physical health and possibly higher levels of inflammatory activity preceding the Great Recession, which may have engendered a greater vulnerability to recession-related hardship. Nonetheless, these initial findings point to the contribution of cumulative recession hardship as a driver of inflammatory physiology, which could synergize and potentiate the effects of other social and health factors. For example, recession-related hardships explained more variance in IL-6 and CRP when compared with other commonly considered risk factors such as smoking and chronic health conditions.

In prior MIDUS findings, indicators of HWB and EWB independently predicted cardiometabolic health outcomes (33) and buffered the negative health effects of low SES on inflammatory physiology (20,22). Furthermore, longitudinal findings from MIDUS indicated that prerecession to postrecession indicators of HWB and EWB were relatively stable and thus may be resilient to economic shocks (31). Exploratory analyses in the current study examined HWB and EWB as moderators of the association between recession hardship and inflammatory markers and examined evidence of mitigation or disabling of the influence of well-being on inflammatory markers. None of the two-way or three-way interactions were statistically significant for HWB or EWB. Tests of interaction effects require high levels of statistical power. When data become available, inclusion of the third wave of the core MIDUS biomarker sample will help to increase the sample size. Other cohort studies that were similarly in progress before and after the

Great Recession, or during other periods of social and economic upheaval are needed to examine the interactive effects of recession hardship, social disadvantage, and psychological resources in relation to physical health. For example, cohort studies that were ongoing in 2020 have the potential to examine the social and economic derailments that accompanied the COVID-19 pandemic.

The current findings contribute to the extant literature by testing the linkages between recession hardship and two inflammatory proteins that are often implicated as pathophysiological precursors of symptomatic disease and mediators of the adverse health effects of stressful life events. Although many of the effects identified in our models were modest, the findings revealed potential pathways through which economic hardship and societal discord can undermine health. Even at lower, subclinical levels, chronic increases in IL-6 and CRP values convey a dysregulation of inflammatory physiology with implications for future health, especially as adults age. For example, systemic inflammation is a key pathway that drives the progression of atherosclerotic cardiovascular disease (20,68), which is an underlying cause of about 50% of all mortality in affluent modern countries (69). Even with aggressive interventions to reduce inflammatory activity, IL-6 levels before treatment have been shown to predict an increased risk of future cardiovascular events and all-cause mortality (21). These findings underscore the need for social policies that lessen the cumulative exposure to recession hardships that are disproportionately experienced by educationally disadvantaged and minoritized groups.

The inclusion of multiple sociodemographic and health covariates, the cross-sectional design, and relatively small sample size for this type of analysis may have precluded our ability to detect interactions between recession hardship, preexisting social vulnerability, and the buffering benefits of PWB. Future research with larger sample sizes is needed to clarify the mediating and moderating mechanisms that contribute to the effects of preexisting vulnerability and recession-related hardships on inflammatory markers. Health-promoting behaviors and effective emotion regulation may be challenged by inequities in access to wealth (3) and health care (37) that worsened after the Great Recession. Further analyses of prerecession and postrecession assessments of emotion regulation, health behaviors, and social inequities in the MIDUS cohort will be able to elucidate more mediators and psychosocial moderators of the health impact of recession-related hardships.

Several limitations warrant consideration and should be addressed in future research. The effect sizes for the main effects predicting health outcomes were small, which is not uncommon in complex analyses of large heterogeneous samples (70). Similar effect sizes were found in other analyses of socioeconomic and psychological predictors of inflammatory physiology (27). In addition, many factors may have contributed to the magnitude of the observed associations. First, obesity has a strong influence on CRP and IL-6, and recession hardships were positively associated with BMI. Increases in adiposity, changes in diet, and increases in sedentary inactivity may therefore lie on the causal pathway between recession hardships and inflammatory proteins, but cross-sectional mediational analyses are not usually recommended (71). Second, there was sample selectivity of MIDUS respondents in the biomarker subproject. Respondents who agreed to the biomarker assessment tended to be more highly educated than the larger survey cohort. Black/AA respondents were mostly from a city-specific sample. Although inclusion of participants from

Milwaukee, WI, provided the opportunity to test for disparities in recession hardship exposure and health by race, it is unknown whether the findings generalize to other racial and ethnic groups in all urban and rural regions of the United States. Milwaukee is known to be a particularly segregated city (72), and this subsample would have been exposed to both discrimination (73) and adverse economic conditions (74). Thus, some of the racial differences in the effects of recession hardship exposure could be larger than in other geographical regions. Another limitation was the categorization of other racial and ethnic groups (e.g., Hispanic/Latino(a), Asian, Native Hawaiian/Alaskan Native) into one heterogeneous category. This categorization likely masked some of the additional variation in recession hardship exposure and vulnerability that would be important to consider in these populations.

Despite these limitations, the present study made the following contributions. First, the investigation incorporated a comprehensive measure of recession-related experiences ranging from job loss to financial strain and to housing hardship assessed 3 to 4 years after the official end of the Great Recession in 2009. Because of policy decisions at the time that favored economic austerity, it is now known that the economy did not fully recover until 2015 (15). The current study thus provided a more complete picture of the cumulative impacts of the Great Recession compared with some studies conducted in the immediate aftermath. Second, although previous studies have focused primarily on more immediate health outcomes, this inquiry examined inflammatory physiology about 5 to 8 years after the start of the Great Recession. Third, the effects of recession hardship on inflammatory markers were examined in the context of preexisting vulnerabilities (i.e., minoritized race/ethnicity status and low educational attainment) and individual differences in PWB.

CONCLUSIONS

This analysis integrated biopsychosocial and economic theories of health inequities with an assessment of two physiological indices of health to identify the ways in which the impact of the Great Recession was contoured by race and education and resilience factors like PWB. The findings convey that educational and racial disadvantage increased exposure to a severe economic recession. Differential exposure to recession hardship may contribute to disparities in health and longevity. Specifically, cumulative recession hardship was linked to two indices of inflammatory activity, which are predictors of long-term health. Future research is needed to identify the other pathways and processes through which recession hardship activates inflammatory physiology and undermines physical and mental health.

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