# **REGULAR ARTICLE**

# Effects of the Great Recession on Educational Disparities in Cardiometabolic Health

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## Abstract

**Background** Macroeconomic crises can exaggerate existing educational disparities in health. Few studies, however, have examined whether macroeconomic crises get under the skin to affect educational disparities in health-related biological processes.

*Purpose* This study aimed to examine the effect of the economic recession of 2008 (i.e., Great Recession) on educational disparities in cardiometabolic risk and self-reported psychological distress.

*Methods* Data were drawn from two subsamples of the Midlife in the United States (MIDUS) study: the second wave of the MIDUS sample (pre-recession cohort, N = 985) and the refresher sample (post-recession cohort, N = 863). Educational attainment was categorized into high school education or less, some college, and bachelor's degree or higher. Outcomes included metabolic syndrome, C-reactive protein, and interleukin-6, as well as self-reported perceived stress, depressive symptoms, and financial distress.

*Results* Results showed that having a bachelor's degree or higher (compared to having a high school education or less) was more strongly associated with decreased metabolic syndrome symptoms in the post-recession cohort than the pre-recession cohort, above and beyond demographic, health, and behavioral covariates. These findings did not extend to systemic inflammation or psychological distress.

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*Conclusions* Our findings suggest that chronic macroeconomic stressors may widen the educational gap in physical health, particularly cardiometabolic health, by modifying biological and anthropometric risk factors implicated in metabolic syndrome.

Keywords Great Recession · Metabolic syndrome · Inflammation · Psychological distress · Education · Socioeconomic status

# Introduction

The economic recession of 2008, also known as the Great Recession, started in December 2007 and ended in June 2009 [1]. It was recognized as the longest recession experienced by the U.S. post-World War II [2]. During the Great Recession, the unemployment rate nearly doubled, increasing from 5.0% to 9.5% in June 2009 [1], the house price declined by 27.5%, and the median net worth of American families dropped by 38.8% [3]. Recession-related hardships have been linked to poor mental and physical health outcomes, including depression [4], self-reported physical health [5], and cardiovascular disease risk [6].

Notably, the Great Recession did not affect all segments of the U.S. population equally. For example, low-education workers have been found to be disproportionally impacted by the Great Recession [7, 8]. In 2010, at the unemployment peak, the unemployment rate for individuals without a high school degree was three times higher than those of college graduates [7]. During the recovery from the Great Recession between 2010 and 2016, individuals with a bachelor's degree were three times more likely to be hired than those with just some college experience (but no degree) and 10 times more likely to be hired than those with a high school diploma or less [8]. The Great Recession is an important

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example of how the macrosocial environment, including economic downturns and poor recovery, may impact the health of vulnerable populations, such as those with low educational attainment. However, whether and how this macroeconomic event impacted health or exacerbated the pre-recession educational gradient in health remains understudied.

Education, together with other indicators of socioeconomic status (SES), such as income and occupation, is a robust social determinant of mental and physical health [9, 10]. Recent data suggest that the educational gradient in health, particularly in the USA, has been growing despite the fact that its eradication has been a public health priority for years [11, 12]. For example, low educational attainment has been consistently associated with increased risk factors for cardiovascular disease, the leading cause of death in the USA [13, 14]. In addition to clinical endpoints, educational disparities are also evident at the preclinical level (e.g., biological risk). For example, individuals with lower education (vs. higher education) are more likely to be diagnosed with metabolic syndrome (MetS) and have higher levels of systemic inflammation [15–17], both of which are interrelated risk factors for cardiometabolic disease [18, 19].

A few studies have examined the impact of macroeconomic crises, including the Great Recession, on education-driven and, more generally, SES-driven health disparities. For example, using data collected before and during the Great Recession in Spain, Bartoll and colleagues [20] found a widening gap in self-reported health as a function of education among adult men, but not women. In another study, educational disparities in selfreported physical health were more pronounced during the Southeast Asian economic crisis of the late 1990s in South Korea compared to the period preceding the crisis [21]. Data from the USA also support the hypothesis that economic recessions exaggerate SES disparities in health. When comparing two cohorts from the Midlife in the United States (MIDUS) study recruited before and after the Great Recession, Goldman et al. [22] found that individuals with low SES in the post-recession cohort were more likely to experience increased levels of psychological distress (e.g., negative affect) and decreased levels of psychological well-being (e.g., positive affect) than their low-SES counterparts in the pre-recession cohort. Interestingly, a similar study conducted in England (i.e., Health Surveys of England) did not find any associations between the Great Recession and the widening educational disparities in self-reported mental health [23]. The majority of studies in this literature focused on self-reported mental health, which is often non-specific and more subjective than objective measures of health. Few studies have examined the effects of the Great Recession on educational disparities in biological risk.

This work is necessary to understand whether global stressors, such as macroeconomic crises, exacerbate educational disparities in medical morbidity and mortality [11, 12].

Differences in stress exposure are one key mechanism hypothesized to impact the effects of the Great Recession on educational disparities in biological risk [24]. As discussed above, workers with lower educational attainment (e.g., high school education or less) were more likely to experience recession-related hardships, including job loss, prolonged unemployment, and financial strain following the Great Recession, than workers with higher educational attainment [7, 8, 25]. For example, a recent study utilizing the MIDUS data collected after the Great Recession found a significant relationship between low education and increased recession hardships (e.g., job loss, declared bankruptcy) [5]. In other studies, experiences of recession-related hardships have been associated with biological risk factors, such as elevated systemic inflammation and MetS [6, 26-28]. In addition to being exposed to more stressors, individuals with lower educational attainment might also be more vulnerable to biological wear and tear of recession-related hardships because of a depleted pool of intrapersonal (e.g., mastery), interpersonal (e.g., support), and tangible (e.g., physical assets) resources at their disposal [29]. Thus, individuals with lower educational attainment may not only be more likely to experience recession-related hardships but also less likely to cope with stress effectively, putting them at higher risk of experiencing poor biological health following the Great Recession.

The primary purpose of the current study was to examine the effect of the Great Recession on educational disparities in two risk factors for cardiometabolic health: systemic inflammation indexed by circulating interleukin-6 (IL-6) and C-reactive protein (CRP) and MetS. We specifically focused on education as the sole indicator of SES due to its stability through time, an appealing property when considering different cohorts [30], the fact that education, as an individual-level indicator, is a precursor to other SES measures, including income and occupation, and the fact that it was less likely to be impacted by the economic recessions than other SES indicators, such as income. To achieve this goal, we utilized two cross-sectional samples from the MIDUS study, one recruited before and the other one after the Great Recession. We hypothesized that educational disparities in systemic inflammation and MetS would be more pronounced in the post-recession cohort than pre-recession cohort, as indicated by stronger negative associations between education and inflammation and MetS in the postrecession cohort. Given previous findings on the effects of the Great Recession on SES disparities in self-reported psychological well-being [22, 23], we also examined the effects of the Great Recession on educational disparities in self-reported psychological distress, including perceived stress, depressive symptoms, and financial distress. Previous studies have highlighted the need to assess SES-specific stress (vs. general stress [e.g., global perceived stress]) to facilitate a better understanding of SES disparities in health [31]. Also, a recent study indicated that there was a growing educational gap in financial distress between the mid-2000s and mid-2010s and that such widening educational disparities might be partially driven by Great Recession-related hardships [25]. We hypothesized that there would be stronger negative associations between education and perceived stress, depressive symptoms, and financial distress in the postrecession cohort than pre-recession cohort.

# Methods

## **Participants**

Data were drawn from the biomarker subsamples of the second wave (2004-2009) of the Midlife in the United States (MIDUS 2, M2, N = 4,963 core sample + 592 Milwaukee African American sample) and the MIDUS Refresher (MR, N = 3,577 core sample + 508 Milwaukee African American Sample) cohorts. The core MIDUS sample consisted of a national representative sample of noninstitutionalized adults initially recruited in 1995 and 1996 (MIDUS 1). The MR was a new national sample recruited in 2011–2014 to supplement the core MIDUS 1 sample with a paralleled age and gender distribution [5]. One of the purposes of the MR sample was to examine the impact of the Great Recession on health by comparing it with the pre-recession, core MIDUS sample (www.midus.wisc.edu). A subsample of 1,255 adults from M2 participated in the biomarker project between 2004 and 2009. Of the 1,255 participants, 270 (21.5%) completed the biomarker project after January 1, 2008 (i.e., after the Great Recession). These participants were dropped from the analytic sample, resulting in a final sample of 985 participants from M2 (ages 34-84, 44.5% male, 78.8% White). The biomarker subsample of MR consisted of 863 adults (ages 25-76, 47.9% male, 69.0% White), whose data were collected between 2012 and 2016.

For both M2 and MR, biological data collection involved a 24-hour stay at one of three General Clinical Research Centers (GCRCs). The M2 biomarker subsample was comparable to the M2 participants that did not participate in the biomarker project on most of the demographic variables (i.e., sex, age, race, marital status, income, body mass index [BMI], chronic conditions, physician visits, self-rated health), except for education,

with the M2 biomarker subsample being more educated. Another difference between the two samples was smoking behavior, with the M2 biomarker subsample being less likely to smoke than the rest of the M2 participants [32]. The MR biomarker subsample was comparable to the MR survey sample on sex, race, marital status, BMI, and chronic conditions. The MR biomarker subsample, however, was older, more educated, less likely to smoke, had higher income, more physician visits, and better self-related health than MR survey participants who did not participate in the biomarker project [33]. The procedure for the data collection was approved by the Institutional Review Boards at the University of Wisconsin-Madison, Georgetown University, and the University of California, Los Angeles. Written informed consent was obtained from all participants.

### Measures

### Education

Education was assessed using the highest level of educational attainment on a 12-point scale, ranging from 1 = no school/some grade school to <math>12 = doctoral/professional degree. In our study, education was categorized into three groups: high school or less (the reference group), some college, and bachelor's degree, or higher [7, 8, 20, 23]. Previous studies have shown that adults with different educational credentials (i.e., high school or less, some college, and bachelor's degree or higher) experienced distinct patterns of job loss during the Great Recession and different economic recovery trajectories after the Great Recession [7, 8]. As discussed above, such educational disparities in recession-related hardships (e.g., job loss) may be one of the critical factors contributing to the potential impact of the Great Recession on the widening educational gap in health.

## Perceived stress

Perceived stress was assessed using the 10-item Perceived Stress Scale, which was collected during the biological clinic visit [34, 35]. Each item was answered on a 5-point scale, ranging from 0 = never to 4 = very often. A composite score for perceived stress was created by summing scores across the 10 items. For a few participants with missing data on one item (i.e., 0.9% of the total M2 and MR samples), that item was replaced with the mean score of the remaining nine completed items. The Cronbach's alpha was 0.87 in the M2 subsample and 0.86 in the MR subsample.

#### Depressive symptoms

Depressive symptoms were assessed using the 20-item Center for Epidemiological Studies Depression Scale [36, 37], which was collected during the biological clinic visit. Each item was answered on a 4-point scale, ranging from 0 = rarely or none of the time to 3 = most or all of the time. A composite score for depressive symptoms was calculated by summing responses across the 20 items. Missing responses on one item (i.e., 0.8% of the total M2 and MR samples) were substituted with the average score of the remaining 19 completed items. The Cronbach's alpha was 0.90 in the M2 subsample and 0.88 in the MR subsample.

## Financial distress

Following previous MIDUS studies on financial distress [25, 38], financial distress was assessed using five items: (a) current financial situation on an 11-point scale, ranging from 0 = worst to 10 = best; (b) anticipated financial situation 10 years in the future on an 11-point scale, ranging from 0 = worst to 10 = best; (c) the amount of control over the financial situation on an 11-point scale, ranging from 0 = no control at all to 10 = very much control; (d) having enough money to meet needs on a 3-point scale, ranging from 1 = more money than neededto 3 = not enough money; and (e) difficulty in paying monthly bills on a 4-point scale, ranging from 1 = verydifficult to 4 = not at all difficult. Responses were recorded so that higher scores reflected higher levels of financial distress and then standardized and averaged to create a composite for financial distress. The Cronbach's alpha was 0.77 in both samples.

## Metabolic syndrome

MetS were assessed using the five criteria proposed by the National Cholesterol Education Program Adult Treatment Panel III [39]. The five criteria are blood pressure  $\ge 130/85$  mm/Hg, waist circumference > 102 cm for men and > 88 cm for women, triglycerides  $\ge$  150 mg/dL, high-density lipoprotein (HDL) cholesterol < 40 mg/dLfor men and < 50 mg/dL for women, and fasting glucose levels  $\geq 110 \text{ mg/dL}$ . For both samples, these five indicators were assessed using the same procedures at the same laboratories (for biomarkers). Blood pressure and waist circumference were assessed by clinicians at the GCRCs. Three consecutive blood pressure measurements were obtained, and the second and third blood measurements were averaged to calculate blood pressure. Triglycerides, HDL cholesterol, and glucose were determined using a fasting blood sample taken before breakfast on the second day of the GCRC visit. The lipid panel was assayed using a Roche Cobas analyzer (Roche Diagnostics, Indianapolis, IN). The inter-assay and intra-assay coefficient of variations (CVs) for triglycerides were, respectively, 1.0% and 1.6% in the M2, and 2.5% and 1.6% in the MR. The inter-assay CV for HDL cholesterol was 6.5% in the M2 and 3.6% in the MR, and the intra-assay CV

ranged from 1.1% to 1.4% in the M2 and MR. Glucose was assessed using an enzymatic assay on an automated analyzer (Roche Modular Analytics P) in the M2 and an enzymatic colorimetric assay on the Cobas c502 analyzer (Roche Diagnostics) in the MR. The inter-assay and intra-assay CVs for glucose were, respectively, 1.0% and 1.0% in the M2, and were, respectively, 1.0% and 1.1% in the MR. A composite score was created for MetS symptoms by a count of the criteria described above that participants met (hereafter referred to as MetS symptoms to distinguish this count variable from the binary variable indicating the presence of MetS).

# Systemic inflammation

CRP and IL-6 were determined using the same procedures at the same laboratories in the M2 and MR. Specifically, CRP was initially assayed using the BNII nephelometer (Dade Behring, Inc., Deerfield, IL). For samples with undetectable CRP, they were re-assayed using the MSD immunoelectrochemiluminescent platform (Meso Scale Diagnostics, #K151STG). Given the technical difficulties associated with the use of plasma in MSD kits, the CRP assays beginning in 2016 were conducted on serum using the MSD technology. Corrections were applied for CRP values assayed using the MSD technology with serum at the MIDUS BioCore. The inter-assay and intra-assay CVs ranged from 2.1% to 5.7% in the M2 and ranged from 1.1% to 4.4% in the MR. IL-6 was assayed using the Quantikine high-sensitivity ELISA kit (R&D Systems, Minneapolis, MN). The inter-assay and intraassay CVs were, respectively, 12.3% and 3.3% in the M2, and were, respectively, 15.7% and 3.7% in the MR. The natural log transformation was employed for CRP and IL-6 to correct skewed distribution.

### Covariates

Some key demographic, health, and behavioral covariates identified from prior studies on cardiometabolic health were included in the current analyses [33, 40]. Demographic covariates included sex (0 = male, 1 = female), race (0 = White, 1 = others [i.e., Black/African]American, Native American or Alaska Native Aleutian Islander/Eskimo, Asian, Native Hawaiian or Pacific Islander, and others to be specified]), marital status (0 = others, 1 = married), and age. Health covariates included medications taken to manage hypertension, high cholesterol, and diabetes (0 = no, 1 = yes) and the total number of chronic health conditions (e.g., stroke, fever) out of a list of 30 conditions. Behavioral covariates included current or ever smoked regularly  $(0 = n_0)$ 1 = yes), alcohol use (0 = non-regular alcohol use [< 3 days per week],  $1 = \text{regular alcohol use} [\le 3 \text{ days per}]$ week]) [41], and regular physical activity for 20 min or more at least 3 times/week (0 = no, 1 = yes). Smoking

was initially categorized into four groups: current smoke regularly, ever smoked regularly, ever smoked but not regularly, and never smoked. Given that there were no differences in cardiometabolic risk factors between individuals who had never smoked and those who had ever smoked but not regularly and between participants who had ever smoked regularly and those who were current smokers (ps > .05), smoking was dichotomized into two categories as described above.

#### Statistical Analyses

The *t*-tests and chi-square tests were conducted to test potential differences in the sample characteristics between the pre- and post-recession cohorts (M2 vs. MR). Due to age differences in the M2 and MR, partial correlations between study variables were carried out while controlling for age. Moderation models were performed in Mplus 7.0 [42]. Moderation models for continuous outcome variables (e.g., CRP) were conducted using the ordinary least squares regression, and models for the count outcome variables (i.e., MetS symptoms) were conducted using the Poisson regression. The effects of the Great Recession on educational disparities in health outcomes were determined by comparing the strength of the association between education and health outcomes between two cohorts (i.e., M2 vs. MR). Models were first performed with age being controlled for (Model 0), then adjusted for other demographic and health covariates (Model 1), and additionally adjusted for behavioral covariates (Model 2). The percentage of participants with missing data was about 4.3%, and multiple imputation (i.e., 20 imputed datasets) was employed to handle missing data [43].

A few sensitivity analyses were performed. First, to address the potential confounding effect attributed to age differences between M2 and MR, we tested the potential three-way interaction among cohort, age, and education. Second, moderation analyses were rerun by treating MetS symptoms as a binary outcome (0 = no presence of MetS, 1 = presence of MetS, usinga cutoff score of 3) [39]. Moderation analyses werealso performed for the presence of each of the fiveMetS symptoms <math>(0 = no, 1 = yes). Third, we reran the moderation model for CRP by dropping participants having CRP values greater than 10 mg/L (N = 89)from the analyses [44].

# Results

Table 1 displays the characteristics of participants in the M2 and MR. The M2 biomarker subsample was comparable to the MR biomarker subsample as to sex, physical

activity, and the number of chronic health conditions (ps > .05). However, compared to participants in the MR, participants from the M2 were older and more likely to be white, married, and current/ever regular smoker (ps < .01). They were also more likely to use hypertension, high cholesterol, and diabetes medication and were less educated and less likely to drink alcohol regularly (ps < .05). There were no differences in perceived stress, depressive symptoms, IL-6, or CRP between the M2 and MR samples (ps > .10), but participants from the M2 had more MetS symptoms than those in the MR (t = -3.78, p < .001). In addition, participants in the MR reported higher levels of financial distress than their counterparts in the M2 (t = 3.96, p < .001). Table 2 presents the partial correlations between study variables by cohort, adjusting for age.

Moderation model revealed significant interactive effects between cohort and education on MetS symptoms (Model 0: cohort × some college: b = 0.22, p = .010; cohort × bachelor's degree or higher: b = 0.31, p < .001). Simple slope analyses showed that having a bachelor' degree or higher and having some college (compared to having a high school education or less) were both more strongly associated with decreased MetS symptoms in the MR than in the M2 (b = -0.50, p < .001 vs. b = -0.19, p = .001; b = -0.20, p = .004 vs. b = 0.03, p = .60, respectively). That is, the differences in MetS symptoms between high school or less and bachelor's degree or higher and between high school or less and some college were larger in the MR than M2, indicating that educational disparities in MetS symptoms were more pronounced in the post-recession cohort than pre-recession cohort. The larger differences in MetS symptoms between high school or less and bachelor's degree or higher in the postrecession cohort remained similar after the addition of other demographic, health, and behavioral covariates (Model 2; cohort × bachelor's degree or higher: b = 0.20, p = .019; simple slope: b = -0.35, p < .001 vs. b = -0.16, p = .005). The differences in MetS symptoms between high school or less and some college in the MR and M2 also remained similar after adjusting for other demographic, health, and behavioral covariates, though the interactive effect became statistically nonsignificant (cohort × some college: b = 0.16, p = .052; simple slope: b = -0.14, p = .031 vs. b = 0.02, p = .68). To further visualize these interactive effects, mean values of MetS symptoms were estimated from Model 2 using the first imputed dataset (the results were similar across 20 imputed datasets) and plotted using package ggplot in R [45]. Differences in MetS symptoms between high school or less and bachelor's degree or higher and between high school or less and some college were statistically significant in both cohorts, except that there were no differences between high school or less and some college in the M2 (see Fig. 1).

#### Table 1 Descriptive characteristics by cohort

Variables (mean, SD)	Overall ( <i>N</i> = 1,848)	Cohort		
		M2 ( <i>N</i> = 985)	MR ( <i>N</i> = 863)	р
Age (year)	53.3 (12.8)	55.4 (11.8)	50.8 (13.4)	<.001
Female ( <i>n</i> , %)	997 (54.0)	547 (55.5)	450 (52.1)	.15
White ( <i>n</i> , %)	1,367 (74.0)	775 (78.8)	592 (69.0)	<.001
Married $(n, \%)$	1,146 (62.0)	643 (65.3)	503 (58.5)	.003
Current or ever smoked regularly $(n, \%)$	820 (44.4)	478 (48.5)	342 (39.6)	<.001
Regular alcohol use $(n, \%)$	412 (22.3)	191 (19.4)	221 (25.6)	.001
Having physical activity $(n, \%)$	1,382 (74.8)	752 (76.3)	630 (73.0)	.099
Chronic health conditions	2.45 (2.51)	2.52 (2.53)	2.37 (2.49)	.19
Medication use $(n, \%)$	880 (47.6)	492 (49.9)	388 (45.0)	.032
Education $(n, \%)$				
High school or less	430 (23.3)	281 (28.6)	149 (17.3)	
Some college	556 (30.1)	293 (29.8)	263 (30.5)	
Bachelor's degree or higher	859 (46.5)	409 (41.6)	450 (52.2)	<.001
Perceived stress	12.4 (6.4)	12.3 (6.3)	12.5 (6.4)	.45
Depressive symptoms	9.0 (8.1)	8.8 (8.3)	9.3 (7.9)	.23
Financial distress	0.0 (0.8)	-0.1 (0.8)	0.1 (0.8)	<.001
Waist circumference (centimeters)	98.5 (18.2)	98.2 (17.3)	98.8 (19.2)	.44
Systolic blood pressure (mm Hg)	130.0 (17.7)	132.0 (18.3)	127.7 (16.6)	<.001
Diastolic blood pressure (mm Hg)	76.7 (10.4)	75.8 (10.5)	77.7 (10.3)	<.001
Triglycerides (mg/dL)	126.2 (116.0)	134.3 (142.5)	116.9 (73.8)	.001
HDL cholesterol (mg/dL)	56.6 (18.6)	54.5(17.5)	59.0 (19.6)	<.001
Fasting glucose (mg/dL)	101.5 (25.7)	101.4 (25.5)	101.6 (26.0)	.81
Metabolic syndrome symptoms	1.7 (1.3)	1.9 (1.3)	1.6 (1.3)	<.001
Presence of metabolic syndrome $(n, \%)$	494 (27.1)	298 (30.7)	196 (23.0)	<.001
Interleukin-6 (pg/mL)	2.9 (2.7)	3.0 (3.0)	2.8 (2.4)	.13
C-reactive protein (µg/mL)	3.0 (5.1)	3.1 (5.1)	3.0 (5.2)	.56

*Note.* M2 = the second wave of Midlife in the United States; MR = Midlife in the United States Refresher. *p* values from *t*-tests for continuous variables and chi-square tests for categorical variables.

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Table 2	Partial correlation	between study	variables by	v cohort	while controlling for age
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Variables	1	2	3	4	5	6	7
1. Education	-	12***	16***	26***	13***	15***	15***
2. Perceived stress	14***	-	.75***	.39***	.08*	.11**	.04
3. Depressive symptoms	19***	.74***	-	.38***	.09**	.14***	.08*
4. Financial distress	32***	.37***	.35***	-	.11***	.21***	.15***
5. Metabolic syndrome symptoms	23***	.13***	.16***	.20***	-	.27***	.31***
6. Interleukin-6	21***	.10**	.22***	.23***	.42***	-	.51***
7. C-reactive protein	25***	.11**	.15***	.18***	.39***	.60***	-

*Note.* The upper diagonal matrix displays correlation matrix for the second wave of Midlife in the United States (MIDUS 2, M2) biomarker subsample, and the lower diagonal matrix displays correlation matrix for MIDUS refresher (MR) biomarker subsample. Education was treated as a continuous variable.

p < .05; p < .01; p < .001; c .001.

Similarly, there was an interactive effect between education and cohort on IL-6 (Model 0: cohort × bachelor's degree or higher: b = 0.19, p = .038, but not cohort × some college: b = 0.17, p = .095). Simple slope analyses showed that having a bachelor's degree or higher (compared to having a high school education or less) was more strongly associated with decreased IL-6 in the MR than M2 (b = -0.46, p < .001 vs. b = -0.27, p <

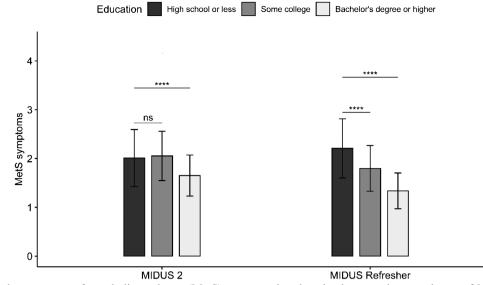


Fig. 1. Estimated mean scores of metabolic syndrome (MetS) symptoms by education between the second wave of Midlife in the United States (MIDUS 2) biomarker subsample and MIDUS Refresher biomarker subsample. Covariates, including age, sex, race, marital status, medication use, chronic health conditions, smoking, alcohol use, and physical activity, were controlled (Model 2). Error bar represents 95% CI; ns, not significant at p < .05; \*\*\*\* p < .0001.

.001), indicating larger differences in IL-6 between high school or less and bachelor's degree or higher in the postrecession cohort. Such a pattern of differences, however, became nonsignificant when controlling for health and behavioral covariates (see Table 3).

Cohort also moderated the relationship between education and CRP (Model 0: cohort  $\times$  some college: b = 0.32, p = .048; cohort × bachelor's degree or higher: b = 0.39, p = .007). Simple slope showed that having a bachelor' degree or higher and having some college (compared to having a high school education or less) were more strongly associated with decreased CRP in the MR than M2 (b = -0.80, p < .001 vs. b = -0.41, p < .001.001; b = -0.40, p = .001 vs. b = -0.08, p = .44, respectively). Similarly, these results indicated that there were larger differences in CRP between high school or less and some college and between high school or less and bachelor's degree or higher in the post-recession cohort. This pattern of differences in CRP between high school or less and some college became nonsignificant when controlling for other demographic and health covariates (Model 1; cohort × some college: b = 0.22, p = .16). The differences in CRP between high school or less and the bachelor's degree or higher in the MR and M2 remained of comparable magnitude after including other demographic and health covariates (Model 1; cohort  $\times$ bachelor's degree or higher: b = 0.30, p = .030; simple slope: b = -0.59, p < .001 vs. b = -0.29, p = .001) as well as further adjusting for behavioral covariates; however, they failed to reach statistical significance in the latter model (Model 2; cohort  $\times$  bachelor's degree or higher: b = 0.27, p = .051; simple slope: b = -0.52, p < .001 vs. b = -0.25, p = .005).

There were no interactive effects between education and cohort on perceived stress (Model 0: cohort  $\times$  some college: b = -0.65, p = .43; cohort × bachelor's degree or higher: b = -0.12, p = .88) or depressive symptoms (cohort × some college: b = -0.59, p = .59; cohort × bachelor's degree or higher: b = 0.42, p = .66). These results remained statistically nonsignificant after controlling for health and behavioral covariates (see Table 4). Cohort, however, moderated the association between education and financial distress (Model 0: cohort × bachelor's degree or higher: b = 0.20, p = .048). Simple slope analyses showed that having a bachelor's degree or higher was more strongly associated with decreased levels of financial distress in the MR than in the M2 (b = -0.67, p < .001 vs. b = -0.47, p < .001). The interactive effect between education and cohort on financial distress did not reach statistical significance when adjusting for all covariates (see Table 4).

## Sensitivity analyses

Moderation analyses for potential age differences showed that there were no three-way interactive effects among cohort, age, and education on any health outcomes (*ps* > .10). The interactive effects between education and cohort on MetS remained significant (cohort × some college: b = 0.47, p = .007; cohort × bachelor's degree or higher: b = 0.42, p = .011), when MetS was treated as a binary variable (about 27% reported the presence of MetS). After controlling for all covariates, significant interactive effects emerged between cohort and some college (b = 0.41, p = .021), but not between cohort and bachelor's degree or higher (b = 0.29, p = .089). Results

Variables	Metabolic syndrome symptoms	e symptoms	Interleukin-6		C-reactive protein	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Some college (vs. HS or less)	-0.15 (0.07)*	-0.14(0.07)*	-0.15 (0.07)*	-0.14(0.07)*	-0.29 (0.12)*	-0.28 (0.12)*
Bachelor's or higher (vs. HS or less)	$-0.39(0.07)^{***}$	$-0.35(0.07)^{***}$	-0.26 (0.07)***	-0.21 (0.07)**	$-0.59(0.11)^{***}$	$-0.52(0.11)^{***}$
Cohort	-0.04(0.07)	-0.05(0.06)	-0.07 (0.07)	-0.07 (0.07)	-0.19(0.11)	-0.18 (0.12)
Some college × cohort	0.15(0.09)	0.16(0.08)	0.08(0.09)	(0.08)	0.22 (0.15)	0.23 (0.15)
Bachelor's or higher × cohort	$0.21 (0.09)^{*}$	$0.20(0.08)^{*}$	(60.0) $(0.00)$	(0.01 (0.09)	$0.30~(0.14)^{*}$	0.27(0.14)
Female	$-0.17(0.03)^{***}$	$-0.20(0.03)^{***}$	0.01 (0.03)	0.00(0.03)	$0.37 (0.06)^{***}$	$0.35 (0.06)^{***}$
White	0.00(0.04)	-0.03(0.04)	$0.23 (0.04)^{***}$	$0.20 (0.04)^{***}$	$0.16(0.07)^{*}$	0.12(0.07)
Married	-0.03(0.04)	-0.01(0.04)	$-0.13(0.04)^{**}$	$-0.10(0.04)^{**}$	-0.10(0.06)	-0.07 (0.06)
Age	(0.00 (0.00)	0.00(0.00)	$0.01 (0.00)^{***}$	$0.01 (0.00)^{***}$	-0.00(0.00)	-0.00(0.00)
Chronic health conditions	$0.03 (0.01)^{***}$	$0.03 (0.01)^{***}$	$0.05 (0.01)^{***}$	$0.04 (0.01)^{***}$	$0.04\ (0.01)^{**}$	$0.04 (0.01)^{**}$
Medication use	$0.35 (0.04)^{***}$	$0.34 (0.04)^{***}$	$0.24 (0.04)^{***}$	$0.23 (0.04)^{***}$	$0.36\ (0.06)^{***}$	$0.34 (0.06)^{***}$
Current/ever smoked regularly		0.01(0.03)		0.08(0.04)*		0.08 (0.06)
Physical activity		$-0.18(0.04)^{***}$		$-0.20(0.04)^{***}$		$-0.34(0.06)^{***}$
Regular alcohol use		$-0.22(0.04)^{***}$		$-0.11 (0.04)^{**}$		$-0.17 (0.07)^{**}$
<i>Note.</i> Unstandardized coefficients (standard errors) were presented. Model 0 controlled for age, and results were reported in the main text. Model 1 controlled for demographic and health covariates. Model 2 controlled for demographic and health covariates. Model 2 controlled for demographic m(R)	l errors) were presentee mooranhic health and	<ol> <li>Model 0 controlled for the heavioral covariates.</li> </ol>	r age, and results were re Tohort was coded as 0 =	ported in the main text.	Model 1 controlled for d	emographic and Refresher_MR)

 Table 3
 Results of moderation models for metabolic syndrome and systemic inflammation

States Refresher (MILDUS Refresher, MIK) United in the Miduite ⊳ as Conort was coded health covariates; Model 2 controlled for demographic, health, and behavioral covariates. and 1 = the second wave of MIDUS (MIDUS 2, M2). HS = high school.

p < .05; p < .01; p < .01; p < .001.

Variables	Perceived stress		Depressive symptoms	ns	Financial distress	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Some college (vs. HS or less)	0.41 (0.61)	0.48 (0.61)	0.39 (0.75)	0.48 (0.74)	-0.20(0.08)*	-0.19(0.08)*
Bachelor's or higher (vs. HS or less)	-0.37(0.56)	-0.15(0.56)	-0.93 (0.66)	-0.61 (0.65)	$-0.45(0.08)^{***}$	-0.42 (0.08)***
Cohort	1.10(0.60)	1.21(0.60)*	1.10 (0.75)	1.26 (0.74)	$-0.18(0.08)^{*}$	-0.18(0.08)*
Some college $\times$ cohort	-1.49(0.79)	-1.49(0.78)	-1.92(0.99)	-1.91(0.98)	0.00(0.10)	0.00(0.10)
Bachelor's or higher × cohort	-0.98 (0.73)	-1.08 (0.72)	-1.08(0.89)	-1.22(0.87)	0.08(0.09)	0.07 (0.09)
Female	0.37 (0.29)	$0.54\ (0.30)$	-0.34(0.36)	-0.15(0.37)	0.01 (0.04)	0.01 (0.04)
White	0.63(0.36)	0.59(0.36)	$0.94 (0.46)^{*}$	0.85(0.46)	0.10(0.04)*	0.09~(0.04)*
Married	-0.26 (0.32)	-0.14(0.32)	$-1.56(0.39)^{***}$	$-1.39(0.39)^{***}$	$-0.25(0.04)^{***}$	-0.23 (0.04)***
Age	$-0.12(0.01)^{***}$	$-0.13(0.01)^{***}$	$-0.13(0.01)^{***}$	$-0.14(0.02)^{***}$	$-0.01(0.00)^{***}$	$-0.01(0.00)^{***}$
Chronic health conditions	$0.72 (0.08)^{***}$	$0.70 (0.08)^{***}$	$1.14 (0.10)^{***}$	$1.11 (0.10)^{***}$	$0.08(0.01)^{***}$	$0.08 (0.01)^{***}$
Medication use	0.11 (0.32)	0.05(0.32)	0.37 (0.40)	0.28(0.39)	0.03(0.04)	0.02(0.04)
Current/ever smoked regularly		0.58(0.30)		0.72 (0.37)*		$0.07 (0.04)^{*}$
Physical activity		$-1.10\left( 0.34 ight) ^{**}$		-1.71 (0.42) <sup>***</sup>		$-0.11(0.04)^{**}$
Regular alcohol use		0.80(0.34)*		0.93(0.44)*		-0.06 (0.04)
<i>Note.</i> Unstandardized coefficients (standard errors) were presented. Model 0 controlled for age, and results were reported in the main text. Model 1 controlled for demographic and health covariates: Model 2 controlled for demographic and health covariates. Model 2 controlled for demographic m(R)	d errors) were presente emographic, health, an	ed. Model 0 controlled for the bayional covariates.	r age, and results were re Cohort was coded as 0 =	sported in the main text. = Midlife in the United S	Model 1 controlled for d tates Refresher (MIDUS	emographic and Refresher. MR)

Table 4 Results of moderation models for perceived stress, depressive symptoms, and financial distress

ssher, MK) neatth covariates; Model z controlled for demographic, health, and benavand 1 = the second wave of MIDUS (MIDUS 2, M2). HS = high school.

p < .05; p < .01; p < .01; p < .001.

for individual MetS symptoms showed significant interactive effects between education and cohort on waist-hip ratio (cohort  $\times$  some college: b = 0.44, p = .009; cohort × bachelor's degree or higher: b = 0.49, p = .002), glucose (cohort × some college: b = 0.50, p = .006; cohort × bachelor's degree or higher: b = 0.69, p < .001), and high-density lipoproteins (cohort × bachelor's degree or higher: b = 0.37, p = .029), but not blood pressure or triglycerides (ps > .40). The results remained significant after controlling for all covariates, except that the interactive effect between education and cohort on high-density lipoproteins became statistically nonsignificant (cohort × bachelor's degree or higher: b = 0.28, p = .099). In addition, results from sensitivity analyses for CRP were similar to those reported above. That is, there was a significant interactive effect between education and cohort on CRP when including age as a covariate (cohort × bachelor's degree or higher: b = 0.30, p = .023); however, this interactive effect became statistically nonsignificant when adjusting for other demographic, health, and behavioral covariates (cohort × bachelor's degree or higher: b = 0.22, p = .084).

# Discussion

Educational disparities in health are ubiquitous. Individuals with less education score higher on various indexes of biological risk and have higher rates of medical morbidities and early mortality than their counterparts with more education [15–17, 46]. Whether chronic global stressors, such as the Great Recession, have widened educational disparities in biological processes contributing to the socioeconomic gradient in health remains to be established. The key objective of the current study was to examine this research question by comparing the strength of the relationship between educational attainment and three markers of cardiometabolic risk (i.e., IL-6, CRP, and MetS) in two cohorts, one collected before the Great Recession and one collected during the years following the Great Recession. We found that educational disparities in MetS symptoms were larger in the post-recession cohort than in pre-recession cohort. Specifically, having a bachelor's degree or higher and having some college (compared to having a high school education or less) were more strongly associated with decreased MetS symptoms in the post-recession cohort than pre-recession cohort. The size of these effects remained of similar magnitude after the inclusion of demographic, health, and behavioral covariates, though differences in the strength of the association between having some college and MetS symptoms in these two cohorts did not reach statistical significance. Also, educational disparities in CRP (but not IL-6) were more salient in the post-recession cohort than pre-recession cohort. Similarly, we found that having a bachelor's degree or higher was more strongly associated with decreased CRP in the post-recession cohort than the pre-recession cohort. The larger educational differences in CPR between individuals with a bachelor's degree or higher and those with a high school education or less in the postrecession cohort remained similar after the inclusion of demographic and health covariates, though they did not reach statistical significance when behavioral covariates were included in the model.

Interestingly, we found that the widening educational disparities in the post-recession period were only evident for MetS and CRP, but not IL-6, though IL-6 plays a key role in inducing the release of CRP. One potential explanation may be related to the potential differential effects of acute and chronic stress on the innate immune system, with the former more likely to modulate the early phases of the inflammatory response (i.e., the releases of cytokines) and the latter more likely to affect acutephase proteins, such as CRP [47, 48]. Our findings concerning CRP were no longer significant after adjusting for behavioral covariates (e.g., smoking), perhaps suggesting that some lifestyle behavioral factors might act as mechanisms for the growing educational gap in health outcomes, including systemic inflammation [49]. Coincidentally, recent evidence has shown a decreased prevalence of smoking during the post-recession period among U.S. adults, for except those with the lowest SES [50]. Whether potential SES differences in behavioral changes in response to the Great Recession act as an intermediary for the association between the Great Recession and the widening educational disparities in health need to be formally tested using longitudinal research designs.

A critical strength of the current study was the inclusion of biological risk factors as outcomes. Much of the work on how macroeconomic crises affect educationdriven, and more generally, SES-driven health disparities, have focused on mortality and self-reported health outcomes. There are limited published data on the biological processes through which macroeconomic stressors may get under the skin to affect existing SES disparities in health-related biological processes. Notably, a few previous studies have indicated the adverse effects of macroeconomic crises on certain biological risk factors, including systemic inflammation and MetS [6, 27, 28]. Our results expand this growing literature by suggesting that the Great Recession may also exaggerate the existing educational disparities in biological risk, which, in turn, may contribute to educational disparities in morbidity and mortality [11, 12, 51]. Future studies, however, are needed to explicitly test the potential mediation role of biological risk factors examined in

this study in linking economic recessions to educational disparities in morbidity and early mortality. Also, our results seem to suggest that such widening educational disparities in biological risk factors may be likely driven by elevated health gains among individuals with high education in the post-recession cohort. However, given the cross-sectional nature of data in this study, longitudinal studies are warranted to validate this proposition.

Another objective of the current study was to examine educational disparities in self-reported perceived stress, depressive symptoms, and financial distress. Prior research, including evidence from MIDUS studies, showed that SES gradients in self-rated health and psychological and financial distress were larger following economic recessions [20, 22, 25, 52], though not all studies supported this conclusion [23]. In our study, we found no evidence suggesting that educational disparities in perceived stress and depressive symptoms changed as a function of the Great Recession. Our results are somewhat inconsistent with previous findings from the MIDUS study showing the widening SES gap in mental health in the post-recession cohort [22]. These mixed results may be due to the different sample sizes employed in the previous study (full MIDUS survey sample) and ours (restricted MIDUS biomarker subsample), resulting in lower statistical power. Alternatively, given the differences in mental health outcomes (e.g., negative affect, life satisfaction) examined in the previous MIDUS study [22] and our study, our null results may suggest that macroeconomic crises likely affect certain aspects of well-being, but not others [24]. Another possible explanation for these null effects may be related to the lack of specificity of perceived stress and depressive symptoms in reflecting distress associated with the Great Recession. Germane to this point, our study found that educational disparities in financial distress were more notable in the post-recession cohort than pre-recession cohort, though such growing educational disparities did not reach statistical significance after the inclusion of demographic, health, and behavioral covariates. Lastly, our null findings on self-reported psychological outcomes can be interpreted in light of the Skin-Deep Resilience Hypothesis, according to which striving for success in the face of adversities can lead to good psychological health at the expense of physical health [53, 54]. In the Refresher sample, it is possible that adults with low educational attainment put considerable effort when dealing with recession-related hardships, which might have resulted in faster recovery in terms of their psychological well-being but not physical health. Although tempting, this interpretation needs to be corroborated by future studies, particularly given previous mixed findings of the impact of the Great Recession on SES disparities in mental health [22, 23].

Notably, results from this study may provide important implications for understanding the impact of other global stressors on health, such as the COVID-19 pandemic, that have disastrous effects on the global economy [55]. Many countries, including the USA, have issued mandatory stay-at-home orders as a public health response to slow down the transmission of this new infectious disease. However, enforced stay-at-home orders have led to significant economic costs and increased job losses [56, 57]. In the USA, the unemployment rates rose from 3.8% in February 2020 to 13.0% in May 2020 [58]. Similar to the educational gap in job loss during the Great Recession, the unemployment rate in May 2020 among those without a high school diploma was 2.6 times higher than those with a bachelor's degree or higher [58]. Such a similar pattern of educational disparities in job loss between the Great Recession and the COVID-19 downturn may imply that the COVID-19 pandemic may also be likely to widen the existing educational disparities in health in the USA. Notably, future studies on the impact of the COVID-19 pandemic on health disparities may benefit from adopting an intersectionality framework. Recent studies have documented the differential effects the COVID-19 pandemic has had on job loss as a function of multiple identity dimensions, including education, race/ethnicity, gender, and age [59].

Results from this study should be interpreted in light of several limitations. First, in this study, we focused on educational attainment as the only measure of SES. There were several reasons for exclusively focusing on education. First, it could be argued that education is less likely to be impacted by economic crises, including the Great Recession, than other measures of SES, such as income or wealth. In this regard, the stability of education across time was an important advantage in the present study where comparisons were made between education and biological risk factors assessed before and after the recession across different cohorts. Second, education, which is often completed in early adulthood, prior to the onset of many chronic diseases, is less susceptible to concerns of reverse causality (e.g., poor health leading to lower SES). This is an important aspect to consider, particularly in cross-sectional studies such as the one reported here. Nonetheless, replicating the reported findings with other measures of SES (e.g., occupation) is an important direction for future research. For example, it is likely that global stressors, including the COVID-19 pandemic, could exaggerate the existing occupational disparities in health [9, 10]. The COVID-19 pandemic not only has disproportionately impacted individuals with different levels of educational attainment but also impacted those with different occupations [58, 60]. Studies suggest that blue-collar workers have been more likely to experience job loss than white-collar workers during the COVID-19

lockdown, likely because many blue-collar jobs are less likely to be done remotely [61].

Another limitation of our study has to do with the fact that participants in the post-recession cohort were better educated than participants in the pre-recession cohort, potentially limiting representation at the lower end of the socioeconomic spectrum in the post-recession cohort. This, however, may result in an underestimation of the effects reported in this study. Also, the age differences in the two cohorts may limit the interpretation of the results reported in the study. However, the statistically nonsignificant three-way interactions among education, age, and cohort on health outcomes suggest that the reported widening educational disparities in biological risk factors during the post-recession is not likely attributed to age differences between these two cohorts.

Also, although the widening educational disparities in MetS reported in this study were robust to the inclusion of a variety of demographic, behavioral, and health covariates, some other unexamined factors may exist that dampen the interpretation that exaggerated educational disparities in MetS were solely driven by the Great Recession. Social (e.g., changes in family structure) and economic trends (e.g., declining economic mobility), as well as changes in large social and economic policies (e.g., Affordable Care Act), might be responsible for some of the findings reported here. For example, recent studies have reported the increasing educational gap in nonmarital childbearing in the past decades [62] and the declining economic mobility [63]. Such changes in family structure and economic trends may have played a role in contributing to the increasing educational disparities in health reported in this study [64].

In addition, this study did not consider the potential differences in access to recession-related interventions across participants. Recession-related policy responses, such as the American Recovery and Reinvestment Act of 2009, are one of the important factors affecting the extent to which health disparities unfold in response to the Great Recession [24]. The widening educational gap in health reported in this study may be underestimated if recession-related interventions mitigated some of the negative health consequences for those individuals with the fewest socioeconomic resources [24]. Also, this study may not fully capture the impact of the Great Recession on educational disparities in health, given that the assessment of biological outcomes and psychological distress in the post-recession cohort was extended to 2016, far beyond the end of the recession. It should be noted, however, that previous research has reported significant effects of the Great Recession on MetS several years after the end of the economic crisis, at least among

certain populations (e.g., African American youth adults) [6]. Last, this study included a relatively homogeneous sample on the basis of race/ethnicity. Other population-based studies (e.g., Health and Retirement Study, AddHealth) may be useful to corroborate our results and fully understand how intersecting social identities (e.g., ethnic minorities with low SES) affect the impact that economic recessions have on health.

Despite these limitations, this study provides the first preliminary evidence indicating that the Great Recession might have exaggerated existing educational disparities in MetS. Our results showed larger differences in MetS symptoms between those with a bachelor's degree or higher and those with a high school education or less in the post-recession cohort than in the pre-recession cohort, above and beyond demographic, health, and behavioral covariates. These results highlight that global chronic stressors, such as the Great Recession, may get under the skin to affect biological processes.

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#### **Compliance with Ethical Standards**

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards Authors Yanping Jiang, Jennifer Morozink Boylan, and Samuele Zilioli, declare that they have no conflict of interest.

Authors' Contributions Y. Jiang conceptualized the research, analyzed and interpreted the data, and drafted the original manuscript. J. M. Boylan contributed to the conceptualization of the research and drafted the original manuscript. S. Zilioli contributed to the conceptualization of the research and provided critical revisions to the manuscript.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The data collection procedure for this study was approved by the Institutional Review Boards at the University of Wisconsin-Madison, Georgetown University, and the University of California, Los Angeles.

**Informed Consent** Informed consent was obtained from all individual participants included in the study.

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