SSM - Population Health 7 (2019) 100391

Contents lists available at ScienceDirect

SSM - Population Health

journal homepage: www.elsevier.com/locate/ssmph

Article

Longitudinal effects of race, ethnicity, and psychosocial disadvantage on systemic inflammation

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ARTICLE INFO

Keywords:
United States
Inflammation
Minority health
Discrimination
Education
Depression

ABSTRACT

Objective: Psychosocial factors likely contribute to racial and ethnic inequalities in cardiovascular diseases (CVDs). However, precise social, psychological, and physiological pathways linking race and ethnicity to the development of CVDs are not well understood. Systemic inflammation, commonly indexed by C-reactive protein (CRP), is a biomarker for CVD risk and progression. The objective of this study was to identify mediating pathways from race and ethnicity to CRP through social, psychological, and behavioral variables.

Methods: Using data from 12,382 participants aged 51 and older in the Health and Retirement Study, structural equation models tested for direct and indirect effects of race and ethnicity on CRP measured over four years through educational disadvantage, everyday discrimination, depressive symptoms, external locus of control, and smoking.

Results: Educational disadvantage mediated Black-White and Hispanic-White disparities in baseline CRP directly, as well as indirectly through elevated depressive symptoms, higher external locus of control, and smoking. Educational disadvantage also mediated Black-White and Hispanic-White disparities in CRP change directly, as well as indirectly through higher external locus of control and smoking. Independent of education, discrimination mediated Black-White differences in baseline CRP via elevated depressive symptoms, higher external locus of control, and smoking. Discrimination also mediated Black-White disparities in CRP change via external locus of control.

Conclusions: Results from this population-based, longitudinal study support the view that racially patterned social disadvantage is prospectively associated with longitudinal inflammatory processes, and some of these effects are independently mediated by psychological and behavioral factors. Biopsychosocial pathways to health disparities also differ between minority groups.

1. Introduction

Racial and ethnic inequalities have been documented for a wide array of health outcomes, including cardiovascular disease (CVD), which represents a leading cause of death among adults in the U.S. (Kochanek, Murphy, & Xu, 2015). However, pathways linking race and ethnicity to the development of CVD are poorly understood. To date, most studies have focused on individual components of what is likely a much larger set of interrelated biopsychosocial processes. An improved understanding of pathways linking the experiences of racial and ethnic minority groups to increased risk could reveal opportunities for interventions to reduce CVD morbidity and combat racial and ethnic health inequalities. The current study examines mediators of racial and ethnic inequalities in systemic inflammation, which is an important indicator of CVD risk and progression (Ridker, Rifai, Rose, Buring, & Cook, 2002) that is disproportionately experienced by Black and Hispanic adults, compared with non-Hispanic Whites (Albert, 2007; Crimmins, Kim, Alley, Karlamangla, & Seeman, 2007; Mitchell & Aneshensel, 2016).

As a social construct, self-identified race and ethnicity is a proxy for exposure to racism, the organized system that phenotypically categorizes population groups and preferentially allocates societal resources to groups regarded as superior (Bonilla-Silva, 1997). Substantial research on health inequalities across racial and ethnic groups has focused on interpersonal discrimination as one pathway by which racism can affect health, but interpersonal discrimination is only one aspect of racism that can affect health (Williams & Mohammed, 2009). Institutional discrimination is another aspect of racism that may contribute to health disparities via racially patterned socioeconomic disadvantage. Socioeconomic status can be operationalized as educational attainment, income and/or occupation, but educational attainment is the main

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https://doi.org/10.1016/j.ssmph.2019.100391
Received 27 August 2018; Received in revised form 14 March 2019; Accepted 21 March 2019
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gateway to later occupational and economic status (Pearlin, Schieman, Fazio, & Meersman, 2005). In addition, educational attainment is least likely to be affected by health events after young adulthood (Crimmins, Hayward, & Seeman, 2004) and shows the strongest and most consistent independent relationships with CVD risk and health (Winkleby, Jatulis, Frank & Fortmann, 1992). Thus, the current study examined both interpersonal discrimination and educational attainment as social factors relevant to racial and ethnic inequalities in health.

The biopsychosocial approach to understanding racial and ethnic inequalities in health builds on a more general stress-coping model (Lazarus & Folkman, 1984) and posits that conceptualizing racially patterned social experiences as "stressful" may help to explain group differences in health outcomes (Clark, Anderson, Clark, & Williams, 1999). Indeed, interpersonal discrimination and low educational attainment both represent stressors with the potential to trigger psychological and physiological stress responses. Socioeconomic stressors are among the most impactful of stressful life experiences (Pearlin et al., 2005), and the experience of discrimination may be particularly harmful due to its unpredictability and uncontrollability (Williams & Mohammed, 2009).

Randomized controlled experiments directly testing whether interpersonal discrimination or low socioeconomic status causes negative psychological sequelae are not readily available. However, longitudinal studies indicate that discrimination predicts subsequent depressive symptoms, independent of baseline depressive symptoms, and not vice versa (Brown et al., 2000; Pavalko, Mossakowski, & Hamilton, 2003). Studies using instrumental variable analyses or natural experiments support the view that low educational attainment causes depressive symptoms (Chevalier & Feinstein, 2007; McFarland & Wagner, 2015; Mezuk, Myers, & Kendler, 2013). In addition to depressive symptoms, observational data indicate that individuals with lower educational attainment report less perceived control (Bailis, Segall, Mahon, Chipperfield, & Dunn, 2001; Bruce & Thornton, 2004; Pearlin, Nguyen, Schieman, & Milkie, 2007; Pudrovska, Schieman, Pearlin, & Nguyen, 2005; Schieman, 2001), slower increases in perceived control during young adulthood, and faster declines in perceived control in older adulthood (S. K. Lewis, Ross, & Mirowsky, 1999; Mirowsky & Ross, 2007; Schieman, 2001). Qualitative studies also consistently identify a perceived lack of control as a common response to discriminatory situations (Broman, Mavaddat, & Hsu, 2000; Bullock & Houston, 1987). Together, these studies support the hypothesis that interpersonal discrimination and lower educational attainment can be conceptualized as "stressful" and are linked to negative psychological sequelae, including depressive symptoms and low perceived control.

Stress-related activation of the sympathetic nervous system (i.e., norepinephrine release), as well as chronic activation of the hypothalamic-pituitary-adrenal axis (i.e., glucocorticoid resistance), both upregulate inflammatory processes that can have broad effects on cardiovascular health (Slavich & Irwin, 2014). A common marker of systemic inflammation is the level of circulating C-reactive protein (CRP), which is an acute-phase protein synthesized by the liver and secreted in response to inflammation. Growing evidence indicates that psychosocial factors are associated with inflammatory response, as indexed by CRP. For example, educational disadvantage has been linked to higher CRP levels in epidemiological studies (Ranjit et al., 2007). In addition, a recent prospective study of approximately 2500 women found that greater discrimination predicted higher CRP levels over seven years (Beatty Moody, Brown, Matthews, & Bromberger, 2014), extending previous cross-sectional findings (Friedman, Williams, Singer, & Ryff, 2009; T. T.; Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010). Finally, both depressive symptoms (Howren, Lamkin, & Suls, 2009; Kop et al., 2002) and low perceived control (Elliot, Mooney, Infurna, & Chapman, 2017) have been associated with higher CRP.

A related mechanism by which racially patterned social stress (e.g., discrimination, socioeconomic disadvantage) could contribute to disparities in cardiovascular disease risk is described by the Environmental Affordances Model (Mezuk, Abdou et al., 2013). This model proposes that certain negative health behaviors can serve as self-regulatory coping strategies that help to preserve mental health in the short term but can have deleterious effects on physical health in the long term (Jackson & Knight, 2006; Jackson, Knight, & Rafferty, 2010). Smoking is one such negative health behavior with a well-documented history of negative impacts on cardiovascular health. Furthermore, despite many years of smoking cessation interventions, non-Hispanic Blacks and Hispanics are less likely to receive and use these interventions than Whites, even after controlling for socioeconomic status and healthcare utilization (Cokkinides, Halpern, Barbeau, Ward, & Thun, 2008). Thus, the Environmental Affordances Model predicts that health behaviors may also mediate links between social variables and health, as well as contribute to smaller indirect effects involving psychological variables.

The current study sought to examine psychosocial mediators of racial and ethnic health inequalities using longitudinal data from a large, nationally representative sample of Black, Hispanic, and White adults participating in the Health and Retirement Study (HRS). A mediation model simultaneously examined multiple pathways from race and ethnicity to CRP over four years through social (i.e., interpersonal discrimination, educational attainment), behavioral (i.e., smoking), and psychological (i.e., depressive symptoms, external locus of control) factors.

2. Method

2.1. Participants

Data were obtained from the Health and Retirement Study (HRS), a nationally representative sample of Americans over age 50 followed since 1992 (Sonnega & Weir, 2014). Details of the HRS longitudinal panel design, sampling, and all assessment instruments are available on the HRS website (http://hrsonline.isr.umich.edu). Participants in HRS are interviewed every two years. In 2006, the HRS initiated an enhanced face-to-face interview, which included collecting biomarker data and giving participants a psychosocial questionnaire, which they were asked to return via mail. A random one half of the sample was selected to participate in these procedures in 2006, and the other half was selected to participate in 2008. These individuals then participated in a follow-up face-to-face interview four years later (i.e., 2010 for participants originally interviewed in 2006, 2012 for participants originally interviews in 2008). In the current study, data obtained in 2006 and 2008 were combined to form a baseline wave, and data obtained in 2010 and 2012 were combined to form a follow-up wave. Biomarker response rates were 83.0% in 2006, 87.0% in 2008, 85% in 2010, and 87.0% in 2012 (http://hrsonline.isr.umich.edu).

Of participants aged 51 or older, the current study included only participants who self-identified as non-Hispanic Black, Hispanic (of any race), or non-Hispanic White. Two participants were excluded for non-availability of data on race and ethnicity, and 282 participants were excluded for non-membership in one of these three groups. Characteristics of the 12,382 individuals included in the current study are provided in Table 1. Data on education were available for 12,364 participants, data on discrimination were available for 11,197 participants, data on depressive symptoms were available on 12,380 participants, data on external locus of control were available on 11,199 participants, and data on smoking were available for 12,293 participants. Biomarker data were available for 11,747 participants at baseline (2006 or 2008) and 8307 participants at follow-up (2010 or 2012). All participants provided written informed consent, and all study procedures were approved by the University of Michigan institutional review board.

Table 1
Sample characteristics at baseline displayed as mean (standard deviation) unless otherwise indicated.

	Whole sample (N = 12,382)	Non-Hispanic Black (N = 1626)	Hispanic (N = 1117)	Non-Hispanic White (N = 9639)	Group differences
Baseline wave (% 2006)	51.3	50.2	46.9	52.0	B = W < H
Age (51–101 years)	68.8 (9.9)	67.6 (9.6)	66.2 (9.4)	69.4 (9.9)	H < B < W
Gender (% women)	58.9	64.1	61.0	57.8	W < H = B
Smoking (% yes)	13.0	17.4	12.5	12.4	H = W < B
C-reactive protein (NHANES-equivalent)					W < H < B
% Low (< 1 μg/mL)	28.9	25.3	23.3	30.1	
% Moderate (1–3 μg/mL)	33.9	26.7	36.9	34.7	
% High (> 3 μg/mL)	37.3	48.0	39.8	35.1	
Education (0–17 years)	12.5 (3.2)	11.6 (3.2)	8.9 (4.5)	13.1 (2.6)	H < B < W
Discrimination (1–6)	1.6 (0.7)	1.8 (0.9)	1.6 (0.8)	1.6 (0.7)	W = H < B
Depressive symptoms (0–10)	1.4 (2.0)	1.8 (2.1)	2.1 (2.4)	1.3 (1.8)	W < B < H
External locus of control (1–6)	2.2 (1.2)	2.3 (1.2)	2.6 (1.3)	2.2 (1.2)	W < B < H

Note. NHANES = National Health and Nutrition Examination Survey; B = Non-Hispanic Black; H = Hispanic; W = Non-Hispanic White.

2.2. Measures

2.2.1. Race and ethnicity

Race was assessed with the question "What race do you consider yourself to be: White, Black or African American, American Indian, Alaska Native, Asian, Native Hawaiian, Pacific Islander, or something else?" Ethnicity was assessed with the question, "Do you consider yourself Hispanic or Latino?" The current study included three, mutually exclusive categories: non-Hispanic Black, non-Hispanic White, and Hispanic (of any race). The largest category, non-Hispanic White, was selected as the reference group.

2.2.2. CRP

A detailed description of the HRS biomarker study is available (Crimmins et al., 2013). In brief, high-sensitivity assays primarily conducted at the University of Vermont were used to detect CRP levels in blood spots. As per HRS recommendations, the current study used National Health and Nutrition Examination Survey (NHANES)-equivalent values of CRP, which are based on serum blood. In line with American Heart Association and Centers for Disease Control and Prevention guidelines, CRP levels were categorized in terms of CVD risk: low (< 1 mg/L), moderate (1–3 mg/L), and high (> 3 mg/L) (Pearson et al., 2003).

2.2.3. Social variables

Educational attainment was self-reported from 0 to 17 years and centered at the sample median (12 years) to facilitate parameter interpretation. Interpersonal discrimination at baseline was assessed with the five-item Everyday Discrimination Scale (Williams, Yu, Jackson, & Anderson, 1997), administered as part of the psychosocial questionnaire. Items included, "You are treated with less courtesy or respect than other people," "You receive poorer service than other people at restaurants or stores," "People act as if they think you are not smart," "People act as if they are afraid of you," and "You are threatened or harassed." Items are rated for frequency on a 6-point Likert-type scale (1 = Almost every day to 6 = Never). In the current study, mean scores were reversed prior to analysis so that higher scores correspond to greater discrimination. The Everyday Discrimination Scale has been validated across a variety of populations (Barnes et al., 2004; Gonzales et al., 2016; Williams et al., 1997). Alpha for this measure in the current sample was 0.80.

2.2.4. Psychological and behavioral variables

Depressive symptoms in the week leading up to the baseline visit were assessed with eight items from the Center for Epidemiologic Studies Depression scale (CES-D; Radloff, 1977), which were modified into a yes/no format. Higher scores correspond to more depressive symptoms. External locus of control at baseline was assessed with five items from the Perceived Control scale (Lachman & Weaver, 1998), administered as part of a leave-behind questionnaire. Items included, "I often feel helpless in dealing with the problems of life," "Other people determine most of what I can and cannot do," "What happens in my life is often beyond my control," "I have little control over the things that happen to me," and "There is really no way I can solve problems I have." Items are rated for agreement on a 6-point Likert-type scale (1 = Strongly disagree to 6 = Strongly agree). Scores represent the average score across items, and higher scores correspond to more external locus of control. Alpha for this measure in the current sample was 0.86. Current smoking at baseline was operationalized as self-reported smoking status, with current non-smokers as the reference group.

2.2.5. Covariates

Analyses controlled for baseline age (centered at the sample median, 68 years), gender (male as reference category), and year of baseline assessment (2006 as reference category).

2.3. Analytic strategy

Descriptive statistics were computed in SPSS. Associations between race/ethnicity and CRP over four years were examined using structural equation models in Mplus. All models were weighted using HRS biomarker sample weights, which correct for over- or under-sampling and allow results to be generalizable to the larger population from which the sample was drawn (i.e., U.S. adults over age 50). Models were estimated using maximum likelihood estimation with robust standard errors (MLR). Missing data were managed with full information maximum likelihood (FIML). While FIML effectively handles non-random missingness related to variables included in the model, it does not account for missingness due to unobserved variables.

Baseline and follow-up CRP were estimated using latent difference score (LDS) models (McArdle & Nesselroade, 1994). Rather than calculating raw difference scores, the LDS model defined a latent variable corresponding to the residual of follow-up CRP, which was not predicted by baseline CRP. In the LDS model, features of CRP change that are of interest (e.g., mean CRP change, inter-individual variability in CRP change, relationship between baseline CRP and CRP change) are modeled as explicit parameters (McArdle, 2009).

An initial model examined associations between Black race and Hispanic ethnicity and CRP over four years within a single model, controlling for age, gender, and baseline year. Next, the social (i.e., education, discrimination), psychological (i.e., depressive symptoms, external locus of control) and behavioral (i.e., smoking) variables were added to this model to examine psychosocial pathways linking race and ethnicity to CRP over four years. As shown in Fig. 1, baseline CRP and

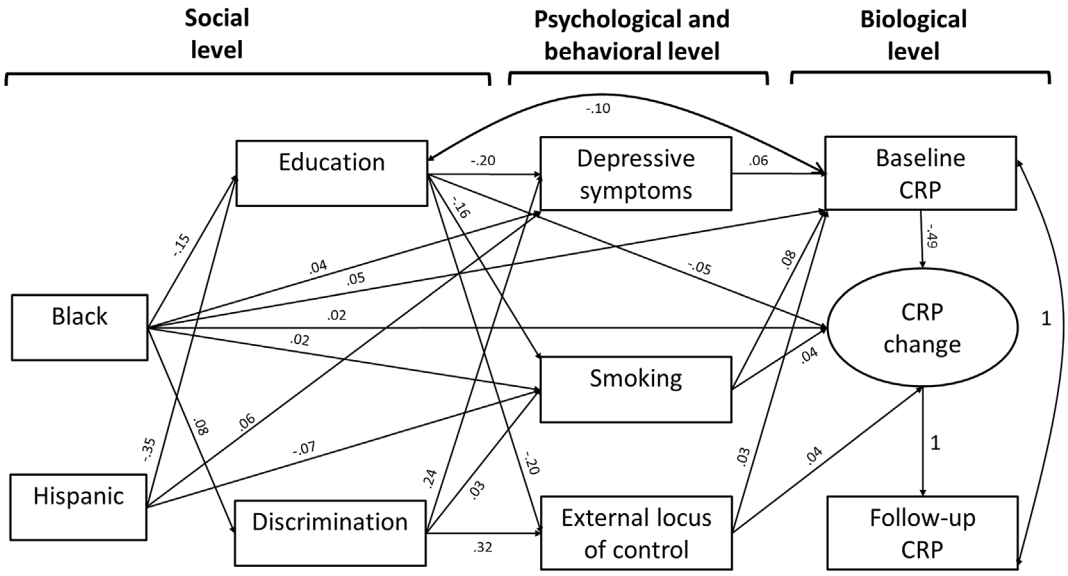

Fig. 1. Schematic of significant paths in the mediation model. Values shown are standardized estimates. For simplicity, covariates (i.e., age, gender, baseline assessment wave) and a correlation between contemporaneously-measured depressive symptoms and external locus of control are not shown. *Note.* CRP = C-reactive protein.

CRP change were regressed onto Black race and Hispanic ethnicity, all psychological, behavioral, and social variables, and all covariates in a single mediation model. In addition, each of the psychological and behavioral variables was regressed onto social variables, race and ethnicity, and covariates. Social variables were regressed onto race, ethnicity and covariates. Direct effects are associations between race and ethnicity and CRP over four years that are independent of all mediators and covariates. Indirect effects are specific effects of race and ethnicity on CRP over four years that operate through the mediator(s) and are calculated as the product of all regression paths within a given pathway. Total effects are the sum of all direct and indirect effects. The fit of all models was evaluated with the following commonly-used indices: comparative fit index (CFI), root-mean-square error of approximation (RMSEA), and standardized root-mean square residual (SRMR). CFI > 0.95, RMSEA < 0.06, and SRMR < 0.05 were used as criteria for adequate model fit (Hu & Bentler, 1999).

3. Results

3.1. CRP and race and ethnicity

Fit of the initial model examining the effects of race and ethnicity on baseline and follow-up CRP independent of covariates was perfect: CFI = 1.000; RMSEA = 0.000 (90% CI: 0.000 to 0.000), SRMR = 0.000. Independent of covariates, CRP increased over the four-year follow-up period (standardized estimate = 0.505; 95% CI: 0.467–0.543; $p < 0.001$). As shown in Table 2, individuals with higher baseline CRP evidenced smaller increases. Both Black and Hispanic individuals had significantly higher baseline CRP than non-Hispanic White individuals did. Black, but not Hispanic, individuals also showed a greater increase in CRP over the four-year study period. Higher baseline CRP was also associated with the later evaluation wave and female gender. Greater increase in CRP was also associated with female gender.

3.2. Psychosocial pathways from race and ethnicity to CRP

The mediation model fit well: CFI = 0.989; RMSEA = 0.047 (90% CI: 0.038–0.055), SRMR = 0.013. Associations between race and ethnicity and CRP were partially mediated by the social, psychological, and behavioral factors, as evidenced by attenuated direct effects and multiple significant indirect effects detailed in Table 3. Standardized estimates of significant paths are summarized in Fig. 1. Together, these mediators explained 34.67% of the association between Black race and baseline CRP and 32.35% of the association between Black race and CRP change. The association between Hispanic ethnicity and baseline CRP was fully mediated by the psychosocial variables, as the positive association between Hispanic ethnicity and baseline CRP (see Table 2) became negative and non-significant in the mediation model (see Table 3).

Table 2
Demographic predictors of baseline CRP and four-year change in CRP.

	Standardized estimate	95% CI	*p*
Baseline CRP			
Wave	0.022	0.004–0.040	0.046
Age	−0.018	−0.036–0.000	0.092
Female	0.097	0.079–0.116	< 0.001
Black	0.075	0.059–0.091	< 0.001
Hispanic	0.033	0.016–0.050	0.001
CRP change			
Baseline CRP	−0.479	−0.491 to −0.467	< 0.001
Wave	0.011	−0.009–0.030	0.360
Age	−0.004	−0.025–0.017	0.778
Female	0.030	0.010–0.049	0.013
Black	0.034	0.017–0.052	0.001
Hispanic	0.003	−0.016–0.022	0.773

Note. CRP = C-reactive protein; CI = Confidence Interval.

3.2.1. Baseline CRP

There were nine significant indirect effects of Black race on baseline CRP. First, Black race was associated with lower educational attainment, and lower educational attainment was associated with higher baseline CRP. In addition to this single-mediator path, Black race was associated with higher baseline CRP through all three of the psychological and behavioral correlates of lower educational attainment: more depressive symptoms, greater external locus of control, and smoking. Specifically, lower educational attainment was associated with more depressive symptoms, greater external locus of control, and smoking. In turn, each of these psychological and behavioral variables independently predicted higher baseline CRP. Next, Black race was associated with higher baseline CRP through all three of the psychological

Table 3
Standardized direct and indirect effects of race and ethnicity on CRP, estimated within a single model.

	Black race			Hispanic ethnicity		
	Estimate	95% CI	*p*	Estimate	95% CI	*p*
Baseline CRP						
Direct effect	0.049	0.033–0.065	< 0.001	−0.012	−0.030–0.006	0.285
Specific indirect effects through:						
Education	0.015	0.012–0.018	< 0.001	0.036	0.029–0.043	< 0.001
Education, depressive symptoms	0.002	0.001–0.002	< 0.001	0.004	0.002–0.006	< 0.001
Education, external locus of control	0.001	0.000–0.002	0.014	0.000	0.000–0.000	0.014
Education, smoking	0.002	0.001–0.003	< 0.001	0.005	0.003–0.006	< 0.001
Discrimination	0.001	0.000–0.003	0.211	0.000	−0.001–0.000	0.539
Discrimination, depressive symptoms	0.001	0.001–0.002	< 0.001	0.000	0.000–0.000	0.486
Discrimination, external locus of control	0.001	0.000–0.001	0.021	0.000	0.000–0.000	0.494
Discrimination, smoking	0.000	0.000–0.000	0.046	0.000	0.000–0.000	0.501
Depressive symptoms	0.002	0.001–0.004	0.003	0.003	0.002–0.005	0.001
External locus of control	0.000	−0.001–0.000	0.335	0.001	0.000–0.001	0.155
Smoking	0.002	0.000–0.004	0.045	−0.006	−0.008 to −0.004	< 0.001
CRP change						
Direct effect	0.023	0.005–0.040	0.038	−0.018	−0.038–0.002	0.148
Specific indirect effects through:						
Education	0.007	0.004–0.011	< 0.001	0.017	0.009–0.025	< 0.001
Education, depressive symptoms	0.000	0.000–0.001	0.123	0.002	0.000–0.003	0.121
Education, external locus of control	0.001	0.000–0.002	0.018	0.003	0.001–0.004	0.017
Education, smoking	0.001	0.000–0.002	0.003	0.002	0.001–0.004	0.003
Discrimination	0.000	−0.002–0.002	0.964	0.000	0.000–0.000	0.964
Discrimination, depressive symptoms	0.000	0.000–0.001	0.135	0.000	0.000–0.000	0.525
Discrimination, external locus of control	0.001	0.000–0.002	0.024	0.000	0.000–0.000	0.496
Discrimination, smoking	0.000	0.000–0.000	0.081	0.000	0.000–0.000	0.509
Depressive symptoms	0.001	0.000–0.002	0.141	0.001	0.000–0.003	0.143
External locus of control	0.000	−0.001–0.000	0.330	0.001	0.000–0.002	0.162
Smoking	0.001	0.000–0.002	0.081	−0.003	−0.005 to −0.001	0.006

Note. CRP = C-Reactive Protein; CI = Confidence Interval.

and behavioral correlates of discrimination: more depressive symptoms, greater external locus of control, and smoking. Specifically, greater discrimination was associated with more depressive symptoms, greater external locus of control, and smoking. As noted above, more depressive symptoms, greater external locus of control, and smoking each independently predicted higher baseline CRP. There was no association between discrimination and baseline CRP independent of these psychological and behavioral variables.

Independent of the social variables (i.e., education and discrimination), Black race was also associated with higher baseline CRP through depressive symptoms and smoking. Specifically, Black individuals reported more depressive symptoms and were more likely to smoke, independent of education and discrimination. In turn, both depressive symptoms and smoking were associated with higher baseline CRP. Black individuals did not report more external locus of control independent of education and discrimination.

There were six significant indirect effects of Hispanic ethnicity on baseline CRP. First, Hispanic ethnicity was associated with lower educational attainment, and lower educational attainment was associated with higher baseline CRP. In addition to this single-mediator path, Hispanic ethnicity was associated with higher baseline CRP through all three of the psychological and behavioral correlates of lower educational attainment: more depressive symptoms, greater external locus of control, and smoking. Specifically, lower educational attainment was associated with more depressive symptoms, greater external locus of control, and smoking. In turn, each of these psychological and behavioral variables independently predicted higher baseline CRP. Independent of education, Hispanic ethnicity was also associated with more depressive symptoms. In turn, more depressive symptoms predicted higher baseline CRP. Finally, there was a single *negative* indirect effect of Hispanic ethnicity on baseline CRP. Independent of the social and psychological variables, Hispanic individuals were less likely to smoke, and smoking was associated with higher baseline CRP.

3.2.2. CRP change

There were four significant indirect effects of Black race on CRP change. First, Black race was associated with lower educational attainment, and lower educational attainment was associated with greater increase in CRP. In addition to this single-mediator path, Black race was associated with lower educational attainment, which predicted greater external locus of control and smoking, and both greater external locus of control and smoking predicted greater increase in CRP. Independent of education, Black race was also associated with greater discrimination. In turn, greater discrimination predicted greater external locus of control, and greater external locus of control predicted greater increase in CRP.

While there were no total or direct effects of Hispanic ethnicity on CRP change, there were four significant indirect effects. Specifically, Hispanic ethnicity was associated with lower education, and lower education was associated with greater increase in CRP. Lower education was also associated with greater external locus of control and smoking, each of which predicted greater increase in CRP over the four-year study period. Finally, there was a single *negative* indirect effect of Hispanic ethnicity on CRP change. Independent of the social and psychological variables, Hispanic individuals were less like to smoke, and smoking was associated with greater increase in CRP.

3.2.3. Sensitivity analysis

While the choice to model the psychological variables (i.e., depressive symptoms and external locus of control) as outcomes of the social variables (i.e., education and discrimination) was based on both theoretical (Clark et al., 1999) and empirical (Brown et al., 2000; Pavalko et al., 2003) work, these variables were measured concurrently in the primary model in order to allow for an examination of longitudinal change in the CRP outcome. The fit of models in which the ordering of (1) discrimination and depressive symptoms; or (2) discrimination and external locus of control was reversed were worse than the fit of the original model, in which depressive symptoms and

external locus of control were both regressed onto discrimination (see Supplementary Table 1). However, adding in a modification that allowed for a correlation between education and depressive symptoms in the alternative model in which depressive symptoms predicted concurrently-measured discrimination resulted in the best overall fit.

Because cross-sectional mediation paths involving discrimination and the psychological factors (depressive symptoms and external locus of control) may be upwardly biased due to their concurrent measurement, a sensitivity analysis was conducted to compare cross-sectional coefficients between discrimination, depressive symptoms, and external locus of control measured at baseline (2006/2008) to longitudinal coefficients between baseline discrimination (2006/2008), follow-up depressive symptoms (2010/2012) and follow-up external locus of control (2010/2012). These longitudinal coefficients were slightly smaller than the corresponding cross-sectional coefficients shown in Fig. 1 (0.22 versus 0.24 for discrimination → depressive symptoms; 0.27 versus 0.32 for discrimination → external locus of control), indicating some upward bias. Finally, an additional sensitivity analysis confirmed that discrimination reported in 2006/2008 prospectively predicted both depressive symptoms (standardized estimate = 0.094; 95% CI: 0.072–0.116; $p < 0.001$) and external locus of control (standardized estimate = 0.118; 95% CI: 0.093–0.143; $p < 0.001$) four years later, controlling for previous values of depressive symptoms and external locus of control, respectively.

4. Discussion

This national, population-based, longitudinal study provides evidence for deleterious effects of racially and ethnically patterned psychosocial disadvantage on systemic inflammation. In line with the biopsychosocial framework, educational disadvantage was associated with worse CRP for both Black and Hispanic adults directly, as well as indirectly through the links between educational disadvantage and negative psychological and behavioral sequelae: higher depressive symptoms, greater external locus of control, and smoking. Independent of education, discrimination was also associated with worse CRP via depressive symptoms, external locus of control, and smoking for Black adults. Together, these results support the view that social disadvantage is associated with inflammatory processes for racial and ethnic minorities, though the current data cannot prove causation. Results further suggest that a portion of these associations is mediated by depressive symptoms, external locus of control, and smoking.

4.1. Social factors

Findings from this study support existing evidence that educational disadvantage contributes to racial and ethnic disparities in health (Phelan, Link, & Tehranifar, 2010). Our findings further indicate that at least one pathway by which racially patterned educational disadvantage might adversely affect health involves the negative psychological and behavioral correlates of lower educational attainment. Substantial literature demonstrates associations between lower educational attainment and adult depression (Akhtar-Danesh & Landeen, 2007; Eaton, Muntaner, Bovasso, & Smith, 2001; Kaplan, Roberts, Camacho, & Coyne, 1987; Ladin, 2008; Sargeant, Bruce, Florio, & Weissman, 1990), lower educational attainment and lower perceived control (Mirowsky & Ross, 2007), and lower educational attainment and smoking (Hu, Davies, & Kandel, 2006).

Education may promote psychological resilience by enhancing a vast array of personal and social resources, such as literacy (Nguyen et al., 2017), higher-status occupations (Link, Lennon, & Dohrenwend, 1993), higher income (Quesnel-Vallée & Taylor, 2012), embeddedness in cohesive social structures (Kawachi, Kennedy, Lochner, & Prothrow-Stith, 1997), and cultural entitlement (ten Kate, de Koster, & van der Waal, 2017). Through its socioeconomic benefits, education may reduce exposure to adversity throughout life and provide resources to handle crises (Turner & Noh, 1983; Wheaton, 1980). As a developmental environment, schooling also provides graded opportunities to succeed in increasingly challenging tasks, nurturing a sense of control (Mirowsky & Ross, 2007). It has been proposed that the negative association between education and smoking may reflect the use of smoking as a coping strategy in the face of environmental stressors (Mezuk, Abdou et al., 2013; Pomerleau & Pomerleau, 1984), and individuals with lower educational attainment are also less likely to benefit from anti-smoking campaigns (Escobedo & Peddicord, 1996). The fact that associations between education and CRP were not entirely mediated by the psychological and behavioral variables examined in this study points to additional pathways. For example, higher educational attainment is a prominent predictor of health behaviors beyond smoking, such as healthy eating and exercise (Hampson, Goldberg, Vogt, & Dubanoski, 2007). Additional research is needed to more fully characterize the protective effects of educational attainment.

In contrast to education, discrimination did not predict CRP above and beyond its psychological and behavioral consequences. The stress of discrimination accumulates over time (Essed, 1990a, 1990b; Williams et al., 1997), and discrimination has previously been linked to elevated depressive symptoms and external locus of control (Zahodne, Sol, & Kraal, 2019; Banks, Kohn-Wood, & Spencer, 2006; Earnshaw et al., 2016), as well as behaviors that may serve as self-regulatory coping strategies to preserve mental health in the face of social stressors, such as smoking (Mezuk, Abdou et al., 2013). The current study is consistent with these previous results indicating that discrimination is associated with negative psychological sequelae and negative health behaviors. Further, these findings warrant future longitudinal investigations of systemic inflammation as a potential downstream negative health effect of discrimination. A recent cross-sectional study in HRS documented a positive association between the experience of *major* discrimination and CRP (Cobb, Parker & Thorpe, 2018). The current study extends this previous work by focusing on *everyday* discrimination, incorporating follow-up CRP data, and examining psychological and behavioral mediators of the association between discrimination and CRP.

4.2. Psychological and behavioral factors

The results of this study are also consistent with previous work indicating that psychological factors predict subsequent inflammatory responses (Elliot et al., 2017; Stewart, Rand, Muldoon, & Kamarck, 2009). Specifically, depressive symptoms are consistently associated with elevated levels of CRP (Danner, Kasl, Abramson, & Vaccarino, 2003; Elovainio et al., 2006; Gimeno et al., 2009). The current study extends a previous longitudinal analysis of depressive symptoms and CRP in the HRS (Niles, Smirnova, Lin & Donovan, 2018) by examining its social antecedents and racial differences, as well as by including other related psychological and behavioral factors. Depressive symptoms increase sympathetic nervous activity, decrease parasympathetic nervous activity, and increase sedentary behavior and body mass index, all of which have been shown to promote inflammation (Di Gregorio et al., 2005; Kop & Gottdiener, 2005). Depressive symptoms may also directly promote inflammatory responses by activating the immune response (Maes, 1995; Maier & Watkins, 1998). While some longitudinal studies indicate that depressive symptoms predict subsequent inflammatory response, but not vice versa (Stewart et al., 2009), other studies indicate that depressive symptoms and inflammation exhibit a bi-directional relationship (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008; Matthews et al., 2010). Studies with greater longitudinal follow-up are needed to more directly characterize the nature of the relationship between depressive symptoms and CRP.

While fewer studies have examined associations between external locus of control and inflammatory response, a recent analysis in the HRS also documented a positive association between external locus of control and subsequent CRP (Elliot et al., 2017). The current study

extends this previous work by considering racial and ethnic differences in perceived control. Having a sense of control may lead to less inflammation by reducing the impact of stressors, in line with experimental studies of other positive psychological factors such as optimism and self-esteem (Brydon, Walker, Wawrzyniak, Chart, & Steptoe, 2009; O'Donnell, Brydon, Wright, & Steptoe, 2008). In the current study, external locus of control was associated with a greater four-year increase in CRP, independent of baseline CRP, which supports a prospective association between perceived control and subsequent inflammation. However, additional experimental work is needed to confirm the direction of effects.

Finally, substantial evidence supports an association between smoking and inflammatory markers, including CRP (Tibuakuu et al., 2017; Tracy et al., 1997). Smoking modulates inflammation by activating a biological pathway that results in increased transcription of genes involved in the immune response (Goncalves et al., 2011; Rom, Avezov, Aizenbud, & Reznick, 2013). Interestingly, levels of CRP among former smokers are similar to those of individuals who have never smoked, supporting the beneficial health effects of smoking cessation (Tibuakuu et al., 2017).

4.3. Racial and ethnic differences

This study found differences in longitudinal CRP between Black and Hispanic participants. Compared to non-Hispanic Whites, Black participants showed higher levels of CRP at baseline and greater increases in CRP over the four-year study period. In contrast, there was only a significant Hispanic-White difference in baseline CRP. Indeed, Hispanic-White differences in inflammatory response are not always reported in studies of older adults (Albert, 2007), which has been interpreted in the context of intragroup variability among Hispanics and/or the "Hispanic paradox" of comparable or favorable health outcomes despite lower socioeconomic status (Crimmins et al., 2007; Mitchell & Aneshensel, 2016). Most strongly supported for Mexican Americans, the "Hispanic paradox" may reflect selective migration (Markides & Eschbach, 2005).

Another potential contributor to the "Hispanic paradox" involves smoking behavior (Perez-Stable et al., 2001). In the U.S., Hispanic adults are less likely to smoke than non-Hispanic Whites, though there is great variability in smoking behaviors across Hispanic subgroups (Kaplan et al., 2014), and Hispanic smokers are less likely to be advised to quit than non-Hispanic Whites (Lopez-Quintero, Crum, & Neumark, 2006). Independent of education and discrimination, Hispanic participants in this study were less likely to smoke than non-Hispanic Whites, while Black participants were more likely to smoke than non-Hispanic Whites. This difference resulted in a positive indirect effect of Black race on CRP through smoking and a negative indirect effect of Hispanic ethnicity on CRP through smoking. Furthermore, greater exposure to social exposures (i.e., low educational attainment and/or discrimination) among both Blacks and Hispanics was associated with higher baseline CRP and greater increase in CRP over time through smoking, suggesting additional points of intervention among disadvantaged subgroups.

Finally, while educational disadvantage mediated associations between both Black race and Hispanic ethnicity and higher CRP, discrimination only mediated associations involving Black race. In the U.S., Black individuals report higher levels of discrimination than non-Hispanic Whites across age, gender, and socioeconomic status (Forman, Williams, Jackson, & Gardner, 1997). In contrast, many studies report no difference in reports of discrimination across Hispanics and non-Hispanic Whites (T. T. Lewis, Yang, Jacobs, & Fitchett, 2012). This finding may reflect the items included in the Everyday Discrimination Scale, which were developed based on qualitative interviews with Black women (Essed, 1990a, 1990b; Williams et al., 1997). The lack of a significant Hispanic-White difference in discrimination may also reflect intragroup variability among Hispanic adults, as the perception of discrimination varies according to ethnic identity, immigrant status, and age of immigration among Hispanics (Pérez, Fortuna, & Alegria, 2008).

4.4. Limitations and strengths

Limitations of this study include the availability of only two time points of biomarker data, which precluded our ability to use growth curve modeling to more reliably evaluate associations involving cross-sectional level of inflammation versus changes in inflammation. However, the use of longitudinal biomarker data in this study strengthens our interpretation that psychosocial factors are prospectively associated with CVD risk, though these data cannot prove causation. Another limitation is the use of concurrent self-report measures of discrimination, depressive symptoms, and locus of control to predict concurrent CRP and four-year change in CRP. Indeed, a sensitivity analysis replacing concurrently-measured depressive symptoms and external locus of control with their values four years later indicated that the original mediation paths may be slightly upwardly biased. However, additional analyses confirmed that discrimination prospectively predicted both psychological factors four years later, controlling for previous values. Nonetheless, future studies should consider the contribution of response bias to associations between self-report variables and include additional longitudinal measures collected over varying time scales to clarify the direction of effects.

Another limitation was that smoking was the only health behavior examined in this study. We recommend that future research incorporate additional health behaviors to more fully model the complexity of racial and ethnic disparities (Mezuk, Abdou et al., 2013). In addition, only self-reported health conditions were available. Future work is needed to confirm effects in the context of additional, directly-measured information on specific health conditions. Because this study focused on only three racial and ethnic groups with adequate representation in the HRS, future studies are also needed to investigate long-term biological impacts of social and psychological stress among other groups. Finally, other limitations of the current study include possible selection bias above and beyond that addressed via sampling weights, assuming linear associations among the continuous variables (e.g., education), information bias from self-report, confounding due to uncontrolled for variables (e.g., region of residence, national origin), and chance findings from multiple comparisons.

Strengths of this study include its large sample of older adults from three racial and ethnic groups. The use of sampling weights allow the current results to be generalizable to the larger population of U.S. adults over age 50. The primary contribution of this study is its theoretically guided integration of biological, psychological, behavioral, and social variables into an empirical model. Links revealed by this model, as well as differences in these pathways between two minority groups (i.e., non-Hispanic Black and Hispanic), provide additional information regarding potential biopsychosocial pathways from race and ethnicity to CVD risk that can guide future work. Because this model reflects only a subset of a more comprehensive biopsychosocial model of racial and ethnic disparities in health, additional work is needed to integrate other structural (e.g., neighborhood disadvantage), behavioral (e.g., diet), and biological (e.g., cortisol) factors.

While the psychosocial factors examined in this study fully mediated the association between Hispanic ethnicity and baseline CRP, the fact that they only explained a portion of the association between Black race and CRP further underscores the need for more comprehensive models. The size of independent associations between each psychological factor (i.e., depressive symptoms and external locus of control) and CRP were between one third and three quarters of the size of the association between current smoking and baseline CRP, and the size of association between external locus of control and CRP change was the same as that between smoking and CRP change. Thus, the psychological factors examined in this study explained a substantial proportion of

racial and ethnic variation in CRP and may warrant as much attention as other modifiable factors.

5. Conclusion

In conclusion, this nationally representative, longitudinal study helps to clarify health inequalities by detailing pathways through which the psychosocial experiences of Black and Hispanic adults are associated with systemic inflammation. By modeling multiple biopsychosocial pathways from race and ethnicity to CRP within a single model, this study provides an important replication of previous findings from studies that focused on only a subset of these pathways. Such replication is important for confirming that targeting only one pathway is unlikely to fully eliminate racial disparities and for demonstrating that certain related constructs (e.g., psychological and behavioral factors such as depressive symptoms, external locus of control, and smoking) may represent distinct opportunities for behavioral health interventions. In addition, this study makes a novel contribution to the literature on health disparities by demonstrating specific ways in which these biopsychosocial pathways to health disparities may differ for different minority groups (i.e., Hispanic versus non-Hispanic Black), which could guide more culturally-relevant prevention and intervention efforts. Because CRP is an important indicator of CVD risk, pathways involving education, discrimination, depressive symptoms, external locus of control, and smoking indicate that interventions targeted at these levels may have potential cardiovascular health benefits.

Ethics approval

This study used publicly-available data from the Health and Retirement Study, which was approved by the University of Michigan Institutional Review Board.

Acknowledgments

This research was supported in part by grants from the National Institutes on Aging [grant numbers R01AG054520, R00AG047963]. The HRS (Health and Retirement Study) is sponsored by the National Institute on Aging (grant number NIA U01AG009740) and is conducted by the University of Michigan. The authors report no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ssmph.2019.100391.

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