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Socioeconomic Status, Preeclampsia Risk and Gestational Length in Black and White Women

Kharah M. Ross[1] · Christine Dunkel Schetter[2] · Monica R. McLemore[3] · Brittany D. Chambers[4,5] · Randi A. Paynter[4] · Rebecca Baer[4,6] · Sky K. Feuer[4,7] · Elena Flowers[8] · Deborah Karasek[4,7] · Matthew Pantell[9] · Aric A. Prather[10] · Kelli Ryckman[11] · Laura Jelliffe-Pawlowski[4,5]

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Abstract

Background Higher socioeconomic status (SES) has less impact on cardio-metabolic disease and preterm birth risk among Black women compared to White women, an effect called "diminishing returns." No studies have tested whether this also occurs for pregnancy cardio-metabolic disease, specifically preeclampsia, or whether preeclampsia risk could account for race-by-SES disparities in birth timing.

Methods A sample of 718,604 Black and White women was drawn from a population-based California cohort of singleton births. Education, public health insurance status, gestational length, and preeclampsia diagnosis were extracted from a State-maintained birth cohort database. Age, prenatal care, diabetes diagnosis, smoking during pregnancy, and pre-pregnancy body mass index were covariates.

Results In logistic regression models predicting preeclampsia risk, the race-by-SES interaction (for both education and insurance status) was significant. White women were at lower risk for preeclampsia, and higher SES further reduced risk. Black women were at higher risk for preeclampsia, and SES did not attenuate risk. In pathway analyses predicting gestational length, an indirect effect of the race-by-SES interaction was observed. Among White women, higher SES predicted lower preeclampsia risk, which in turn predicted longer gestation. The same was not observed for Black women.

Conclusions Compared to White women, Black women had increased preeclampsia risk. Higher SES attenuated risk for preeclampsia among White women, but not for Black women. Similarly, higher SES indirectly predicted longer gestational length via reduced preeclampsia risk among White women, but not for Black women. These findings are consistent with diminishing returns of higher SES for Black women with respect to preeclampsia.

Keywords Health disparities · Socioeconomic status · Race/ethnicity · Preeclampsia · Gestational length

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✉ Kharah M. Ross
kharah.ross@ucalgary.ca

[1] Owerko Centre, Alberta Children's Hospital Research Institute, University of Calgary, #355, 3820 24th Ave, Calgary, AB T3B 2X9, Canada

[2] Department of Psychology, University of California Los Angeles, Los Angeles, CA, USA

[3] Department of Family Health Care Nursing, University of California San Francisco, San Francisco, CA, USA

[4] California Preterm Birth Initiative, University of California San Francisco, San Francisco, CA, USA

[5] Department of Epidemiology and Biostatistics, University of California San Francisco, San Francisco, CA, USA

[6] Department of Pediatrics, University of California San Diego, San Diego, CA, USA

[7] Department of Obstetrics, Gynecology and Reproductive Sciences, University of California San Francisco School of Medicine, San Francisco, CA, USA

[8] Department of Physiological Nursing, University of California San Francisco, San Francisco, CA, USA

[9] Department of Pediatrics, University of California San Francisco, San Francisco, CA, USA

[10] Department of Psychiatry, University of California San Francisco, San Francisco, CA, USA

[11] Department of Epidemiology, University of Iowa, Iowa City, IA, USA

Introduction

Preeclampsia, defined as gestational hypertension accompanied by proteinuria, is a costly, burdensome disease that affects 2% to 8% of all pregnancies, and accounts for 10% to 15% of maternal deaths globally [1]. Preeclampsia diagnosis also increases the risk for adverse pregnancy outcomes, particularly preterm birth or shorter gestational length [2]. In general, Black women [3–5] and women with lower socioeconomic status (SES) [6, 7] are at increased risk for preeclampsia. Although most studies have examined race/ethnicity and SES as separate predictors of preeclampsia risk, race/ethnicity and SES are conflated in the USA, with Black women being more likely to be of a lower SES compared to White women [8]. When the interrelationship of race/ethnicity and SES is considered, it is usually accepted that poor Black women are at highest risk for poor health outcomes compared to higher SES Black women [9, 10].

A growing body of research, however, shows that higher SES does not necessarily confer the same health gains for Black individuals as for White individuals. Compared to White individuals, among Black individuals, higher SES can confer either smaller health benefits [11], no health benefits [12], or even increased risk for poor health outcomes [13]. This phenomenon has been referred to as "diminishing returns" or "widening health disparities" [14–16], and has been observed across a range of outcomes, including mortality risk [17–20], self-rated health [21–23], mental health [24–27], insomnia [12], and molecular aging as indexed by telomere length [28]. In particular, this effect has been robustly observed for cardio-metabolic indicators and disease risk, including body mass index (BMI) [12, 27], inflammation [interleukin-6 (IL6), C-reactive protein (CRP)] [11, 29], epigenetic modification of inflammation-related pathways [30], allostatic load [31], glycemic control [32], postpartum cardio-metabolic health [13], and risk for type II diabetes mellitus [33, 34], hypertension [35, 36], myocardial infarction, and stroke [37]. "Diminishing returns" have also been observed in the context of pregnancy [38, 39]. Relative to trends observed in White women, higher SES among Black women does not confer the same reduction in risk for preterm birth [40–50], low birth weight [43, 50–56], or infant mortality risk [57 for exceptions, see 58–66]. As such, considering only the independent effects of race/ethnicity and SES could result in important, health-relevant patterns being overlooked, with implications for the health of Black women and their children.

Given that preeclampsia increases the risk for preterm birth [2, 67, 68], and that the "widening disparities" effect is particularly emergent for cardio-metabolic risk [11–13, 27, 29–37], the literature was reviewed for any evidence of this phenomenon for pregnancy cardio-metabolic diseases, particularly preeclampsia. We found only one study that tested an interactive effect of race/ethnicity and SES for preeclampsia risk [69]. In this study, a 2 × 2 design was used to compare differences in preeclampsia risk between White women and women of other races/ethnicities and high SES versus low SES groups ($N = 41{,}496$). Rates of preeclampsia were lowest for White women in the higher SES category (22 per 1,000), and highest for women of other races/ethnicities in the lower SES category (54.9 per 1,000) [69]. Given that women of different races and ethnicities were collapsed into a single group, it is not clear how to interpret these findings for Black women. As such, it is not known whether the differential effect of higher SES on Black versus White women applies to preeclampsia, with downstream implications for race/ethnicity and SES disparities in preterm birth.

The purpose of the present investigation is to examine whether race (Black vs. White) and SES (using education and insurance status as proxies) independently and interactively predict risk for preeclampsia, without pre-existing hypertension. This was tested in a population-based pregnancy cohort. It was hypothesized that (1) Black women would have increased risk for preeclampsia as compared to White women; (2) lower SES would be associated with increased risk for preeclampsia; and (3) race and SES would interact, such that higher SES would be associated with reduced risk for preeclampsia for White women, but not for Black women. Exploratory analyses also tested whether race, SES, and the race-by-SES interaction were associated with gestational age at birth, as mediated by preeclampsia risk.

Methods

Participants

The sample consisted of 718,604 Black and White women drawn from a population-based cohort of singleton California births (2007–2012) [70, 71]. The birth cohort file is maintained by the California Office of Statewide Health Planning and Development, and contains data from linked birth and death certificates, and maternal and infant characteristics, hospital discharge diagnoses, and procedures [International Classification of Diseases, 9th Revision, Clinical Modification; ICD9] [72] from 1 year before to 1 year after delivery. Women were included in the current analyses if they had information available for key variables (education, insurance status, race, and preeclampsia diagnosis). Women were excluded if they were less than 18 years of age or pregnant with fetuses known to have congenital or chromosomal anomalies. Methods and protocols for the study were approved by the Committee for the Protection of Human Subjects within the Health and Human Services Agency of the State of California.

Race, Education, Insurance Status, Preeclampsia and Birth Gestational Age

Maternal race was coded as White (0) or Black (1).[1] Two proxies for SES were considered: Education and insurance status. Education was dichotomized, such that women were classified as having lower education (high school or less; 0) or higher education (more than high school; 1). Health insurance was coded based on receipt of MediCal (California's health insurance for low-income persons). Women were coded as having Medi-Cal (0) or not (1). Thus, low SES was operationalized two ways, as having a high school education or less, or receipt of MediCal health insurance. Education and insurance status were moderately associated, $r = .424$. Two interaction terms were calculated by multiplying together the race and SES terms.

Women who developed preeclampsia during their pregnancy were identified by hospital discharge diagnosis (ICD-9 codes 642.4, 642.5, and/or 642.6). In order to isolate pregnancy-induced cardio-metabolic disease, women with pre-existing hypertension were excluded. Preeclampsia was coded as "preeclampsia diagnosis" (1) or "no preeclampsia diagnosis" (0). Gestational age at birth was obtained from best obstetric estimate in the birth certificate record.

Covariates

The following covariates were included in the study: maternal age at delivery (years), parity [nulliparous (1) vs. multiparous (0)], prenatal care (month that care commenced), any diabetes diagnosis (pre-existing or gestational) [yes (1) vs. no (0)], cigarette smoking during pregnancy [yes (1) vs. no (0)], and maternal pre-pregnancy body mass index (BMI). Pre-pregnancy BMI was calculated by converting height and weight to metric units and dividing weight by height-squared (kg/m^2), with exclusion of women with BMI values < 19 kg/m^2 and women with BMI values greater than 3 standard deviations above the mean (> 45 kg/m^2), given severe underweight and severe obesity status.

Analytic Approach

First, associations between preeclampsia risk and race, SES, and the race-by-SES interactions were tested using logistic regression models in SPSS v.24 [73]. Separate models were run for both SES indicators, i.e., education and insurance status. All covariates were included in all models. In models where education was a predictor, insurance status was a covariate, and vice versa. For all dichotomous variables, comparisons were made relative to groups coded as "0" (e.g., White race, high school education or less, MediCal insurance status, multiparous, no diabetes, no smoking during pregnancy). Significant race-by-SES interactions were probed using dummy coding, specifically by comparing high SES White women with low SES White women, low SES Black women, or high SES Black women with respect to preeclampsia risk.

Second, to test whether risk for preeclampsia could account for associations between race, SES, and the race-by-SES interactions with gestational age at birth, mediation models were tested using path analyses. Models were run using the *lavaan* package [74] and full-information maximum likelihood (FIML) in R v.3.5.1 [75]. The conceptual model in Fig. 1 was translated as follows: Race, SES (either education or insurance status), and the race-by-SES interaction term were each entered as exogenous/predictor variables, preeclampsia diagnosis was the mediator, and gestational age at birth was the outcome. Exogenous variables were allowed to co-vary with each other. Significant race-by-SES interactions were probed in path analyses by splitting the sample by race and running models testing the same mediation model, excluding race and the interaction term as predictors. Mediation models were just-identified, i.e., they included all possible paths, and so have zero degrees of freedom. As such, fit indices cannot be calculated or reported.

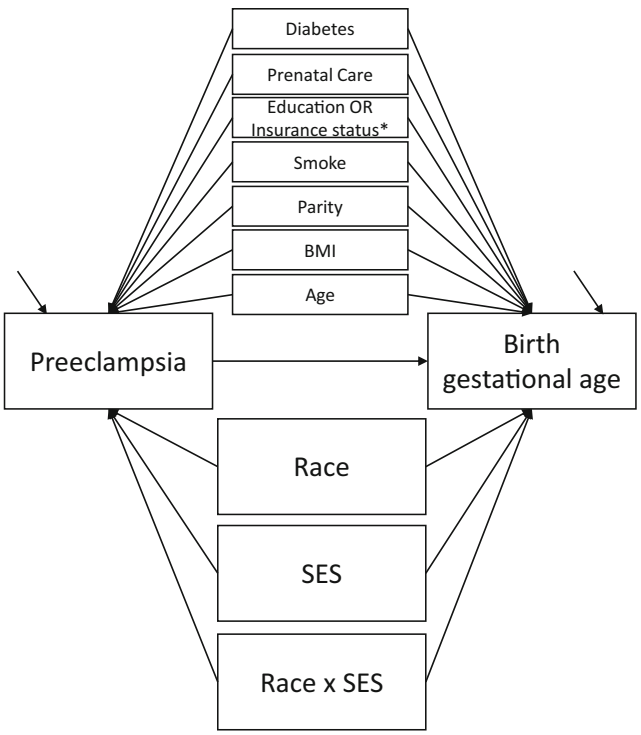

Fig. 1 Path analysis testing associations between race, SES, and race x SES with birth gestational age as mediated by preeclampsia risk. Predictor/exogenous variables were allowed to co-vary with each other (not shown). BMI = body mass index. *When SES was indexed by education, models covaried for insurance status, and vice versa

[1] Both White and Black categories include women born in and outside the USA.

Results

Sample Characteristics

The sample consisted of 111,801 Black women and 607,151 White women. Sample characteristics are presented in Table 1. Compared to White women, Black women had fewer years of formal education (48.8% high school education or less vs. 25.9% high school education or less), $\chi^2(1) = 31{,}555$, $p < .01$. Black women were also more likely to have public health insurance (59.8 vs. 23.8%), $\chi^2(1) = 54{,}461$, $p < .01$, were more likely to be diagnosed with preeclampsia (4.7 vs. 2.8%), $\chi^2(1) = 1{,}568$, $p < .01$, and tended to have shorter gestational duration (38.6 ± 2.1 weeks vs. 38.9 ± 1.7 weeks), $t(718{,}950) = 60.0$, $p < .01$. With respect to covariates, compared to White women, Black women were more likely to be younger at delivery (27.0 ± 6.0 years vs. 30.0 ± 5.7 years), $t(718{,}950) = 161$, $p < .01$, to have a higher pre-pregnancy BMI (27.2 ± 5.7 kg/m^2 vs. 25.3 ± 5.0 kg/m^2), $t(718{,}950) = -114$, $p < .01$, to begin prenatal care later (2.7 ± 1.5 months vs. 2.5 ± 1.2 months), $t(718{,}950) = -57.8$, $p < .01$, were more likely to smoke (8.5 vs. 6.6%), $\chi^2(1) = 542$, $p < .01$, and tended to be multiparous rather than nulliparous (43.9 vs. 38.6%). No differences emerged between Black and White women with respect to diabetes diagnoses (6.3 vs. 6.4%), $\chi^2(1) = .01$, $p = .95$.

Parallel analyses were run for both SES indicators (education and insurance status). The same pattern of results emerged across analyses for both education and insurance status.

Preeclampsia Risk

Race was independently associated with risk for preeclampsia in both logistic models, such that Black women were more likely to develop preeclampsia than White women, independent of education, OR = 1.56, .95CI(1.48, 1.64), or insurance status, OR = 1.55, .95CI(1.48, 1.63) (Table 2). Both SES indicators were additionally associated with risk for preeclampsia, such that women with high SES, either indexed as more education, OR = .872, .95CI(.838, .907), or non-MediCal insurance, OR = .899, .95CI(.862, .937), had lower risk for preeclampsia compared to women with low SES. Significant race-by-SES interactions were also observed, $p < .030$. Compared to White women with high SES, White women with low SES and Black women with both high and low SES had statistically significant increased odds of preeclampsia (Fig. 2). The magnitude of risk for preeclampsia, however, was highest for Black women of any SES. Moreover, Black women with high or low SES did not significantly differ with respect to preeclampsia risk.

Birth Gestational Age

Full results of path analyses testing indirect associations between race, SES (education or insurance status), and the race-by-SES interactions on gestational length, as mediated by preeclampsia risk, are reported in Supplemental Table 1. Coefficients from models where education was included as the SES predictor are labeled "education," and those where insurance status was included as the SES predictor are labeled "insurance."

Table 1 Sample characteristics. BMI = body mass index

	Mean ± SD or % (*N*)	
Variable	Black	White
N	100.0 (111,801)	100.0 (607,151)
Less than high school	13.7 (15,298)	4.9 (30,050)
High school	35.1 (39,251)	21.0 (127,796)
College	46.3 (51,725)	57.9 (351,751)
Post-secondary	4.9 (5,527)	16.1 (97,554)
Public health insurance (MediCal)	59.8 (61,187)	23.8 (137,410)
Preeclampsia	4.7 (5,216)	2.8 (16,803)
Gestational length (weeks)	38.6 ± 2.19	38.9 ± 1.65
Age (years)	27.0 ± 6.03	30.0 ± 5.72
Diabetes	6.3 (7,041)	6.4 (38,271)
Nulliparous	38.6 (43,415)	43.9 (266,373)
Prenatal care initiation (months)	2.69 ± 1.53	2.45 ± 1.21
Cigarette smoking	8.5 (9,495)	6.6 (39,945)
Pre-pregnancy BMI (kg/m^2)	27.2 ± 5.73	25.3 ± 5.04

Table 2 Logistic regression model summary, predicting preeclampsia risk from race, education and the race-by-education interaction, controlling for smoking, parity, age, pre-pregnancy BMI, prenatal care, medical insurance, and any diabetes diagnosis. From a population-based California cohort of singleton births (2007–2012). BMI = body mass index

Variable	*b*	SE	OR	.95CI	*p*
Smoking during pregnancy*	.046	.029	1.05	.99, 1.11	.111
Nulliparous*	1.13	.016	3.11	3.01, 3.20	< .001
Maternal age	.016	.001	1.02	1.01, 1.02	< .001
Pre-pregnancy BMI	.065	.001	1.07	1.07, 1.07	< .001
Prenatal care†	− .005	.006	1.00	.98, 1.01	.386
Insurance status*	.080	.018	1.08	1.04, 1.12	< .001
Diabetes*	.598	.023	1.82	1.74, 1.90	< .001
Race*	.442	.027	1.56	1.48, 1.64	< .001
Education*	− .137	.020	0.872	0.838, 0.907	< .001
Race x Education*	.077	.035	1.08	1.01, 1.16	.030
Smoking during pregnancy*	.045	.029	1.05	.988, 1.11	.121
Nulliparous*	1.13	.016	3.11	3.01, 3.20	< .001
Maternal age	.016	.001	1.02	1.01, 1.02	< .001
Pre-pregnancy BMI	.065	.001	1.07	1.07, 1.07	< .001
Prenatal care†	− .005	.006	.995	.983, 1.01	.349
Education*	− .115	.018	.892	.861, .923	< .001
Diabetes*	.598	.023	1.82	1.74, 1.90	< .001
Race*	.439	.026	1.55	1.48, 1.63	< .001
Insurance status*	− .107	.021	.899	.862, .937	< .001
Race x Insurance*	.092	.036	1.10	1.02, 1.18	.010

*all comparisons are made relative to the group coded as "0" (White race, high school or less, using MediCal, no preeclampsia diagnosis, multiparous, no diabetes, and no cigarette smoking during pregnancy), † Defined as month that prenatal care was started

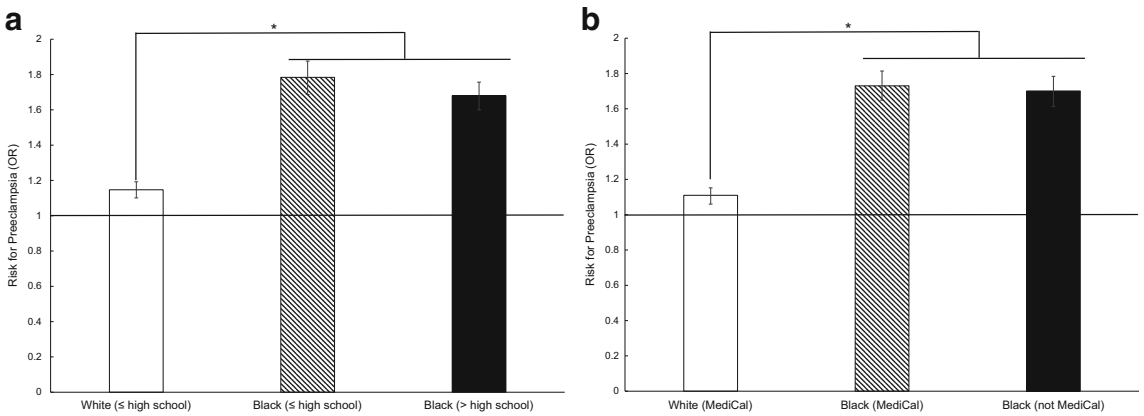

Fig. 2 Risk for preeclampsia (OR, 95% CI) for low SES white women, low SES Black women, and high SES Black women, compared with high SES White women, from a population-based California cohort of singleton births (2007–2012). **a** Odds ratios for SES as indexed by education. Compared to White women with more education, White women with less education and Black women with both more and less education had increased odds of preeclampsia, *p*'s < .001. The magnitude of increased risk for preeclampsia, however, was higher for Black women in both less, OR = 1.78, .95CI(CI, 1.69, 1.88), and more educated group, OR = 1.68, .95CI(1.60, 1.76), relative to White women with less education, OR = 1.15, .95CI(1.10, 1.19). **b** Odds ratios for SES as indexed by insurance status. Compared to White women not using MediCal, White women using MediCal and Black women both using and not using MediCal had increased odds of preeclampsia, *p*'s < .001. The magnitude of increased risk for preeclampsia was higher for Black women both using MediCal, OR = 1.73, .95CI(1.65, 1.81), and not using Medical, OR = 1.70, .95CI(1.62, 1.79), relative to White women using MediCal, OR = 1.11, .95CI(1.07, 1.16)

Direct and indirect effects were detected for race. Independent of preeclampsia diagnosis or either SES indicator, Black women had shorter gestational length compared to White women, $b_{education} = -.289$, SE = .009, $p < .001$; $b_{insurance} = -.289$, SE = .009, $p < .001$, and Black women were also more likely to be diagnosed with preeclampsia, which in turn was associated with lower gestational age at birth, $b_{education} = -.023$, SE = .001, $p < .001$; $b_{insurance} = -.023$, SE = .001, $p < .001$. We also found indirect and direct effects for both SES indicators. Independent of preeclampsia status and race, low SES, i.e., less educated, $b_{education} = .072$, SE = .006, $p < .001$, or being on MediCal insurance, $b_{insurance} = .067$, SE = .006, $p < .001$, was associated with shorter gestational length. Additionally, low SES women were more likely to be diagnosed with preeclampsia, which in turn was associated with shorter gestational length, $b_{education} = .005$, SE = .001, $p < .001$; $b_{insurance} = .003$, SE = .001, $p < .001$.

For both SES indicators, indirect effects of the race-by-SES interaction terms were detected, $b_{education} = -.004$, SE = .002, $p = .031$; $b_{insurance} = -.004$, SE = .002, $p = .028$, suggesting that the effect of SES on gestational length via preeclampsia diagnosis varied depending on race. To probe the interaction, the sample was split by race and path models were run independently in each subsample for both SES indicators. Among White women, being high SES continued to predict longer gestations through reduced risk for preeclampsia (Fig. 3). However, among Black women, the indirect effect of SES on gestation length via preeclampsia diagnosis was not significant. For Black women, high SES was not associated with risk for preeclampsia, although a preeclampsia diagnosis continued to predict lower gestational age at birth (see Fig. 3).

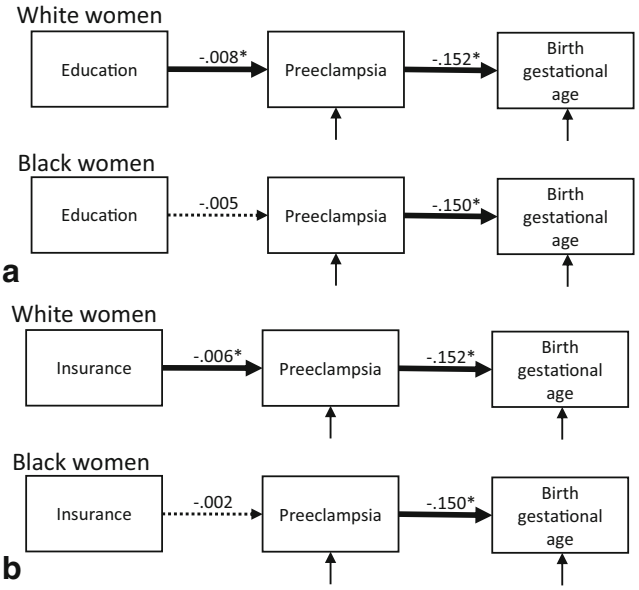

Fig. 3 Path models of the indirect association between SES and gestational age at birth via preeclampsia for White and Black women, from a population-based California cohort of singleton births (2007–2012). **a** Path models for SES as indexed by education. For White women, the indirect effect of education on gestational length via preeclampsia diagnosis was significant, $b = 0.005$, SE = 0.001, $p < 0.001$, such that higher education was associated with lower risk for preeclampsia, which in turn predicted longer gestation. Among Black women, the indirect effect of education on gestational length via preeclampsia diagnosis was not significant, $b = 0.003$, SE = 0.002, $p = 0.140$. For Black women, greater education was not associated with risk for preeclampsia, $b = -0.002$, SE = 0.001, $\beta = -0.005$, $p = 0.140$, although a preeclampsia diagnosis continued to predict lower gestational age at birth, $b = -1.56$, SE = 0.031, $\beta = -0.151$, $p < 0.001$. **b** Path models for SES as indexed by insurance status. Among White women, not using MediCal (higher income status) continued to predict longer gestation through reduced risk for preeclampsia, $b = 0.003$, SE = 0.001, $p < 0.001$ (Fig. 3b). Among Black women, however, an indirect effect of insurance status on gestational length via preeclampsia risk was not detected, $b = 0.001$, SE = 0.002, $p = 0.545$. For Black women, insurance status was not associated with risk for preeclampsia, $b = -0.001$, SE = 0.001, $\beta = -0.002$, $p = 0.544$, although a preeclampsia diagnosis continued to predict lower gestational age at birth, $b = -1.56$, SE = 0.031, $\beta = -0.150$, $p < 0.001$. Standardized coefficients are presented. Significant paths are indicated with an "*." Models were adjusted for diabetes, prenatal care, smoking during pregnancy, parity, pre-pregnancy BMI, age at delivery, and either education or insurance status (not shown). All exogenous variables were allowed to co-vary (not shown)

Discussion

In this study, Black women and women who were low SES, whether indexed by education or insurance status, were at increased risk for preeclampsia, compared to women who were White or of higher SES, respectively. Moreover, higher SES, whether indexed by education or insurance status, attenuated preeclampsia risk in White women but not for Black women. These findings are consistent with a broader body of research on "diminishing returns," which indicates that Black women do not necessarily gain the same health benefits from higher SES as do White women [14–16, 38, 39]. This study demonstrates that these differences are evident in a California statewide cohort for preeclampsia, a pregnancy-specific cardio-metabolic disease.

A second purpose of this work was to test whether differences in risk for preeclampsia by race and SES (indexed by either education or insurance status) also predicted gestational length. Path analyses revealed different patterns by race. Among White women, higher SES indirectly predicted longer gestational length through reduced risk for preeclampsia, consistent with a protective effect of higher SES. In contrast, no indirect effects of either SES indicator on gestational length were observed for Black women. These results suggest that smaller or no effects of higher SES on gestational length among Black women could be accounted for in part by higher preeclampsia risk.

A large body of research supports the existence of "diminishing returns" for Black individuals for a range of

health outcomes. These phenomena are counter to the general belief that SES affects health in a positive, linear fashion, with lower SES individuals being at increased risk, and higher SES individuals being at decreased risk, for poor health [76]. As such, individuals at the intersection of multiple marginalized groups, such as Black women with low SES, are hypothesized to have the worst health outcomes [9, 10]. This study and others, however, show that in many instances, Black women with higher SES have similar [12], or sometimes higher [13], risk for adverse health, relative to Black women with low SES. Why higher SES is not as health protective for Black individuals is a topic of interest with a number of possible explanations. Higher SES places Black women in a position where they are more likely to experience particularly noxious stressors, such as racism and discrimination [77–79]. Racism is a pernicious and pervasive phenomenon that encompasses institutional (e.g., differences in access to goods, services, and opportunities by race), interpersonal (e.g., prejudice and discrimination), and internalized (e.g., acceptance of negative messages of a minority member's abilities and intrinsic worth) aspects [80]. Experiencing racism in any of its forms is distressing and can activate physiological stress pathways, e.g., hypothalamic-pituitary-adrenal (HPA) and sympathetic-adrenal-medulla (SAM) axes, with adverse implications for downstream physiology and health [81, 82]. Indeed, in a sample of Black men, higher discrimination experienced as a result of higher SES undermined any protective effect of higher SES on risk for depression [25]. Other factors to consider are differences in the psychosocial environment. For example, higher area-level racism has been associated with increased preterm birth risk in Black women [83]. Indeed, higher area-level racism, as indexed by lower proportion of Black individuals residing in a neighborhood, was associated with increases in infant mortality risk particularly for Black women of higher SES [84]. Work environment, i.e., management support or work recognition, can also vary for Black and White individual [85]. However, whether workplace environments differ for *higher* SES Black individuals has not been directly tested. Nonetheless, among Black individuals, any health benefits accrued due to higher SES could be undone by accompanying exposure to toxic stressors.

In addition, Black women with higher SES could also be more likely to experience structural and cultural forms of racism that act as barriers to *social structures* or *social networks* expected to be available to higher SES individuals [86]. In other words, higher SES among Black individuals may not come with equal access to resources and benefits. For example, although Black women with higher SES are expected to have more consistent access to healthcare, explicit or implicit bias among healthcare professionals could actually reduce overall access to care and quality of treatment [87]. Collectively, this suggests that SES indicators do not necessarily capture the same socioeconomic resources across races, ethnicities, cultures, or groups, and thus do not predict health consistently across race/ethnicity, group, and culture.

In order to overcome discriminatory practices, Black individuals may engage in coping behaviors that are necessary to attain and maintain higher SES but that have negative consequences for health. For example, "John Henryism" is a high-effort coping strategy used by Black individuals to actively confront external stressors or barriers related to race through persistent and sustained efforts [88]. This cluster of coping behaviors could increase risk for disease due to both the high energy needed to sustain them and the potentially inaccurate belief that one is in control over most stressful situations [78]. Another theory, "skin-deep resilience," posits that the coping strategies needed to succeed as a member of a disadvantaged social group have deleterious consequences for physical health, although these strategies result in outward indicators of achievement and success [31, 89]. Another factor that could increase risk for poor health is increased vigilance, or the attention to details required to navigate a space predominately defined by the dominant culture [90]. Black women with higher SES have to navigate White-dominant cultural spaces more often than Black women with lower SES, and so often need to be more vigilant, with downstream consequences of vigilance for cardiovascular health. As such, Black women potentially do not benefit from higher SES because gains are canceled by the extraordinary degree of effort needed to achieve and sustain that status, and the resulting psychological and biological stress of doing so [91]. To our knowledge, no one has determined whether associations between vigilance and health vary by SES in Black women.

A limitation of this study is that data were obtained from hospital discharge diagnoses, and provide limited in-depth information on psychosocial and other factors (e.g., immigration status or country of origin). Additional prospective studies are needed to explore how factors like various forms of racism, discrimination, vigilance, and John Henryism could drive health disparities by SES in Black women. This study is also limited with respect to the SES indicator. For example, information on household income or size is unknown and no measures of subjective SES were available.

To date, "diminishing returns" has predominately been studied in the context of Black Americans. It is reasonable to presume, however, that any minority group that comes from a historical context of discrimination and marginalization and continues to face discrimination and marginalization may not benefit from higher SES the same as the dominant culture. For example, associations between SES and health are also not consistently found in Hispanic populations ("Hispanic health paradox"). Whether "diminishing returns" or "widening health disparities" are evident for other races and ethnicities, such as Native American, Southeast Asian, or Pacific Islander/Native Hawaiian, is not known. Future research on the

intersection of race/ethnicity and SES in other groups is of possible value.

In sum, compared to White women, Black women were at increased risk for preeclampsia, and risk was not attenuated by higher SES, whether indexed by education or insurance status. Differences in preeclampsia risk by race and SES in part accounted for differences in gestational length between Black and White women. These findings highlight the unique challenges faced by Black women in the USA, and the importance of studying health disparities at the intersection of race/ethnicity and SES.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

References

1. Duley L. The global impact of pre-eclampsia and eclampsia. Semin Perinatol. 2009;33(3):130–7. https://doi.org/10.1053/j.semperi.2009.02.010.
2. Sibai B, Dekker G, Kupferminc M. Pre-eclampsia. Lancet. 2005;365(9461):785–99. https://doi.org/10.1016/s0140-6736(05)71003-5.
3. Esakoff TF, Rad S, Burwick RM, Caughey AB. Predictors of eclampsia in California. J Matern Fetal Neonatal Med. 2016;29(10):1531–5. https://doi.org/10.3109/14767058.2015.1057489.
4. Savitz DA, Danilack VA, Engel SM, Elston B, Lipkind HS. Descriptive epidemiology of chronic hypertension, gestational hypertension, and preeclampsia in New York State, 1995-2004. Matern Child Health J. 2014;18(4):829–38. https://doi.org/10.1007/s10995-013-1307-9.
5. Eskenazi B, Fenster L, Sidney S. A multivariate analysis of risk factors for preeclampsia. JAMA. 1991;266(2):237–41.
6. Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. Lancet. 2010;376(9741):631–44. https://doi.org/10.1016/S0140-6736(10)60279-6.
7. Silva LM, Coolman M, Steegers EA, Jaddoe VW, Moll HA, Hofman A, et al. Low socioeconomic status is a risk factor for preeclampsia: the Generation R Study. J Hypertens. 2008;26:1200–8.
8. Reeves R, Rodrigue E, Kneebone E. Five evils: multidimensional poverty and race in America. Washington, DC: Brookings Institute; 2016.
9. Kelly UA. Integrating intersectionality and biomedicine in health disparities research. ANS Adv Nurs Sci. 2009;32(2):E42–56. https://doi.org/10.1097/ANS.0b013e3181a3b3fc.
10. Hogan VK, de Araujo EM, Caldwell KL, Gonzalez-Nahm SN, Black KZ. "We black women have to kill a lion everyday": an intersectional analysis of racism and social determinants of health in Brazil. Soc Sci Med. 2018;199:96–105. https://doi.org/10.1016/j.socscimed.2017.07.008.
11. Fuller-Rowell TE, Curtis DS, Doan SN, Coe CL. Racial disparities in the health benefits of educational attainment: a study of inflammatory trajectories among African American and white adults. Psychosom Med. 2015;77(1):33–40. https://doi.org/10.1097/PSY.0000000000000128.
12. Assari S, Nikahd A, Malekahmadi MR, Lankarani MM, Zamanian H. Race by gender group differences in the protective effects of socioeconomic factors against sustained health problems across five domains. J Racial Ethn Health Disparities. 2016;4:884–94. https://doi.org/10.1007/s40615-016-0291-3.
13. Ross KM, Guardino C, Dunkel Schetter C, Hobel CJ. Interactions between race/ethnicity, poverty status, and pregnancy cardiometabolic diseases in prediction of postpartum cardio-metabolic health. Ethn Health 2018;1–16. https://doi.org/10.1080/13557858.2018.1493433.
14. Assari S. Health disparities due to diminished return among Black Americans: public policy solutions. Soc Iss Policy Rev. 2018;12:112–45.
15. Assari S. Unequal gain of equal resources across racial groups. Int J Health Policy Manag. 2017;7(1):1–9. https://doi.org/10.15171/ijhpm.2017.90.
16. Williams DR, Collins C. US socioeconomic and racial differences in health: patterns and explanations. Annu Rev Sociol. 1995;21:349–86.
17. Assari S, Lankarani MM. Race and urbanity alter the protective effect of education but not income on mortality. Front Public Health. 2016;4:100. https://doi.org/10.3389/fpubh.2016.00100.
18. Everett BG, Rehkopf DH, Rogers RG. The nonlinear relationship between education and mortality: an examination of cohort, race/ethnic, and gender differences. Popul Res Policy Rev. 2013;32(6). https://doi.org/10.1007/s11113-013-9299-0.
19. Hayward MD, Hummer RA, Sasson I. Trends and group differences in the association between educational attainment and U.S. adult mortality: implications for understanding education's causal influence. Soc Sci Med. 2015;127:8–18. https://doi.org/10.1016/j.socscimed.2014.11.024.
20. Assari S. Life expectancy gain due to employment status depends on race, gender, education, and their intersections. J Racial Ethn Health Disparities. 2018;5(2):375–86. https://doi.org/10.1007/s40615-017-0381-x.
21. Farmer MM, Ferraro KF. Are racial disparities in health conditional on socioeconomic status? Soc Sci Med. 2005;60(1):191–204. https://doi.org/10.1016/j.socscimed.2004.04.026.
22. Holmes CJ, Zajacova A. Education as "the great equalizer": health benefits for Black and White adults. Soc Sci Q. 2014;n/a–n/a. https://doi.org/10.1111/ssqu.12092.
23. Cene CW, Halladay JR, Gizlice Z, Roedersheimer K, Hinderliter A, Cummings DM, et al. Associations between subjective social status and physical and mental health functioning among patients with hypertension. J Health Psychol. 2015;21:2624–35. https://doi.org/10.1177/1359105315581514.
24. Assari S. Ethnic and gender differences in additive effects of socioeconomics, psychiatric disorders, and subjective religiosity on suicidal ideation among blacks. Int J Prev Med. 2015;6:53. https://doi.org/10.4103/2008-7802.158913.
25. Hudson DL, Bullard KM, Neighbors HW, Geronimus AT, Yang J, Jackson JS. Are benefits conferred with greater socioeconomic position undermined by racial discrimination among African American men? J Mens Health. 2012;9(2):127–36. https://doi.org/10.1016/j.jomh.2012.03.006.
26. Kubzansky LD, Berkman LF, Seeman TE. Social conditions and distress in elderly persons: findings from the MacArthur Studies of Successful Aging. J Gerontol B Psychol Sci Soc Sci. 2000;55(4):P238–46.
27. Assari S, Thomas A, Caldwell CH, Mincy RB. Blacks' diminished health return of family structure and socioeconomic status; 15 years of follow-up of a national urban sample of youth. J Urban Health. 2018;95(1):21–35. https://doi.org/10.1007/s11524-017-0217-3.

28. Geronimus AT, Pearson JA, Linnenbringer E, Schulz AJ, Reyes AG, Epel ES, et al. Race-ethnicity, poverty, urban stressors, and telomere length in a Detroit community-based sample. J Health Soc Behav. 2015;56(2):199–224. https://doi.org/10.1177/0022146515582100.
29. Paul K, Boutain D, Agnew K, Thomas J, Hitti J. The relationship between racial identity, income, stress and C-reactive protein among parous women: implications for preterm birth disparity research. J Natl Med Assoc. 2008;100(5):540–6. https://doi.org/10.1016/s0027-9684(15)31300-6.
30. Beach SR, Lei MK, Brody GH, Kim S, Barton AW, Dogan MV, et al. Parenting, socioeconomic status risk, and later young adult health: exploration of opposing indirect effects via DNA methylation. Child Dev. 2016;87(1):111–21. https://doi.org/10.1111/cdev.12486.
31. Brody GH, Yu T, Chen E, Miller GE, Kogan SM, Beach SR. Is resilience only skin deep?: rural African Americans' socioeconomic status-related risk and competence in preadolescence and psychological adjustment and allostatic load at age 19. Psychol Sci. 2013;24:1285–93.
32. Wang JT, Wiebe DJ, White PC. Developmental trajectories of metabolic control among White, Black, and Hispanic youth with type 1 diabetes. J Pediatr. 2011;159(4):571–6. https://doi.org/10.1016/j.jpeds.2011.03.053.
33. LaVeist TA, Thorpe RJ Jr, Galarraga JE, Bower KM, Gary-Webb TL. Environmental and socio-economic factors as contributors to racial disparities in diabetes prevalence. J Gen Intern Med. 2009;24(10):1144–8. https://doi.org/10.1007/s11606-009-1085-7.
34. Gaillard TR, Schuster DP, Bossetti BM, Green PA, Osei K. Do sociodemographics and economic status predict risks for type II diabetes in African Americans? Diab Educ. 1997;23:294–300.
35. Kershaw KN, Diez Roux AV, Burgard SA, Lisabeth LD, Mujahid MS, Schulz AJ. Metropolitan-level racial residential segregation and black-white disparities in hypertension. Am J Epidemiol. 2011;174(5):537–45. https://doi.org/10.1093/aje/kwr116.
36. Thorpe RJ Jr, Brandon DT, LaVeist TA. Social context as an explanation for race disparities in hypertension: findings from the Exploring Health Disparities in Integrated Communities (EHDIC) Study. Soc Sci Med. 2008;67(10):1604–11. https://doi.org/10.1016/j.socscimed.2008.07.002.
37. Maynard JW, Fang H, Petri M. Low socioeconomic status is associated with cardiovascular risk factors and outcomes in systemic lupus erythematosus. J Rheumatol. 2012;39(4):777–83. https://doi.org/10.3899/jrheum.110724.
38. Blumenshine P, Egerter S, Barclay CJ, Cubbin C, Braveman PA. Socioeconomic disparities in adverse birth outcomes: a systematic review. Am J Prev Med. 2010;39(3):263–72. https://doi.org/10.1016/j.amepre.2010.05.012.
39. Culhane JF, Goldenberg RL. Racial disparities in preterm birth. Semin Perinatol. 2011;35(4):234–9. https://doi.org/10.1053/j.semperi.2011.02.020.
40. Ahern J, Pickett KE, Abrams B. Preterm birth among African American and White women: a multilevel analysis of socioeconomic characteristics and cigarette smoking. J Epidemiol Community Health. 2003;57:606–11.
41. Braveman PA, Heck K, Egerter S, Marchi KS, Dominguez TP, Cubbin C, et al. The role of socioeconomic factors in Black-White disparities in preterm birth. Am J Public Health. 2015;105(4):694–702. https://doi.org/10.2105/AJPH.2014.302008.
42. Lieberman E, Ryan KJ, Monson RR, Schoenbaum SC. Risk factors accounting for racial differences in the rate of premature birth. N Engl J Med. 1987;317(12):743–8. https://doi.org/10.1056/NEJM198709173171206.
43. Messer LC, Laraia BA, Kaufman JS, Eyster J, Holzman C, Culhane J, et al. The development of a standardized neighborhood deprivation index. J Urban Health. 2006;83(6):1041–62. https://doi.org/10.1007/s11524-006-9094-x.
44. Messer LC, Kaufman JS, Dole N, Savitz DA, Laraia BA. Neighborhood crime, deprivation, and preterm birth. Ann Epidemiol. 2006;16(6):455–62. https://doi.org/10.1016/j.annepidem.2005.08.006.
45. Reagan PB, Salsberry PJ. Race and ethnic differences in determinants of preterm birth in the USA: broadening the social context. Soc Sci Med. 2005;60(10):2217–28. https://doi.org/10.1016/j.socscimed.2004.10.010.
46. Schempf AH, Kaufman JS, Messer LC, Mendola P. The neighborhood contribution to black-white perinatal disparities: an example from two north Carolina counties, 1999-2001. Am J Epidemiol. 2011;174(6):744–52. https://doi.org/10.1093/aje/kwr128.
47. Collins JW Jr, Hammond NA. Relation of maternal race to the risk of preterm, non-low birth weight infants: a population study. Am J Epidemiol. 1996;143(4):333–7.
48. Pickett KE, Ahern JE, Selvin S, Abrams B. Neighborhood socioeconomic status, maternal race and preterm delivery: a case-control study. Ann Epidemiol. 2002;12(6):410–8.
49. Savitz DA, Kaufman JS, Dole N, Siega-Riz AM, Thorp JM Jr, Kaczor DT. Poverty, education, race, and pregnancy outcome. Eth Dis. 2004;14(3):322–9.
50. Collins JW Jr, David RJ, Simon DM, Prachand NG. Preterm birth among African American and White women with a lifelong residence in high-income Chicago neighborhoods: an exploratory study. Ethn Dis. 2007;17:113–7.
51. Slaughter-Acey JC, Holzman C, Calloway D, Tian Y. Movin' on up: socioeconomic mobility and the risk of delivering a small-for-gestational age infant. Matern Child Health J. 2016;20(3):613–22. https://doi.org/10.1007/s10995-015-1860-5.
52. Colen CG, Geronimus AT, Bound J, James SA. Maternal upward socioeconomic mobility and black-white disparities in infant birthweight. Am J Public Health. 2006;96(11):2032–9. https://doi.org/10.2105/AJPH.2005.076547.
53. Gorman BK. Racial and ethnic variation in low birthweight in the United States: individual and contextual determinants. Health Place. 1999;5(3):195–207.
54. Parker JD, Schoendorf K, Kiely JL. Associations between measures of socioeconomic status and low birth weight, small for gestational age, and premature delivery in the United States. Ann Epidemiol. 1994;4:271–8.
55. Schoendorf K, Hogue CJ, Kleinman JC, Rowley D. Mortality among infants of Black as compared to White college-educated parents. N Engl J Med. 1992;326:1522–6.
56. Starfield B, Shapiro S, Weiss J, Liang KY, Ra K, Paige D, et al. Race, family income, and low birth weight. Am J Epidemiol. 1991;134(10):1167–74.
57. Singh GK, Yu SM. Infant mortality in the United States: trends, differentials, and projections, 1950 through 2010. Am J Public Health. 1995;85:957–64.
58. Castrillio SM, Rankin KM, David RJ, Collins JW Jr. Small-for-gestational age and preterm birth across generations: a population-based study of Illinois births. Matern Child Health J. 2014;18(10):2456–64. https://doi.org/10.1007/s10995-014-1484-1.
59. Collins JW, Simon DM, Jackson TA, Drolet A. Advancing maternal age and infant birth weight among urban African Americans: the effect of neighborhood poverty. Ethn Dis. 2006;16:180–6.
60. Collins JW Jr, Rankin KM, David RJ. African American women's lifetime upward economic mobility and preterm birth: the effect of fetal programming. Am J Public Health. 2011;101(4):714–9. https://doi.org/10.2105/AJPH.2010.195024.
61. Goldenberg RL, Cliver SP, Mulvihill FX, Hickey CA, Hoffman HJ, Klerman LV, et al. Medical, psychosocial, and behavioral risk factors do not explain the increased risk for low birth weight among

black women. Am J Obstet Gynecol. 1996;175(5):1317–24. https://doi.org/10.1016/S0002-9378(96)70048-0.
62. Jaffee KD, Perloff JD. An ecological analysis of racial differences in low birthweight: implications for maternal and child health social work. Health Soc Work. 2003;28(1):9–22.
63. Masi CM, Hawkley LC, Piotrowski ZH, Pickett KE. Neighborhood economic disadvantage, violent crime, group density, and pregnancy outcomes in a diverse, urban population. Soc Sci Med. 2007;65(12):2440–57. https://doi.org/10.1016/j.socscimed.2007.07.014.
64. Pearl M, Braveman P, Abrams B. The relationship of neighborhood socioeconomic characteristics to birthweight among 5 ethnic groups in California. Am J Public Health. 2001;91(11):1808–14. https://doi.org/10.2105/Ajph.91.11.1808.
65. Rauh VA, Andrews HF, Garfinkel RS. The contribution of maternal age to racial disparities in birthweight: a multilevel perspective. Am J Public Health. 2001;91(11):1815–24.
66. Sparks PJ. Do biological, sociodemographic, and behavioral characteristics explain racial/ethnic disparities in preterm births? Soc Sci Med. 2009;68(9):1667–75. https://doi.org/10.1016/j.socscimed.2009.02.026.
67. Hedderson M. Gestational diabetes mellitus and lesser degrees of pregnancy hyperglycemia: association with increased risk of spontaneous preterm birth. Obstet Gynecol. 2003;102(4):850–6. https://doi.org/10.1016/s0029-7844(03)00661-6.
68. Xiong X, Saunders LD, Wang FL, Demianczuk NN. Gestational diabetes mellitus: prevalence, risk factors, maternal and infant outcomes. Int J Gynaecol Obstet. 2001;75(3):221–8.
69. Hendricks CH. Delivery patterns and reproductive efficiency among groups of differing socioeconomic status and ethnic origins. AJOG. 1967;97(5):608–24.
70. Baer RJ, Altman MR, Oltman SP, Ryckman KK, Chambers CD, Rand L, et al. Maternal factors influencing late entry into prenatal care: a stratified analysis by race or ethnicity and insurance status. J Matern Fetal Neonatal Med. 2018;32:1–7. https://doi.org/10.1080/14767058.2018.1463366.
71. Baer RJ, Chambers CD, Bandoli G, Jelliffe-Pawlowski LL. Risk of preterm birth by subtype among Medi-Cal participants with mental illness. Am J Obstet Gynecol. 2016;215(4):519 e1–9. https://doi.org/10.1016/j.ajog.2016.06.017.
72. American Medical Association. International Classification of Diseases: ICD-9-CM. Chicago: American Medical Association; 2008.
73. IBM Corp. IBM SPSS Statistics for Windows, Version 24.0. IBM Corp: Armonk; 2016.
74. Rosseel Y. lavaan : an R package for structural equation modeling. J Stat Softw. 2012;48:ePub.
75. R Core Team. R: a language and environment for statistical computing. Vienna: R Foundation for Statistical Computing; 2014.
76. Adler NE, Rehkopf DH. U.S. disparities in health: descriptions, causes, and mechanisms. Ann Rev Public Health. 2008;29:235–52. https://doi.org/10.1146/annurev.publhealth.29.020907.090852.
77. Colen CG, Ramey DM, Cooksey EC, Williams DR. Racial disparities in health among nonpoor African Americans and Hispanics: the role of acute and chronic discrimination. Soc Sci Med. 2018;199:167–80. https://doi.org/10.1016/j.socscimed.2017.04.051.
78. Hudson DL, Neighbors HW, Geronimus AT, Jackson JS. Racial discrimination, John Henryism, and depression among African Americans. J Black Psychol. 2016;42(3):221–43. https://doi.org/10.1177/0095798414567757.
79. Tyson K, Darity W Jr, Castellino DR. It's not "a Black thing": understanding the burden of acting White and other dilemmas of high achievement. Am Sociol Rev. 2005;70:582–605.
80. Jones CP. Levels of racism: a theoretic framework and a Gardener's tale. Am J Public Health. 2000;90:1212–5.
81. Sapolsky RM, Romero LM, Munck AU. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr Rev. 2000;21(1):55–89. https://doi.org/10.1210/er.21.1.55.
82. Irwin MR, Cole SW. Reciprocal regulation of the neural and innate immune systems. Nat Rev Immunol. 2011;11(9):625–32. https://doi.org/10.1038/nri3042.
83. Chae DH, Clouston S, Martz CD, Hatzenbuehler ML, Cooper HLF, Turpin R, et al. Area racism and birth outcomes among Blacks in the United States. Soc Sci Med. 2018;199:49–55. https://doi.org/10.1016/j.socscimed.2017.04.019.
84. Kothari CL, Paul R, Dormitorio B, Ospina F, James A, Lenz D, et al. The interplay of race, socioeconomic status and neighborhood residence upon birth outcomes in a high black infant mortality community. SSM Popul Health. 2016;2:859–67. https://doi.org/10.1016/j.ssmph.2016.09.011.
85. McCluney CL, Schmitz LL, Hicken MT, Sonnega A. Structural racism in the workplace: does perception matter for health inequalities? Soc Sci Med. 2018;199:106–14. https://doi.org/10.1016/j.socscimed.2017.05.039.
86. Hicken MT, Kravitz-Wirtz N, Durkee M, Jackson JS. Racial inequalities in health: framing future research. Soc Sci Med. 2018;199:11–8. https://doi.org/10.1016/j.socscimed.2017.12.027.
87. Maina IW, Belton TD, Ginzberg S, Singh A, Johnson TJ. A decade of studying implicit racial/ethnic bias in healthcare providers using the implicit association test. Soc Sci Med. 2018;199:219–29. https://doi.org/10.1016/j.socscimed.2017.05.009.
88. James SA. John Henryism and the health of African Americans. In: LaVeist T, editor. Race, ethnicity, and health. San Francisco: Jossey-Bass; 2002.
89. Miller GE, Cohen S, Janicki-Deverts D, Brody GH, Chen E. Viral challenge reveals further evidence of skin-deep resilience in African Americans from disadvantaged backgrounds. Health Psychol. 2016;35(11):1225–34. https://doi.org/10.1037/hea0000398.
90. Hicken MT, Lee H, Hing AK. The weight of racism: vigilance and racial inequalities in weight-related measures. Soc Sci Med. 2018;199:157–66. https://doi.org/10.1016/j.socscimed.2017.03.058.
91. Dunkel Schetter C. Psychological science on pregnancy: stress processes, biopsychosocial models, and emerging research issues. Annu Rev Psychol. 2011;62:531–58. https://doi.org/10.1146/annurev.psych.031809.130727.